Conversion Disorder

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ABSTRACT

PURPOSE OF REVIEW: This article provides a broad overview of conversion disorder, encompassing diagnostic criteria, epidemiology, etiologic theories, functional neuroimaging findings, outcome data, prognostic indicators, and treatment.

RECENT FINDINGS: Two important changes have been made to the recent *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*) diagnostic criteria: the criteria that conversion symptoms must be shown to be involuntary and occurring as the consequence of a recent stressor have been dropped. Outcome studies show that the rate of misdiagnosis has declined precipitously since the 1970s and is now around 4%. Functional neuroimaging has revealed a fairly consistent pattern of hypoactivation in brain regions linked to the specific conversion symptom, accompanied by ancillary activations in limbic, paralimbic, and basal ganglia structures. Cognitive-behavioral therapy looks promising as the psychological treatment of choice, although more definitive data are still awaited, while preliminary evidence indicates that repetitive transcranial magnetic stimulation could prove beneficial as well.

SUMMARY: Symptoms of conversion are common in neurologic and psychiatric settings, affecting up to 20% of patients. The full syndrome of conversion disorder, while less prevalent, is associated with a guarded prognosis and a troubled psychosocial outcome. Much remains uncertain with respect to etiology, although advances in neuroscience and technology are providing reproducible findings and new insights. Given the confidence with which the diagnosis can be made, treatment should not be delayed, as symptom longevity can influence outcome.

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INTRODUCTION

The challenges posed by medically unexplained symptoms can be traced back to the origins of Western medicine. The list of luminaries who have turned their attention to the vexing question of etiology is long and distinguished and includes, among others, Hippocrates, Galen, Paracelsus, Robert Burton, William Harvey, Thomas Willis, Thomas Sydenham, William Cullen, Philippe Pinel, Franz Anton Mesmer, Jean-Martin Charcot, Pierre Janet, and Sigmund Freud.[1] Such a prolonged hold over succeeding generations of physicians of diverse specialties hints at the complexity of the disorder. This is reflected in the uncertainty of how best to label these disorders and where they fit in the classification of mental illness. The term *hysteria* disappeared from the *Diagnostic and Statistical Manual of Mental*

KEY POINTS

- The diagnosis of conversion disorder depends on the presence of atypical neurologic-type symptoms that do not conform to the known anatomic and physiologic constructs that support neurologic diagnoses.
- The diagnostic criteria for conversion disorder have been revised recently to reflect two significant conceptual shifts. It is no longer necessary to assert that the symptoms are not intentionally produced or linked to recent stressors.

Disorders, Third Edition (*DSM-III*)[2] in 1968 because of a pejorative association with the wandering womb hypothesis, and a new set of disorders was created to replace it. The 2013 *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*) radically overhauled the nomenclature yet again, discarding somatization disorder, undifferentiated somatoform disorder, and hypochondriasis.[3] Notwithstanding this periodic shuffling of the diagnostic deck, the *DSM-5* and *International Classification of Diseases, Tenth Revision* (*ICD-10*) continue to differ in their approach to taxonomy.[4]

One island of relative stability amid all this semantic flux is the condition of conversion disorder, which was retained in *DSM-5*, although even here the authors added an alternative terminology in parentheses, namely *functional neurological symptom disorder*. In conversion disorder, the focus is purely on atypical neurologic symptoms that do not conform to any neurologic disorder. This article discusses the clinical signs, epidemiology, etiology, diagnostic accuracy, functional brain imaging findings, and treatment of conversion disorder, interspersed with case reports to illustrate salient points.

CLINICAL SIGNS

The *DSM-5* diagnostic criteria for conversion disorder appear in TABLE 10-1. One notable change from the earlier *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM-IV*) criteria has been to drop the assertion that the symptom or deficit is not intentionally produced or feigned.[5] This change is to be welcomed, for it is frequently impossible to discern these factors during the clinical interview. This does not, however, imply that conversion symptoms are willfully produced, and it remains a tacit assumption when it comes to therapy that involuntary factors underpin the etiology. A second conceptual change in the *DSM-5* is that the criterion invoking psychological stressors is no longer considered mandatory; instead, one has the option of coding separately for it. This change is once again welcomed, for symptom onset is not invariably preceded by conflict.

