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Inside-out and up-down side: Conversion Disorder and Physiatry

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Clinical Vignette

PM is a 57-year-old right-hand-dominant female with COPD, hypertension, anxiety, chronic back pain, prior C3-C4 ACDF and lumbar surgeries, who presented to the emergency department with a 10-day history of left-sided numbness, weakness, and with difficulty swallowing. She recalled similar symptoms five months ago, diagnosed with a transiet ischemic attack, and reported that her left side never returned to normal. Pertinent laboratory results revealed mildly elevated white count of 11.9, normal troponin, and COVID-19 testing was negative. Head CT scan with no evidence of infarct, hemorrhage, or mass. PM's symptoms improved in the emergency department with National Institute of Health stroke scale (NIHSS) recorded as one for left leg drift. After discussing with the patient's primary care physician, she was admitted for further work-up and management.

On day two, neurology was consulted, noted give-away weakness throughout and recommended additional work-up. Brain MRI and carotid dopplers were normal. Cervical spine CT was consistent with surgical changes at the prior C3-4 discectomy without evidence of complications. Transthoracic echocardiogram showed an ejection fraction of 60–65% with normal left ventricular size and no patent foramen ovale.

On day three, physical therapy documented inconsistencies in strength and function. PM ambulated 12 feet without an assistive device and required minimal assistance. OT assessment revealed that she could not obtain items as directed, needing moderate assistance in basic ADLs despite antigravity strength in the left upper limb and normal strength in the right upper limb. Additionally, even with good overall strength and patchy decreased light touch without proprioceptive loss, she had notable difficulty maintaining upright posture.

On day four, she was transferred to the UPMC Presbyterian inpatient rehabilitation unit for a multidisciplinary approach to address current functional deficits.

Terminology and Classification

The word hysteria has been a taxonomy challenge for centuries and was removed from the Diagnostic and Statistical Manual of Mental Disorders (DSM) in 1968.[1] DSM-IV, published in 1994, created the somatoform disorders that were controversial and underwent a major change with DSM-5 in 2013. This classification created the somatic symptoms and related disorders category (SSD).[2] SSD includes somatic symptom disorder, illness anxiety disorder (formerly known as hypochondriasis), conversion disorder (unchanged from DSM-IV), and factitious disorder. One very important point is that SSD can now be explained by a medical condition, whereas symptoms of somatoform disorder were always considered medically unexplained. The criterion is that the individual has a maladaptive reaction to a somatic symptom.[3]

Conversion disorder (CD), also known as functional neurologic symptom disorder (FND), is a disease entity that is not referable to an organic neurologic or medical etiology.[4] The presentation may involve sensory and motor deficits as well as nonepileptic seizures suggesting neurologic dysfunction. However, comprehensive neurologic examination, imaging studies, and lab testing are normal.

There are several types of conversion disorder, each distinguished by the predominant symptomatology: motor, sensory, pseudo-seizures, and mixed presentation. Abnormal involuntary movements that improve with distraction or nonphysiologic maneuvers, previously referred to as psychogenic movement disorders, are now described as functional movement disorders or FMD.[5,6]

Epidemiology

The annual incidence of conversion disorder varies widely.[7] In two studies, the prevalence rate of CD was found to be 1-3% of the general population.[8,9] In contrast, a survey of general practitioners showed 18 patients in a catchment area of 37,000 fulfilling criteria for conversion disorders.[10] Carson et al. (2003) found that 30% of patients at a neurology clinic had "medically unexplained symptoms."[11] Studies within a community hospital setting have estimated that 20%–25% of patients have symptoms of CD, with 5% meeting strict criteria.[12,13] CD is more prevalent in rural areas and affects individuals with lower socioeconomic status, less education, and low psychological sophistication.[14] An increased rate of conversion disorder is well documented in patients with a past history of sexual or physical abuse. Overall, the female:male ratio is 2-10:1 and typical onset is the second to fourth decades.[8,10,15,16]

Pathophysiology and Etiology

The pathogenesis and exact cause of CD remains poorly understood. Similar to other disorders, genetic, neurobiological, and psychosocial factors are involved. Since 1935, the term "conversion" comes from Freud's psychodynamic theory, which theorizes that an individual converts intrapsychic conflict into physical phenomena to reduce stress.[17] Emotional conflict is repressed into the unconscious mind as a defense mechanism and is converted into somatic symptoms. In other psychodynamic models, inadequate coping mechanisms and negative interpersonal relationships that develop when one is younger, may reemerge later in life during another traumatic event or stressor.[18] Thus, a hallmark principle of CD is that signs and symptoms are not consciously or deliberately manifested.

