Published in final edited form as:

Gen Hosp Psychiatry. 2015 ; 37(1): 89–93. doi:10.1016/j.genhosppsy.2014.10.009.

Catatonia in Resource Limited Settings: A Case Series and Treatment Protocol

Stephanie L. Smith, M.D.^{1,2,3}, David J. Grelotti, M.D.^{2,4}, Reginald Fils-Aime, M.D.⁵, Eugenie Uwimana, B.A.⁶, Jean-Sauveur Ndikubwimana⁶, Tatiana Therosme, B.A.⁵, Jennifer Severe, M.D.⁵, Dominique Dushimiyimana⁶, Clemence Uwamariya⁶, Robert Bienvenu⁶, Yoldie Alcindor, B.A.⁵, Eddy Eustache, M.A.⁵, Giuseppe J. Raviola, M.D.^{1,2}, and Gregory L. Fricchione, M.D.⁷

¹Department of Global Health and Social Medicine, Harvard Medical School, 641 Huntington Avenue, Boston, MA, USA 02115 ²Partners In Health, 888 Commonwealth Avenue, 3rd Floor, Boston, MA USA 02215 ³Division of Medical Psychiatry, Brigham and Women's Hospital, 75 Francis Street, Boston, MA USA 02115 ⁴Department of Epidemiology, Harvard School of Public Health, 677 Huntington Avenue, Boston, MA, USA 02115 ⁵Department of Mental Health and Psychosocial Services, Zanmi Lasante, Haiti ⁶Butaro Hospital, Ministry of Health, Burera District, Rwanda ⁷Division of Psychiatry and Medicine, Pierce Division of Global Psychiatry, Massachusetts General Hospital, 55 Fruit Street, Boston, MA USA 02114

Abstract

Objective—The Catatonic Syndrome (“catatonia”) is characterized by motor and motivation dysregulation and is associated with a number of neuropsychiatric and medical disorders. It is recognizable in a variety of clinical settings. We present observations from the treatment of four individuals with catatonia in Haiti and Rwanda, and introduce a treatment protocol for use in resource limited settings

Methods—Four patients from rural Haiti and Rwanda with clinical signs of catatonia and a positive screen using the Bush-Francis-Catatonia Rating Scale were treated collaboratively by general physicians and mental health clinicians with either lorazepam or diazepam. Success in treatment was clinically assessed by complete remittance of catatonia symptoms.

Results—The four patients in this report exhibited a range of characteristic and recognizable signs of catatonia, including immobility/stupor, stereotypic movements, echophenomena, posturing, odd mannerisms, mutism, and refusal to eat or drink. All four cases presented initially to rural outpatient general health services in low resource settings. In some cases, diagnostic

Address correspondence to: Stephanie Smith, M.D., Department of Global Health and Social Medicine, 641 Huntington Avenue, Boston, MA 02115, USA, Phone: +1 617 432 1707, Fax: +1 617 432 2565, Stephanie_Smith@hms.harvard.edu.

Ethical Considerations

Written consent to publish was obtained from all patients in the case series. This series was reviewed by the Harvard University Faculty of Medicine Committee on Human Studies, the National Health Research Council of Rwanda, and the Zanmi Lasante (ZL) Ethics Committee in Haiti, and deemed exempt by the Institutional Review Board of Harvard Medical School, the Rwandan National Ethics Committee, and the ZL Ethics Committee.

uncertainty initially led to treatment with typical antipsychotics. In each case, proper identification and treatment of catatonia with benzodiazepines led to significant clinical improvement.

Conclusion—Catatonia can be effectively and inexpensively treated in resource limited settings. Identification and management of catatonia is critical for the health and safety of patients with this syndrome. Familiarity with the clinical features of catatonia is essential for health professionals working in low resource settings. To facilitate early recognition of this treatable disorder, catatonia should feature more prominently in global mental health discourse.