In the *DSM-5* criteria, the disorder is coded according to symptom type. A significant omission from the list is cognitive impairment, most typically memory. To the authors of the *DSM-5*, memory impairment in the absence of an underlying dementia is better explained by the concept of dissociation (dissociative amnesia), placing the classification at odds with the *ICD-10* approach.

EPIDEMIOLOGY

The data here are mixed for methodologic reasons. Some studies report the frequency of individual conversion symptoms, whereas others refer to the full diagnosis. It is estimated that up to one-fourth of all patients in a general hospital setting have individual symptoms of conversion,[6] with 5% of these meeting the full diagnostic criteria.[7] These figures increase in neurologic populations, in which it is estimated that 20% of patients attending a neurologic outpatient clinic will have symptoms of conversion.[8] Large-scale population-based studies show a good degree of concordance, with incidence rates falling in the 4 per 100,000 to 12 per 100,000 range.[9] Not surprisingly, the incidence rises to 11 per 100,000 to 22 per 100,000 in a primarily psychiatric setting.[10]

Conversion disorder can occur across the lifespan and is more common in women[11] and in those who have a history of abuse, not necessarily sexual, during childhood (CASE 10-1).[12] In keeping with the revised *DSM-5* criteria, an

association exists with stressful life events around the time of symptom onset, although here one must be mindful of retrospective bias in the reporting of these events.

ETIOLOGY

The *DSM* approach to the diagnosis and classification of mental illness is essentially descriptive and shies away from etiologic assumptions given the many uncertainties surrounding causality. One exception is conversion disorder, for which the role of stressors is still acknowledged, even if now not considered obligatory. While it is recognized that precipitating stressors are not always present, the freudian idea of unresolved psychological conflicts manifesting as physical symptoms still persists and is not without validity, as CASE 10-1 illustrates.

DSM-5 Criteria for Conversion Disorder (Functional Neurological Symptom Disorder)[a]

TABLE 10-1

A **One or more symptoms of altered voluntary motor or sensory function**

B **Clinical findings provide evidence of incompatibility between the symptom and recognized neurological or medical conditions**

C **The symptom or deficit is not better explained by another medical or mental disorder**

D **The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation**

Coding note: The *ICD-9-CM* code for conversion disorder is **300.11**, which is assigned regardless of the symptom type. The *ICD-10-CM* code depends on the symptom type (see below).

Specify symptom type:

(F44.4) With weakness or paralysis

(F44.4) With abnormal movement (eg, tremor, dystonic movement, myoclonus, gait disorder)

(F44.4) With swallowing symptoms

(F44.4) With speech symptom (eg, dysphonia, slurred speech)

(F44.5) With attacks or seizures

(F44.6) With anesthesia or sensory loss

(F44.6) With special sensory symptom (eg, visual, olfactory, or hearing disturbance)

(F44.7) With mixed symptoms

Specify if:

Acute episode: Symptoms present for less than 6 months

Persistent: Symptoms occurring for 6 months or more

Specify if:

With psychological stressor (*specify stressor*)

Without psychological stressor

DSM-5 = Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition; *ICD-9-CM = International Classification of Diseases, Ninth Revision, Clinical Modification*; *ICD-10-CM = International Classification of Diseases, Tenth Revision, Clinical Modification*.

[a] Reprinted with permission from American Psychiatric Association.[3]

Implicit in our understanding of conversion disorder is that symptoms are not deliberately, or consciously, produced. With this construct immutable, it is necessary to look beyond psychoanalytic theories for clues as to etiology. Cognitive models have posited that a faulty executive system could channel misperceptions into conscious awareness.[13] Attentional deficits[14] and problems with working memory[15] have been found but not replicated. Impulsivity linked to the processing of novel sensory data, thereby introducing interpretation bias, has been implicated too.[16] While all are of interest, the cognitive data are few and fragmented and have yet to provide a coherent overall theory accounting for symptom misattribution. More research is clearly needed here, but in a disorder

CASE 10-1

A 25-year-old woman was admitted to a neuropsychiatric unit because of sudden unexplained left-sided weakness and sensory loss in the presence of atypical neurologic findings and a normal CT brain scan. She had previously been physically and psychiatrically well.