Cognitive-behavioral models hypothesize that exposure to information related to a specific symptom can lead to the creation of representation in memory. CD occurs when this representation is "activated" by an individual worrying excessively about or looking for signs of the symptom, such as what occurs during psychogenic non-epileptic seizures.[18]

Functional neuroimaging techniques have shed some light on the neural basis of CD. Marshall et al. used PET scans with a CD patient who manifested left-sided paralysis and found significant activation indexed by changes in regional cerebral blood flow in the right anterior cingulate and orbito-frontal cortex.[19,20] Atmaca et al. showed reduced volumes of bilateral basal ganglia and right thalamus in CD compared to people without the disorder.[21,22]

In a study of seven patients with unilateral sensorimotor deficits, Vuilleumier utilized single photon emission computerized tomography (SPECT) that revealed reduced blood flow in the contralateral thalamus and basal ganglia which resolved after recovery of symptoms. This suggests that striato-thalamo-cortical pathways, which may have a role in emotional response modulation associated with motor activity, could be inhibited by emotional stressors and may be involved in CD.[23]

In 2016, a review by Ejareh and Kanaan of convergent functional neuroimaging including functional MRI (fMRI), SPECT, and positron emission tomography (PET), showed alterations in brain circuits but no conclusive theory as to the etiology of CD. Problems included small sample size, divergent symptoms, and paradigm heterogeneity.[24]

Boeckle et al. undertook the first meta-analysis of neuroimaging on motor CD, suggesting functional differences in multiple brain areas between patients with motor CD and healthy controls. Areas of interest repeatedly identified as the possible core

networks for motor CD included dorsolateral and medial prefrontal cortex, superior frontal gyrus, insula, amygdala, and dorsal anterior cingulate cortex.[25]

In a review article by Feinstein in 2011, he cited an fMRI study (Figure 1) on a patient with unilateral sensory loss. Imaging showed normal activation of the contralateral somatosensory region with stimulation of sensate side. However, there was no similar activation of the somatosensory cortex with stimulation of the affected side, but instead activity was seen in the orbitofrontal and anterior cingulate regions. Other studies on motor conversion disorders have had similar results.[26] These findings show an association between CD and areas of the brain involved in emotional regulation. Figure 2 illustrates this further in a schematic representation.

Diagnosis

Conversion disorder is characterized by the American Psychiatric Association as the presence of one or more symptoms of altered voluntary motor or sensory function in the absence of objective clinical findings suggestive of a recognized neurological or medical condition, or mental disorder. Additionally, the symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Table 1).[2] CD symptoms are unconscious or involuntary thus distinguishing it from malingering or factitious disorders which are intentional and deliberate (see Table 2 on page 4).[26,28] Importantly, patients perceive symptoms to be real and not merely explained by psychological factors.[29] Furthermore, persons with CD have similar illness beliefs as those with identifiable neurologic disease.[30]

Table 1: DSM-5 Diagnostic Criteria for Conversion Disorder

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.
Specify symptom type:
• With weakness or paralysis • With abnormal movement (e.g., tremor, dystonic movement, myoclonus, gait disorder) • With swallowing symptoms • With speech symptoms (e.g., dysphonia, slurred speech) • With attacks or seizures

Source: (Copyright © 2013). American Psychiatric Association. All Rights Reserved.[2]