Keywords

catatonia; Africa; Caribbean region; Psychosomatic Medicine; neuropsychiatry; world health

Introduction

The Catatonic Syndrome (“catatonia”) was described as a movement and motivation dysregulation syndrome with multiple medical and psychiatric etiologies well over one hundred years ago [1]. In its classic form, catatonia is characterized by stupor, negativism, withdrawal, and/or mutism, less frequently with phases of excitement and/or autonomic instability. The international prevalence of catatonia likely reaches up to 18% of patients with psychiatric disorders [2], about the prevalence of HIV-seropositivity among patients with tuberculosis worldwide [3]. Although catatonia occurs with multiple neuropsychiatric and medical disorders [4], initial reports linked catatonia solely with schizophrenia, leading to a nosologic error and the prevailing assumption throughout the twentieth century that catatonia denotes schizophrenia [5]. Consequently, catatonia was often treated with antipsychotics, sometimes harmfully. As catatonia has slowly been reclassified in the psychiatric literature as a disorder of movement and motivation secondary to many causes, screening tools and rapidly effective treatments have been articulated to assist clinicians in the diagnosis and treatment of catatonia. The reflexive use of anti-psychotic medications has somewhat abated.

In well resourced health systems, the identification and management of catatonia primarily occurs in specialized psychiatric facilities or in general medical settings with close psychiatric consultation. In resource limited settings, however, a model of care whereby the treatment of catatonia requires specialized psychiatric services may not be feasible. The majority of low income countries worldwide have fewer than one psychiatrist per 100,000 people [6]. Most care for mental disorders is provided by non-specialist health professionals, and referral to specialized psychiatric services are often limited or non-existent. In these settings, recognizing and treating catatonia can be challenging for general clinicians who may have limited training in neuropsychiatric disorders. Yet if not properly treated, catatonia carries the risk of significant morbidity and mortality. [7]

We describe four patients in Haiti and Rwanda in whom explicit recognition of catatonia symptoms and treatment of catatonia with benzodiazepines were associated with complete symptom resolution and return to, or improvement in, baseline functioning level. All four patients presented to general medical services and were primarily managed by non-specialists in collaboration with mental health professionals. Our experience highlights the

need for decision tools designed to assist in the management of catatonia in areas where psychiatric resources are limited. To this end, we articulate a treatment algorithm for use by non-mental health professionals in both outpatient and inpatient general medical settings.

Methods

Four non-consecutive patients with catatonia were identified by retrospective chart review, two from Rwanda and two from Haiti. Patients included in this series met clinical criteria for catatonia and screening on the Bush-Francis Catatonia rating scale, and received either lorazepam or diazepam as first line treatment for catatonia. In all four cases, treatment was provided by non-specialist clinicians in collaboration with mental health professionals. Success in treatment was determined by a complete remission of catatonia symptoms.

Results

Case One

H is a thirty two year-old man in Haiti with a distant history of a psychotic illness, who initially presented to rural outpatient general medical services with “a loss of joy in life.” For approximately one year prior to evaluation, the patient’s family reported that he rarely spoke spontaneously, rarely responded to their questions, often stared, ate and slept very little, and spent a great deal of time in the evening in the kitchen purposelessly making noise. H had no other history of mood or anxiety symptoms, medical problems, or a family history of mental illness. H was seen by two general medicine clinicians, one who was at a loss to explain his condition and another who initially thought that H was depressed.

On exam with general and mental health outpatient services, H was hypoactive, sitting and standing for long periods without activity. He would walk when commanded by his father and would repeat or mimic the actions of others. He had an intense stare, waxy flexibility and maintained postures for greater than ten minutes before being reposed into a normal relaxed position. A physical and neurological exam was otherwise normal. On the Bush Francis Catatonia Rating Scale (BFCRS), he had 8/14 items on screening. Available labs including HIV and syphilis serologies and a complete blood count, were negative. A head CT scan showed only nonspecific diffuse volume loss.

H was started on oral lorazepam 1 mg three times daily for catatonic symptoms. His speech and movement improved after two days and the patient began describing auditory hallucinations and delusions about God’s wish to provide him an ideal woman. Three days after starting lorazepam, risperidone 0.5 mg twice daily was started for psychosis while lorazepam was maintained. Although largely resolved by day three, the patient’s catatonia symptoms were completely resolved by the second outpatient visit two weeks later.

Case Two

F is an eighteen year-old high-school student with no psychiatric history who was originally admitted to a general hospital in Haiti because of “problems with her head” including one week of the abrupt onset of “silly behavior”, reduced sleep, and constant, purposeless motor unrest. There was no report of trauma, drug use, or alcohol use. The admitting general

physician checked available labs including complete blood count, syphilis and HIV serologies, and thinking that that her disorganized behavior resulted from psychosis, initially prescribed haloperidol 5 mg orally twice daily, with diazepam 5 mg orally at night.