Neurologic examination revealed a flaccid left arm and leg, dense anesthesia in the affected limbs, and midline truncal anesthesia. Muscle stretch reflexes were normal and Babinski signs were absent. Her mental state examination revealed distress and bemusement at her condition and resentment that she had been admitted to a psychiatric unit rather than a neurology unit. Her paralysis and sensory loss made it very difficult for her to manage her activities of daily living, including her hygiene, and her inability to walk meant she was reliant on a wheelchair to get around. She denied a history of recent stressors, and her family confirmed this. She was reluctant to talk about herself or divulge details of her personal history other than to confirm that she lived alone, earned a good wage, enjoyed her work, and had a good circle of friends.

Two weeks into her admission and faced with no symptom improvement and ongoing recalcitrance to self-revelation, a sodium amytal interview was undertaken with her permission. Under light sedation from the IV barbiturate, the patient was encouraged to open up and be more forthcoming. Suitably relaxed, she revealed that she had been raped 5 years earlier, had become pregnant, and had an abortion. She had never previously divulged this to family or friends because of an intense feeling of shame. For 5 years, she had lived with this secret and gotten on with her life until 1 month previously, when she had fallen in love with a coworker who had not reciprocated her affections. Soon thereafter, the loss of sensation had begun, quickly followed by paralysis. The abreaction (ie, release of emotional tension after her recall of the traumatic event) induced by the amytal interview was the catalyst that opened the door to psychotherapy and, in time, complete symptom resolution.

COMMENT

This case provides compelling evidence of emotional upset, involuntarily suppressed, that underlies the quasineurologic difficulties of an individual with conversion disorder. It also demonstrates how recovery can take place when these suppressed feelings are revealed and then addressed.

with protean manifestations, it remains unclear whether a central unifying cognitive theory exists for conversion disorder as a whole or whether specific theories are needed for each type of symptom. Furthermore, it is uncertain whether cognitive deficits will mesh with the physiologic findings that are symptom specific, such as those pertaining to premovement EEG potentials noted in functional myoclonus,[17] changes in the blink reflex recovery cycle in atypical (psychogenic) blepharospasm,[18] or abnormalities in neuroimaging (discussed later in this article). Which, in turn, begs the question: Is it important that they do? When searching for diagnostic construct validity, it is, of course, preferable for the data from multiple sources to align coherently, but conversion may be the outlier here. Perhaps this can account for why the psychological concept of suppressed emotional problems finding an outlet by their conversion into physical symptoms remains attractive as a general theory, even if it superficially blurs the margins when it comes to explaining symptom specificity.

DIAGNOSTIC ACCURACY

Notwithstanding all the uncertainties surrounding the underpinnings of conversion disorder, one indirect marker of diagnostic validity is the degree to which the diagnosis is made correctly. Here the data offer reassurance. A systematic review of 27 studies involving 1466 subjects with a median duration of 5 years of follow-up revealed a consistent 4% rate of misdiagnosis (ie, the presence of a neurologic condition rather than a conversion disorder) from 1970 onward. Of note is that this represented a steep decline from misdiagnosis rates of 29% and 17% in the 1950s and 1960s, respectively, with improvements in study design rather than improved diagnostic accuracy in the post-CT era credited for this.[19] Further support for diagnostic accuracy and consistency comes from a Scottish study of 1144 patients assessed at baseline by neurologists as having symptoms "unexplained by organic disease." At 18-month follow-up, only four individuals (0.4%) had their diagnoses changed to a definite neurologic condition.[20]

It should be remembered that unexplained symptoms can also arise in the context of a confirmed neurologic diagnosis, such as multiple sclerosis. Evidence suggests that no one particular neurologic disease is more frequently implicated here.[21] Individuals who self-reported more unexplained neurologic symptoms also endorsed more psychiatric symptoms. The complexities of presentations such as these are illustrated in CASE 10-2.