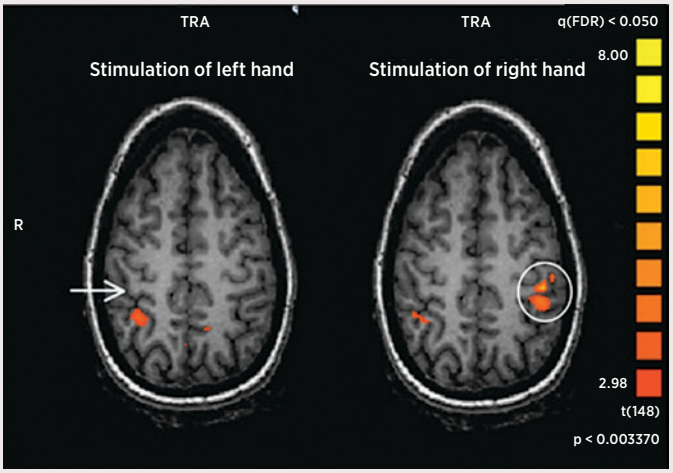

Figure 1: Functional magnetic resonance image showing somatosensory activity evoked by stimulation in a patient with sensory conversion disorder affecting the left hand. When the patient's left hand was stimulated, no activity was seen in the primary somatosensory cortex (arrow). However, increased activity was seen in this area of the brain when the patient's right hand was stimulated (circle).

Source: Feinstein A. Conversion Disorder: advances in our understanding. CMAJ 2011. DOI:10.1503.[26]

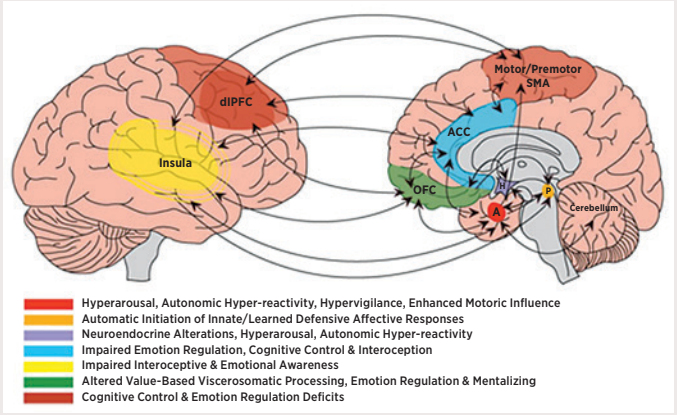

Figure 2: Schematic representation of key regional abnormalities and emerging functional connectivity alterations in FND during emotional processing. Main findings from the literature include heightened limbic (amygdalar) and motor (SMA/PMA/M1, cerebellum) activations; enhanced limbic-motor network functional connectivity; altered prefrontal and paralimbic (ACC, dlPFC, OFC, insula) engagement and elevated functional connectivity with the motor system. A, amygdala; ACC, anterior cingulate gyrus*; dlPFC, dorsolateral prefrontal cortex; H, hypothalamus; OFC, orbitofrontal cortex*; P, periaqueductal grey; SMA, supplementary motor area. *Both ACC and OFC project to periaqueductal grey and hypothalamus.

Source: Pick S, Goldstein LH, Perez DL, Nicholson TR. Emotional processing in functional neurological disorder: a review, biopsychosocial model and research agenda. Journal of Neurology, Neurosurgery, and Psychiatry. 90(6).[27]

Table 2. Differentiating CD From Factitious Disorder and Malingering		
Psychological Basis for the Symptoms of Conversion Disorder[a]		
Type of Disorder	**Symptom Production**	**Motivation**
Conversion disorder	Unconscious	Unconscious
Factitious disorder	Conscious	Unconscious
Malingering	Conscious	Conscious

Source: Haque R, Alavi Z. Mr. Smith is falling every day: conversion disorder in an elderly man. Annals of Long-Term Care: Clinical Care and Aging. 2012;20(11):30-35.[28]

Though the diagnosis of conversion disorder requires ruling out other causes first, thus potentially prolonging the time to diagnosis, Joustra et al. observed that symptom burden increased during the first four weeks but showed little change thereafter. Using somatic symptom questionnaires, the four-week period after onset of symptoms best predicted quality of life and health anxiety.[31]

CD may mimic seizures and weakness from various causes and requires full neurologic work-up to exclude these conditions. Hoover's sign is a clinical examination maneuver to assess inconsistent lower extremity weakness (Figure 3).[32] Table 3 provides a summary of the more common clinical features of conversion disorder.

Sar et al. report that dissociative disorders were present in almost 50% of CD subjects.[33] Dissociative disorders involve problems with identity, emotion, memory, behavior and perception, which disrupts mental functioning. In DSM-5, FND includes dissociative convulsions or pseudoseizures, dissociative anaesthesia and sensory loss and dissociative motor disorder.