F left the hospital after one week and discontinued all medications because her family wished to pursue traditional treatments. F returned to outpatient primary care services one week after her discharge with no symptomatic improvement, at which point a mental health clinician was consulted. F's parents reported that in the hospital, her symptoms had worsened slightly on haloperidol, although the diazepam had been helpful for sleep. On exam, F was hyperactive, spoke in fragmented sentences, exhibited significant motor unrest and impulsivity, purposelessly touching things in the room, exaggerating normal movements, mimicking others' actions, and walking on tip toes. On the BFCRS, she scored 6/14 items. Physical and neurological exam was otherwise normal.

F was started on lorazepam 1 mg orally three times a day for catatonia. Because of family preference, F went home with follow up after one day. F was seen by a general physician after two weeks, at which point she was completely asymptomatic on intermittent lorazepam dosing and had taken her examinations at school. No underlying psychiatric or medical cause for F's catatonia was specifically determined.

Case Three

D is a sixteen year-old girl with one previous episode of a "behavioral disturbance" treated briefly with haloperidol, carbamazepine and diazepam seven months prior to admission to a general hospital in rural Rwanda. D was then asymptomatic until she suddenly disappeared from her home without warning. After three months, the family located the patient in a distant district and reported that as soon as they brought D home, she laid in bed not eating or bathing and urinating on herself. The family initially sought treatment from a traditional healer but this provided no symptomatic improvement. D was brought to the local general hospital after five days without oral intake. On interview, D was laying in on a gurney with one arm posed oddly above her head, exhibited few spontaneous movements, unresponsive to most questions, made little eye contact, and spoke in neologisms and in fragmented sentences. D maintained a rigid position when attempts were made to move her arms and was resistant to attempts to sit her up on the bed. Neurological and physical exams were otherwise normal. Laboratory analysis which included a complete blood count, HIV and syphilis serologies, malaria smear, and liver function tests, was negative.

After arrival on the inpatient medical unit a few hours after admission, under the care of an inpatient general medical physician, D received diazepam 5 mg IM for restlessness, which calmed her but she remained mute, hypoactive, and refusing food, exhibiting waxy flexibility. The following day, she scored 7/14 screening items on BFCRS and was started on Diazepam 5 mg IV TID by a general physician in consultation with hospital mental health services. D was much improved clinically after one day; she was looking around, moving spontaneously, using the bathroom and answering questions appropriately. D reported the voice of the Holy Spirit telling her not to eat but she ate porridge when offered.

Diazepam 5 mg IV was continued three times daily, with complete resolution of catatonia symptoms on hospital day four. On hospital day five, haloperidol 2.5 mg PO was added for psychosis, and D was transitioned to diazepam 10 mg orally daily. On hospital day eight, D's antipsychotic was changed to flupentixol 1 mg daily for continued psychosis. D received flupentixol 1 mg PO and diazepam 10 mg PO simultaneously for seven days with no worsening of catatonia and improvement in psychotic symptoms. D was discharged on hospital day fourteen with flupentixol 1 mg PO and diazepam 10 mg PO daily. On follow up home visit, D showed no symptoms of catatonia or psychosis. She was working regularly cultivating in her family's fields and participating in community activities with peers.

Case Four

G is a twenty eight year-old woman who was transferred from a local primary care health center to a district general hospital in rural Rwanda with six days of mutism, staring, decreased blinking, immobility, stereotypic movements, and food refusal. G had been treated intermittently for several years with anti-psychotics for schizophrenia characterized by paranoia and withdrawn behavior occurring several times a year. At the time of admission she was not regularly taking any psychotropic medications and had been given chlorpromazine for presumed psychosis on presentation at the health center and also on admission to the general hospital.