BRAIN IMAGING

The results from a small, yet compelling, functional MRI (fMRI) task activation literature show that in the presence of sensory and motor conversion symptoms, activation in the respective motor and somatosensory cortices is either absent or reduced. Concomitantly, activation is seen in limbic, paralimbic, and basal ganglia regions, but only in the conversion subjects. From a methodologic standpoint, it is more straightforward to undertake a fMRI study of sensory conversion symptoms given that nothing is asked of the subject other than to lay quietly in the scan while a sensory stimulus is applied. Early studies of sensory conversion were confined to single case reports, but more recently a study of 10 people with unilateral sensory loss was undertaken.[22] A vibrotactile stimulus was applied separately to the anesthetic and sensate regions in a block design, which entailed 4 seconds of stimulation followed by 26 seconds of no stimulation, the latter required to negate habituation to the stimulus. This pattern was repeated

KEY POINTS

- While it is accepted that symptoms of conversion disorder are involuntary and not simulated, their precise origins remain unclear.
- Given the protean manifestations of conversion, it remains unclear from an etiologic perspective whether a single theory can explain the disorder or whether specific theories are required according to symptom presentation.
- Rates of misdiagnosis of a neurologic disorder as a conversion disorder have improved considerably over the decades and are now low. Current estimates are approximately 4%.
- Conversion disorder can still occur in the presence of a confirmed neurologic disorder as long as the signs and symptoms remain atypical and do not conform to established anatomic and physiologic constructs.

CASE 10-2

A 28-year-old woman began experiencing very brief losses of sensation associated with transient weakness in all her limbs. At first, she tried to ignore the phenomena, but as they became more persistent, she discovered that vigorously rubbing the affected areas caused sensation to return and the weakness to resolve. This "remedy" proved short-lived, however, and soon the symptoms began affecting her gait, which became labored, shuffling, and interspersed with knee-bending movements. She consulted a neurologist and two physical medicine and rehabilitation specialists before being referred to a neuropsychiatry clinic. She had no history of prior medical or psychiatric illness. Her developmental history was unremarkable, and detailed inquiry failed to reveal a history of recent stressors or past adverse life events. These facts were confirmed in interviews with her husband and sister.

Her neurologic examination was repeatedly normal. No brain imaging had been completed before the referral, with all three of her earlier physicians noting that the obviously atypical nature of her symptoms, particularly her bizarre gait, left little doubt to them as to the psychiatric origins of her symptoms. This conclusion, in their collective opinions, negated the need for further investigation. Head CT was nevertheless completed and revealed a large left frontal meningioma with considerable edema and midline shift. Rather than express shock or surprise, her face lit up with relief. She felt vindicated. She had always believed something was physically wrong with her and had strongly (and silently) resented the referral to a neuropsychiatrist.

The tumor was subsequently removed successfully, and her neurologic symptoms resolved completely. While the neurologist, physical medicine and rehabilitation specialists, neurosurgeon, and neuropsychiatrist involved in her care had little doubt that the odd gait was functional, less certainty was expressed on the role of such a large brain tumor in her presentation.

COMMENT

It is important to remember that conversion disorder can still be diagnosed in the presence of a confirmed neurologic disorder when the symptoms are grossly atypical, as they were in this patient. In ruling out a possible causative effect from the tumor, no suitable alternative explanation was put forward. As in many patients with conversion disorder, the presentation left the multidisciplinary treatment team with more questions than answers. A final point of interest was the patient's emotional reaction to receiving the news from the neuropsychiatrist that she had a very large brain tumor. The patient's feelings of relief and vindication underscore the importance of physicians validating the symptoms displayed by a person with conversion disorder. To those with atypical weakness, loss of sensation, aphonia, or blindness, the difficulties are all too real and profoundly disabling, even if their disorder is classified as psychiatric and the etiology occasionally linked to emotional factors. It is now known that conversion disorder results from a disturbance in brain function. Conveying this to patients is not only validating but, for many, reassuring too.