It is necessary to rule out organic disease, which by definition is disease accompanied by structural changes in organs or tissues, and a diagnosis of CD can be made when there are inconsistencies clinically. In a 2005 review, Stone noted that an organic condition is diagnosed in about 4% of subjects.[31]

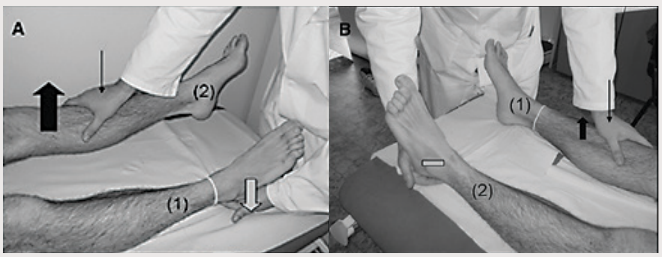

Figure 3: Hoover's sign. (**A**) An involuntary extension of the paretic leg [(**1**) marked with a white elastic] is perceived by the examiner's hand placed under the heel when the contralateral limb (**2**) is forced to flex against resistance (positive Hoover's sign); (**B**) the unperceived downward pressure of the strong heel (**2**) when trying to lift the weak leg (**1**) represents the complementary way of recording the information. Often this part of the sign is overlooked. Black arrow, voluntary movement; gray arrow, involuntary movement; gray bar, lack of involuntary movement; thin arrow, resistance applied by the examiner.

Source: Tremolizzo L, et al. Positive signs of functional weakness, J Neurol Sci (2014), http://dx.doi.org/10.1016/j.jns.2014.03.003.[32]

Table 3. Typical Signs and Symptoms of CD and Differentiating Features	
Symptom	**Distinguising Features and Presentation**
Blindness	In conversion disorder, the patient, though complaining of recent onset of blindness, neither sustains injury while maneuvering around the office nor displays any expected bruises or scrapes. The pupillary reflex is present, thus demonstrating the intactness of the optic nerve, chiasm, tract, lateral geniculate body, and mesencephalon.
Deafness	In conversion deafness, the blink reflex to a loud and unexpected sound is present, thus demonstrating the intactness of the brain stem.
Psychogenic nonepileptic seizures	Patients with psychogenic nonepileptic seizures generally lack response to treatment with antiepileptic drugs or have a paradoxical increase in seizures with antiepileptic drug treatment. The negative history of injury or loss of control of bladder or bowel during the seizure episode is also significant.
Tremor	When weights are added to the affected limb, patients with functional tremor tend to have greater tremor amplitude, whereas in those with organic tremor, the tremor amplitude tends to diminish.
Dystonia	Useful distinguishing features include an inverted foot or "clenched fist," adult onset, a fixed posture that is apparently present during sleep, and the presence of severe pain.
Paralysis	In conversion paralysis, the patient loses the use of half of his or her body or of a single limb, but the paralysis does not follow anatomical patterns and is often inconsistent upon repeat examination.
Syncope	The conversion patient may report feeling faint or fainting, but no autonomic changes are identified, such as pallor, and there is no associated injury. In addition, the fainting spells have a "swooning" character to them, heightening the drama of these events.
Aphonia	Conversion aphonia may be suspected when the patient is asked to cough, for example, during auscultation of the lungs. In contrast with other aphonia, the cough is normally full and loud.
Anesthesia	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.
Paraplegia	In conversion paraplegia, one finds normal, rather than increased, deep tendon reflexes, and the Babinski sign if absent. In doubtful cases, the issue may be resolved by demonstrating normal motor evoked potentials.

Source: Ali S, Jabeen S, Pate RJ, et al. Conversion disorder — mind versus body: a review. Innov Clin Neurosci. 2015 May-Jun;12(5-6):27-33.[5]

Functional assessment is part of the examination. Close observation of strength, mobility and gait will show inconsistent and atypical patterns which are nonphysiologic and do not conform to sensorimotor impairment. There may be impaired insight and judgement as well as incongruent emotional responses.[34]

Treatment

There is no gold standard treatment and there remains a persistent lack of evidence on the therapeutic approach for CD. Large, randomized controlled trials are lacking and only small, retrospective studies show evidence for current therapies. Symptoms of CD may improve over time even without formal treatment in certain patients. In those with more severe symptoms, treatment may be required depending on presenting signs and symptoms.