On interview with general medical services, G was noted to be staring, withdrawn and mute. She made continuous repetitive movements of her hands and upper arms, especially mimicking brushing her teeth and flipping her hands back and forth. G scored 8/14 screening items on the BFCRS. At that point, in consultation with mental health services, chlorpromazine was discontinued and diazepam 10 mg IV BID started. Laboratory analysis including HIV and syphilis serologies, complete blood count and liver function tests yielded no specific abnormalities. After a total of 40 mg of intravenous diazepam G's catatonic symptoms improved and she began reporting anxiety and insomnia due to ongoing voices telling her not to sleep. On hospital day four, haloperidol 5 mg daily was started in addition to continuing diazepam. After two days, G's catatonia symptoms reappeared, with moderate motor unrest including excessive purposeless motion and increased stereotypic movements. Haloperidol was stopped at that point and diazepam increased to 10 mg IV TID. Over the subsequent seventeen days, G's catatonia symptoms slowly resolved. Three days prior to discharge, G was started on risperidone 1 mg BID for ongoing psychosis. Diazepam was changed to oral dosing, 10 mg daily, without return of catatonia symptoms. On discharge from the hospital, G denied psychotic symptoms, and catatonia symptoms had completely resolved.

Discussion

All four cases presented initially to rural general health services in low resource settings. Cases one and two were managed in a community outpatient clinic in Haiti. Cases three and four were managed in a general district hospital in rural Rwanda. In each situation, a collaborative effort between general practitioners and mental health specialists led to the

identification and treatment of catatonia with standard treatments and significant clinical improvement.

These four patients with catatonia may represent only a small subset of affected patients in rural Haiti and Rwanda, but estimates are difficult given the relative lack of prevalence data in resource-limited countries. The World Health Organization's study of the determinants of outcomes of severe mental disorders found that 10% of studied individuals exhibit symptoms of catatonia [8]. However, that study included only patients with schizophrenia. Other data from resource limited countries are derived primarily from inpatient psychiatric settings [9], [10], although one prospective study from India found catatonia in 11% of patients with psychiatric disorders admitted to a general medical hospital [11]. It is possible that the prevalence of catatonia among medical and psychiatric patients in rural resource limited settings is higher than reported in other populations, since limited access to medical and psychiatric care may cause delayed diagnosis of catatonia and contribute to underlying conditions or more severe symptomatology.

As these cases demonstrate, catatonia encompasses a range of characteristic and recognizable signs, including immobility/stupor, stereotypic movements, echophenomena, posturing, odd mannerisms, mutism, and refusal to eat or drink [12]. Usually of acute onset, catatonia typically presents as withdrawn or excited [13]. Despite these classic symptoms, catatonia is not readily recognized even by specialists worldwide. One study suggests that only 11% of patients with catatonia are correctly identified by specialists in inpatient psychiatric settings [14]. Identifying catatonia in settings with high levels of medical and psychiatric co-morbidity may be even more challenging, especially in areas where psychiatric resources are limited. The causes of catatonia are myriad and include psychiatric etiologies such as depression, mania, and psychotic disorders, as well as neurological and medical disorders including medication toxicities (for example, neuroleptics such as haloperidol and chlorpromazine), and withdrawals (for example, dopaminergic medications and GABAergic medications such as benzodiazepines) [15]. Catatonia signs and symptoms are indistinguishable whether arising from psychiatric illness or general medical conditions or toxicities [16].

The two main state variants of catatonia were readily observable in our patients. In case four, both variants were observed at different times. All of our patients initially presented to general medical services and were evaluated by non-specialist clinicians who were initially uncertain of the diagnosis. In several of our cases, the symptoms of catatonia were initially thought to represent psychosis or schizophrenia. An organized approach to evaluating catatonia, such as the Bush-Francis Catatonia Rating Scale (BFCRS) (Table 1 [17]) was useful as an aid for non-specialists to recognize specific signs of catatonia in order to screen in the syndrome. Once the catatonic syndrome was correctly identified, appropriate treatment with benzodiazepines was initiated and maintained successfully in both inpatient and outpatient general medical settings.

The ability to detect medical conditions presenting as catatonia may be challenging in some rural settings. At a minimum, a comprehensive medical history should be obtained, along with vital signs, a physical examination with particular attention to the neurological

examination, mental status examination, and available diagnostic laboratory testing. Any diagnostic approach should especially take into consideration illnesses present in the community and reversible medical etiologies of catatonia. For example, neuromedical conditions such as neurosyphilis, tuberculous meningitis, HIV encephalopathy, cerebral malaria and other central nervous systems infectious should be considered, along with epilepsy (particularly complex partial seizures, temporolimbic epilepsy and non-convulsive status epilepticus), neoplasms and nutritional deficiencies. Closed head injury should also be considered; multiple neurological insults that affect the mesencephalic frontal activating system can lead to an akinetic mutism which is phenomenologically similar to catatonic withdrawal [18]. Less frequent causes of catatonia such as inflammatory and limbic paraneoplastic processes should also be considered, although the ability to confirm these diagnoses may be limited in some areas.