10 times. The results revealed decreased activation of the somatosensory cortex contralateral to the anesthetic region relative to the sensate side. In addition, significantly greater ancillary activation was seen in 10 areas when the stimulus was applied to anesthetic compared to the contralateral sensate regions (FIGURE 10-1). The areas involved were the right paralimbic cortices (anterior cingulate and insula), right temporoparietal junction (angular gyrus and inferior parietal lobe), bilateral dorsolateral prefrontal cortex (middle frontal gyri), right orbital frontal cortex (superior frontal gyrus), right caudate, right ventral-anterior thalamus, and left angular gyrus. These findings were interpreted as evidence of abnormal cerebral activation of networks involved in emotional processing and sensory integration.

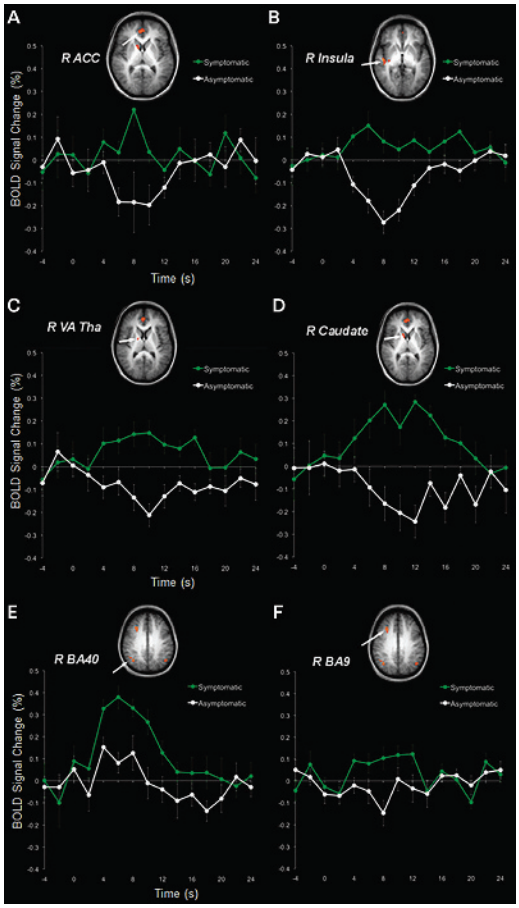

FIGURE 10-1
Event-related group averages of the blood oxygen level dependent (BOLD) response time-locked to the onset of somatosensory stimulation in a study of 10 patients with unilateral sensory loss due to conversion disorder. *A–F*, Ancillary brain regions. *A*, Right anterior cingulate cortex (R ACC); *B*, right insula (R insula); *C*, right ventral anterior nucleus of the thalamus (R Va Tha); *D*, right caudate (R caudate); *E*, right angular gyrus (R BA40); and *F*, right dorsolateral prefrontal cortex (R BA9). Stimulation was applied for a duration of 4 seconds. The *green line* represents stimulation applied to the symptomatic body part and the *white line* to the asymptomatic body part. Error bars represent the standard error.

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The question of whether these abnormal changes in brain activation improve on symptom resolution was addressed in a single-photon emission computed tomography (SPECT) study of seven patients with unilateral sensorimotor loss using passive vibratory stimulation to both hands.[23] All the subjects were tested when symptomatic and 2 to 4 months after recovery. The results revealed a consistent decrease of regional cerebral blood flow in the thalamus and basal ganglia contralateral to the neurologic deficit. The authors showed that these findings were present in each patient and resolved with symptom resolution. Significantly, poorer recovery was predicted by the degree of hypoactivation in the contralateral caudate when symptoms were present. The conclusion derived from these data was that conversion symptoms arose from a dysfunction in striato-thalamo-cortical circuits involved in sensorimotor function. Of note, however, was that

KEY POINT

- **Findings on functional MRI in patients with conversion disorder are specific to symptom type. Depending on the presenting symptom, the data are consistent in showing hypoactivation of cortical (somatosensory) regions coupled with ancillary activation of limbic and basal ganglia structures.**

no hypoactivation was detected in primary sensory or motor areas, unlike the findings from the fMRI literature. The same inconsistencies pertain to regions of ancillary activation for the SPECT data, which did not include the orbitofrontal cortex and anterior cingulate that generally activate in fMRI studies. The reasons for these discrepancies remain unclear; they are unlikely to be due to different imaging modalities but suggest, as do the variable cognitive and physiologic data, that conversion disorder may arise from a composite of interconnected etiologic factors that vary across subjects.