The essential first step is presenting the diagnosis in a tactful way, which is typically not revealed during the initial encounter. Establishing a strong therapeutic alliance and establishing a goal-oriented treatment program is key to success.[5,18]

Gelauff et al recommend a three-stage approach for the treatment of functional motor disorder (Figure 4). In Step 1, the patients are taught that they have a genuine disorder and are encouraged to identify triggers such as trauma or pain. Patients are then referred to PT for intervention (Step 2) in which patients further learn about the nature of their abnormal movements and undergo motor retraining. Step 3 involves more complex multidisciplinary care which may include a combination of the rehabilitation team, psychologist, psychiatry, and neurologist. Other modalities such as hypnosis and transcranial magnetic stimulation may help a subset of patients.[35]

In the rehabilitation setting, the same principle holds true for a multidisciplinary collaborative approach in the treatment of CD. Physiatrists traditionally treat this disorder like organic based disease with motor retraining and behavioral modification modalities. CBT and insight-oriented psychotherapy with concurrent conventional rehabilitation therapies is beneficial for those with CD. Effective symptom management centering on functional improvement is the focus of the rehabilitation program.[34]

A graded approach to activity that responds to a specific patient's activity tolerance may allow better goal attainment.[29] Physical therapy can minimize abnormal gait or motor symptoms in

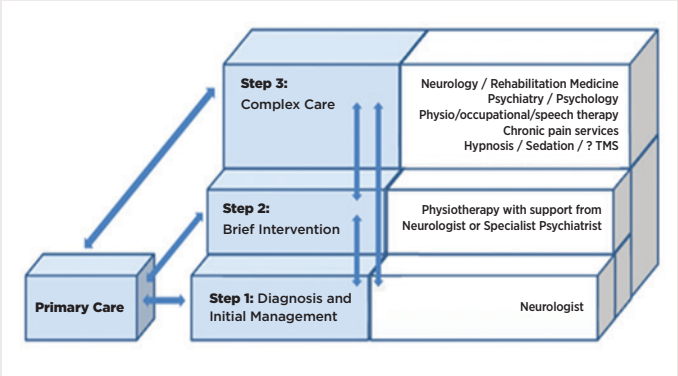

Figure 4: Stepped care approach for functional motor disorder. Note that patients may need to go back to Step 1 from Step 2 or 3 if this has not been successful. (Adapted from Health Improvement Scotland)

Source: Gelauff JM, Dreissen Y et al. Treatment of Functional Motor Disorders. Current Treatment Options in Neurology. 2014 February;16:286.[35]

patients with CD. In a prospective cohort study of 47 patients by Nielsen et al, patients with functional motor disorder showed significant improvement in physical functioning. There are four primary areas of PT intervention, as follows: 1) education, 2) demonstration that normal movement can occur, 3) retraining movement with diverted attention, and 4) changing maladaptive behaviors related to symptoms. To restore function and movement, therapists utilize specific techniques such as graded exercise, visualization, mirrors and video for feedback, desensitization, adaptive aids and electrotherapies.[36,37]

In 2011, the Dutch Multidisciplinary Guideline for Medically Unexplained Symptoms and Somatoform Disorders was published to derive a consensus for evidence-based treatment. The most important recommendation was effective patient-doctor communication for diagnosis and treatment. Additionally, while low risk patients may be effectively treated with patient education and reassurance, moderate risk patients with psychological comorbidities should be co-managed with mental health experts who can incorporate techniques like CBT or mindfulness-based therapies. Higher risk patients may need specific psychiatric consultation.[38]

In most patients with CD, psychotherapy is the first-line treatment. Cognitive-behavioral therapy (CBT) is the most effective method cited in literature. CBT focuses on modifying dysfunctional thoughts and illness beliefs about conversion symptoms and changing problematic behavioral responses.[39]

Behavioral interventions improve self-confidence and help patients understand their psycho-emotional deficits. In a 1996 retrospective study of CD patients treated during inpatient rehabilitation, Speed concluded that behavioral treatment is effective with long-lasting symptom resolution, although clinically but not statistically significant.[18,40]

There is no specific pharmacologic therapy for CD. However, comorbid psychiatric illness can effectively be treated with pharmacotherapy and improve CD. Medications include antidepressants, anxiolytics and mood stabilizers as well serotonin and norepinephrine reuptake inhibitors if pain is a consideration. Antipsychotics such as quetiapine and haloperidol have been investigated in several studies, and less extrapyramidal symptoms were documented using quetiapine although outcomes were similar.[5,18]