The current response rate of acute catatonia to first-line treatment with an oral or parenteral benzodiazepine varies from 70% to 85% [19]. Benzodiazepines are effective for catatonia resulting from both neuropsychiatric and medical disorders, as well as drug induced catatonia, and robust responses of severe catatonic symptoms to benzodiazepines have been reported in a variety of clinical settings worldwide [20]. Patients in our series presenting with de novo catatonic symptoms for less than one week experienced complete resolution of symptoms within one week (cases two and three), whereas patients with chronic or intermittent catatonia (cases one and four, respectively) needed between two and three weeks of daily benzodiazepine administration for remittance of catatonic symptoms. Benzodiazepines are widely available even in resource limited areas, inexpensive and generally safe to use. In Haiti and Rwanda, we used lorazepam and diazepam, respectively, as first line treatment for catatonia. Although a lorazepam “challenge” test is often recommended to confirm the diagnosis of catatonia [21], an empirical therapeutic trial of a benzodiazepine can also be used in order to simplify treatment efforts. Our patients required relatively low doses of benzodiazepines for complete symptom resolution. Higher doses may be required for catatonia lysis; however, this need must be balanced with system capabilities to monitor for respiratory depression and other adverse effects, especially if managing catatonia on an outpatient basis.

In Figure 1, we summarize our initial treatment approach for catatonia, developed collaboratively with non-specialist providers in Haiti and Rwanda. There is consensus that shifting care to non-specialist providers will improve access and care for patients with neuropsychiatric disorders, especially in places where specialist resources are limited [22], [23], [24]. Familiarity with the clinical features of catatonia is essential for health professionals working in low resource settings. Delayed diagnosis or diagnostic confusion can prolong recovery and lead to serious complications such as deep venous thrombosis, pulmonary embolus, decubitus ulcers, or death [25]. In cases one and four, the psychiatric history suggested a diagnosis of a primary psychotic illness, which initially stimulated unopposed antipsychotic administration. Catatonic symptoms can be worsened by anti-psychotics and may precipitate malignant catatonia or the neuroleptic malignant syndrome [26]. Malignant catatonia, characterized by catatonic symptoms associated with fever and hyperautonomia, must be managed emergently with supportive care, antipyretic measures and antihypertensives [27]. Where available, every effort should be made to expeditiously

provide ECT, the most effective treatment for catatonia, when malignant catatonia is diagnosed since mortality is significant if the condition is allowed to persist [28] To prevent major morbidity and premature mortality, catatonia should be considered in all patients presenting with motor dysregulation and changes in mood or thinking, regardless of suspected etiology.

The catatonic syndrome should be taught as a movement and behavior syndrome in both mental health and general medical and nursing curriculums worldwide. This curriculum could take a phenomenological approach to the diagnosis of catatonia. Focusing on observable symptoms may enable health professionals with varying levels of training in neuropsychiatry to accurately identify catatonic symptoms. For example, in Rwanda and Haiti, health workers using antipsychotics have been taught to recognize the signs of neuroleptic malignant syndrome, which is a drug-induced form of malignant catatonia. A similar phenomenological approach to catatonia in general also ensures that appropriate investigations into the etiology of symptoms is completed, and clarifies appropriate treatment pathways.

Catatonia should be a subject of increased focus in the global mental health conversation, given its ease of treatment and the potential to save lives. Because non-physician health care workers deliver most mental health care in low resource settings, catatonia should also be considered for the guidelines for mental health care developed by the World Health Organization. Given its common presentation with the affective and psychotic disorders targeted by the Mental Health Global Action Program (mhGAP), catatonia should also be considered in discussions around task sharing and the creation of training materials for lay health professionals working in mental health.

Acknowledgements

The authors acknowledge the continued support of Dr. Yvonne Kayiteshonga and Dr. Tharcisse Mpunga during the writing of this paper.