Imaging only the motor system presents additional challenges, because it comes with uncertainty as to the subject's volition in relation to the abnormal movements. Is the flaccid limb an involuntary manifestation of conversion or a conscious willed refusal to move, as in feigning? In patients deemed to have conversion paralysis, the findings mirror the data from sensory conversion, albeit with a different region of localization, namely the hypoactivation is now seen in the motor cortex. The pattern of ancillary activations remains fairly consistent,[24] but when it comes to feigning motor symptoms, fMRI studies have demonstrated differences in cerebral activation between patients deemed to have conversion and healthy control subjects who were instructed to simulate paralysis. The imaging findings, however, lack consistency.[25–27]

Finally, a novel fMRI study that looked at how people with conversion disorder process affective stimuli revealed increased connectivity between the right amygdala and right supplementary motor cortex, hinting at a mechanism that could explain how environmental stressors trigger conversion symptoms in some individuals.[28]

PROGNOSIS

A number of methodologic limitations should be kept in mind when assessing how outcome is determined. The conclusion, nevertheless, is that the outcome is generally unfavorable. Symptoms frequently remain the same or worsen with time. Early diagnosis and young age are the two bright prognostic lights.[29] Symptom longevity is considered an ominous sign.[30] The 12-month follow-up data on 717 of 1144 people with "neurologic symptoms unexplained by disease" showed that two-thirds of the sample were unchanged, worse, or much worse.[31] Predictors of poor outcome were patients' beliefs (expectations of nonrecovery), attribution of symptoms to physical rather than psychiatric factors, and the receipt of illness-related financial benefits. Collectively, these three variables could only explain 13% of the variance in outcome.

Adding weight to this unfortunate outcome is the presence of significant psychiatric comorbidity. A 3-year follow-up (standard deviation 2.2; range 1 to 7 years) of 88 patients with motor conversion symptoms revealed a lifetime prevalence of 43% and 62% for major depression and anxiety disorders, respectively, with an additional 27% displaying comorbid depression and anxiety added to these percentages.[30] Almost half the group had personality disorders, although their prognostic role has been questioned by others.[29] The psychosocial impact of all this psychopathology can be considerable too, as revealed in the Scottish Neurological Symptoms Study.[32] A comparison of people with unexplained versus confirmed "organic disease" revealed that the former were

more likely to have stopped work for medical reasons and were consequently in receipt of more disability-related financial benefits.

Some cause for greater optimism has, however, emerged from a recent meta-analysis of treatment outcomes in people with psychogenic nonepileptic seizures. Results from a pooled sample of 228 participants who had received diverse forms of psychological interventions (cognitive-behavioral therapy, psychodynamic therapy, paradoxical intention therapy, and mindfulness, among others) revealed that almost half the sample were seizure free on completion of treatment. Whether this improvement is maintained over time, however, remains unclear.[33]

TREATMENT

Given the somber prognostic data, it should come as no surprise that treatment of conversion disorder can prove challenging. Probably the best evidence comes from cognitive-behavioral therapy. A pilot randomized controlled study of 66 people with nonepileptic seizures found that cognitive-behavioral therapy was superior to standard medical care given over a 4-month period.[34] Encouraged by these results, the authors proposed embarking on a multicenter randomized controlled trial and published their proposed protocol, which included enrolling a sample of close to 300 people with nonepileptic seizures and randomly assigning them to cognitive-behavioral therapy plus standard medical care or standard medical care alone.[35] The results are awaited. The rationale for a more definitive cognitive-behavioral therapy trial is supported by a review of treatment options for the somatoform disorders, a broad spectrum of somaticizing conditions that includes conversion.[36] The study identified 34 randomized controlled trials, 13 of which involved cognitive-behavioral therapy, with efficacy reported in nine of these.