Prognosis

Good prognostic signs include acute onset, early diagnosis, short duration of symptoms, lack of comorbid psychiatric disorders, clearly identifiable stressors and strong patient-clinician alliance. The prognosis is generally poorer for patients with prolonged symptoms greater than one-year, increased number of physical symptoms and poor physical functioning prior to diagnosis. Some patients may experience a return of their symptoms after a stressful event within a year of symptom onset, so ongoing clinical monitoring may be valuable despite the resolution of symptoms.[5,18,26]

Clinical Vignette Outcome

On presentation to the UPMC Presbyterian inpatient rehabilitation unit, PM reported a longstanding history of taking hydrocodone for back pain that was managed by her primary care physician. She also revealed worsening anxiety attacks for at least a year. She was on maintenance duloxetine for chronic pain and took alprazolam for many years. She perseverated on "strange" spasms in her throat and she was fearful of complications from remote cervical spine surgery, both of which further aggravated anxiety. She endorsed episodes of urinary incontinence but was continent of bowel. On examination, she had decreased light touch in the left hemi-body, fair strength in the left upper limb and poor strength in the left leg. Over the next several days, she demonstrated fluctuating strength on manual muscle testing especially when she was observed at a distance.

The rehab team provided support and positive feedback frequently. She fully participated in a program of activities of daily living, mobility and gait training. A family meeting was held initially with PM's daughter and later with the patient included. The main topic of discussion was that the left hemiparesis was not due to a stroke and broached the subject of conversion disorder. Treatment included management of anxiety, psychiatry consultation, and education on her condition. Changes in medications included a rapid cross-taper from duloxetine to escitalopram and hydroxyzine as needed, instead of benzodiazepine. She was agreeable to pursuing outpatient behavioral health treatment post-discharge. Due to great concerns about neck pain, orthopaedic surgery was consulted and cervical spine MRI revealed moderate canal stenosis without cord signal changes. No surgical intervention was recommended, which was reassuring for the patient. PM continued to receive hydrocodone twice daily which controlled her chronic pain. Urinary continence was achieved with timed voids. On discharge, sensorimotor exam was improved in the left hemi-body. Motor testing remained inconsistent and intermittent give-away weakness resolved with distraction. Functionally, she attained modified independent status with ADLs, IADLs, transfers and community distance ambulation without a device on level and uneven terrain.

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A $21 billion health care provider and insurer, Pittsburgh-based UPMC is inventing new models of patient-centered, cost-effective, accountable care. The largest nongovernmental employer in Pennsylvania, UPMC integrates more than 90,000 employees, 40 hospitals, 700 doctors' offices and outpatient sites, and a 3.9 million-member Insurance Services Division, the largest medical insurer in western Pennsylvania. In the most recent fiscal year, UPMC contributed $1.4 billion in benefits to its communities, including more care to the region's most vulnerable citizens than any other health care institution, and paid more than $800 million in federal, state, and local taxes. Working in close collaboration with the University of Pittsburgh Schools of the Health Sciences, UPMC shares its clinical, managerial, and technological skills worldwide through its innovation and commercialization arm, UPMC Enterprises, and through UPMC International. *U.S. News & World Report* consistently ranks UPMC Presbyterian Shadyside among the nation's best hospitals in many specialties and ranks UPMC Children's Hospital of Pittsburgh on its Honor Roll of America's Best Children's Hospitals. For more information, go to UPMC.com.

ABOUT THE DEPARTMENT OF PHYSICAL MEDICINE AND REHABILITATION

- UPMC is proud to have two hospitals* rated high performing for rehabilitation by *U.S. News & World Report*, with both among the top 20 hospitals rated.

  *UPMC Presbyterian Shadyside and UPMC Mercy are rated as high performing for rehabilitation by *U.S. News & World Report*.
- The Department of Physical Medicine and Rehabilitation is consistently a top recipient of NIH funding for rehabilitation-related research.
- The Spinal Cord Injury Program at UPMC is one of only 14 in the country selected by the National Institute on Disability, Independent Living, and Rehabilitation Research (NIDILRR) as a model for other rehab providers.
- Department clinicians lead UPMC's rehabilitation network of more than 90 inpatient, outpatient, and long-term care facilities — one of the country's largest.

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For consults and referrals, please call UPMC's 24-hour physician OnDemand service at **1-866-884-8579**.