Variants of cognitive-behavioral therapy have been tried in different settings. For example, an innovative study with an eye on the practicalities of administering therapy with limited resources provides additional, albeit indirect, evidence of cognitive-behavioral therapy efficacy. Here the focus fell on cognitive-behavioral therapy–guided self-help, which was conveyed to participants in a manual and four half-hour education sessions. When this intervention was compared to usual care in a randomized controlled trial design, it was found to be more effective in reducing subjective health concerns.[37] Another approach has been to focus on training primary care providers in how best to manage the patient with unexplained medical symptoms. The rationale for this is twofold: patients with these disorders often present first to their primary care providers, who may find it frustrating to deal with them, thereby potentially missing the diagnosis or providing ineffective treatment. To assess whether this proved effective, a study was devised using a cognitive-oriented educational program for assessment, treatment, and management of patients with these unexplained symptoms. The primary outcome measure was the degree to which doctors changed their attitudes toward their patients. The intervention proved effective: 12 months after training, the general practitioners felt less anxious and more comfortable in their patient interactions.[38] However, a subsequent randomized controlled trial failed to demonstrate that the training translated into benefits for the somaticizing patients across an array of outcome measures that included quality of life, disability days, and patient satisfaction.[39] On the other hand, a different approach with family doctors that incorporated

KEY POINTS

- Favorable prognostic signs are early diagnosis and younger age, whereas poor prognostic signs include symptom longevity, attribution of symptoms to physical rather than psychological beliefs, expectations of nonrecovery, and the receipt of illness-related financial benefits.
- Limited treatment data suggest that cognitive-behavioral therapy can be effective, although findings from a proposed large multicenter randomized controlled trial are still awaited.

KEY POINTS

- Training geared toward improving the attitude of primary care providers to patients with conversion disorder may improve outcomes.
- Much remains unknown about conversion disorder, but advances in neuroscience are starting to provide key insights into the functional neuroanatomy of the condition and which therapies work best.

reattribution training produced some modest benefits. Based on a core principle that somaticizing behavior, which includes conversion disorder, entails misattribution of symptoms, an 8-hour skill-based training offered to primary care providers was geared toward helping patients correct their faulty belief systems. When applied by primary care providers, the results led to positive shifts in patients' thinking but, interestingly, did not change the incidence of investigations recommended by the primary care providers, their drug prescriptions for the disorder, and their referrals for specialist consultation.[40]

A miscellany of other approaches has been tried as well, with varied effects. While individualized tailored psychotherapy offered some cost-effective benefits,[41] perhaps the most promising intervention is repetitive transcranial magnetic stimulation. This proved effective in 89% of 70 participants with psychogenic paralysis, although it should be noted that the study had no placebo arm.[42] A single case study of psychogenic aphonia did, however, have a built-in placebo. Stimulation of the left dorsolateral prefrontal cortex had no therapeutic effect, whereas shifting the stimulus to the right motor cortex produced symptom resolution within days.[43]

Irrespective of which treatment modality is chosen, some basic management principles should be observed. When a neurologist suspects the diagnosis is conversion, the first step in managing the problem is not to lose the patient's trust. Therefore, validating the patient's symptoms is important. The next step will depend on a number of factors, including the relative acuity of the symptoms and the comfort of the neurologist in treating them. If the symptoms are no more than a few months old, are not incapacitating, and are clearly linked to an identifiable, potentially modifiable stressor, and the patient seems psychologically aware and open to reassurance that the symptoms can resolve, then the neurologist may proceed by recommending practical steps to deal with the stressor while emphasizing the importance of remaining physically and socially active. In more complex situations, a speedy referral to a psychiatrist or psychologist is recommended; however, when doing so, the neurologist must endeavor to retain the confidence of the patient in the medical system. A hasty cavalier referral to a mental health specialist without adequate explanation of why the referral is being made (including a discussion with the patient of conversion disorder as a brain disorder, albeit one best managed by a psychiatrist) runs the risk of alienating the patient from the medical system and complicating the work of the psychiatrist to come.

CONCLUSION

Conversion disorder remains a challenge for the medical profession. While diagnostic accuracy and consistency are reassuring, much remains to be clarified. Future research geared toward a better understanding of a complex etiology will undoubtedly benefit from advances in technology and neuroscience, but as knowledge inches forward, the most pressing issue remains treatment. The results of the proposed multicenter randomized controlled trial are eagerly awaited, while the full therapeutic potential of repetitive transcranial magnetic stimulation has yet to be realized. From this perspective, the future looks optimistic.

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