Funding

None

References

1. Kahlbaum, KL. Catatonia. Levi, Y.; Pridon, T., translators. Baltimore: Johns Hopkins University Press; 1973.
2. Seethalakshmi R, Dhavale S, Suggu K, Dewan M. Catatonic Syndrome: Importance of Detection and Treatment with Lorazepam. *Annals of Clinical Psychiatry*. 2008; 20:5–8. [PubMed: 18297580]
3. World Health Organization. Geneva: World Health Organization; 2012. Global Tuberculosis Report.
4. Gelenberg A. The Catatonic Syndrome. *Lancet*. 1976; 1:1339–1341. [PubMed: 58326]
5. Fink M, Taylor AM. The catatonia syndrome: Forgotten but not gone. *Arch Gen Psychiatry*. 2009; 66:1173–1177. [PubMed: 19884605]
6. World Health Organization: World Mental Health Report. Geneva: World Health Organization; 2001. Mental Health: New Understanding, New Hope.
7. Fricchione GL, Bush G, Fozdar M, Francis A, Fink M. The recognition and treatment of the catatonic syndrome. *J Intens Care Med*. 1997; 12:135–147.

8. Jablensky A, Sartorius N, Ernberg G, et al. Schizophrenia: manifestations, incidence and course in different cultures. A World Health Organization ten-country study. *Psychol Med Monogr Suppl.* 1992; 20:1–97. [PubMed: 1565705]
9. Chalasani P, Helay D, Moriss R. Presentation and frequency of catatonia in new admissions to two acute psychiatric admission units in India and Wales. *Psychol Med.* 2005; 35:1Y9.
10. Tibrewal P, Narayanaswamy J, Zutshi A, Srinivasaraju R, Math SB. Response rate of lorazepam in catatonia: a developing country's perspective. *Prog Neuropsychopharmacol Biol Psychiatry.* 2010; 34:1520–1522. [PubMed: 20804808]
11. Seethalakshmi R, Dhavale S, Suggu K, Dewan M. Catatonic syndrome: importance of detection and treatment with lorazepam. *Ann Clin Psychiatry.* 2008; 20:5–8. [PubMed: 18297580]
12. Francis A. Catatonia: Diagnosis, Classification and Treatment. *Curr Psychiatry Rep.* 2010; 12:180–185. [PubMed: 20425278]
13. Taylor MA, Fink M. Catatonia in Psychiatric Classification: A Home of Its Own. *Am J Psychiatry.* 2003; 160:1233–1241. [PubMed: 12832234]
14. van der Heijden FM, Tuinier S, Arts NJ, Hoogendoorn ML, Kahn RS, Verhoeven WM. Catatonia: disappeared or under-diagnosed? *Psychopathology.* 2005; 38:3–8. [PubMed: 15714008]
15. Fink M, Taylor AM. The catatonia syndrome: Forgotten but not gone. *Arch Gen Psychiatry.* 2009; 66:1173–1177. [PubMed: 19884605]
16. Carroll BT, Kennedy JC, Goforth HW. Catatonic signs in medical and psychiatric catatonias. *CNS Spectr.* 2000; 5:66–69. [PubMed: 18197158]
17. Bush G, Fink M, Petrides G, Dowling F, Francis A. Catatonia. I. Rating scale and standardized examination. *Acta Psychiatrica Scandinavica.* 1996; 93:129–136. [PubMed: 8686483]
18. Fisher CM. Honored guest presentation: abulia minor vs. agitated behavior. *Clin Neurosurg.* 1983; 31:9–31. [PubMed: 6149834]
19. Rosebush P.; Mazurek M. Pharmacotherapy. In: Caroff, SN.; Mann, SC.; Francis, A.; Fricchione, GL., editors. *Catatonia: From Psychopathology to Neurobiology.* Washington, DC: American Psychiatric Press; 2004. p. 141-150.
20. Rosebush PI, Mazurek MF. Catatonia and its treatment. *Schizophr Bull.* 2010; 36:239–242. [PubMed: 19969591]
21. Fricchione GL, Bush G, Fozdar M, Francis A, Fink M. The recognition and treatment of the catatonic syndrome. *J Intens Care Med.* 1997; 12:135–147.
22. Patel V, Araya R, Chatterjee S, Chisholm D, Cohen A, et al. Treatment and prevention of mental disorders in low-income and middle-income countries. *Lancet.* 2007; 370:991–1005. [PubMed: 17804058]
23. Raviola G, Becker AE, Farmer P. A global scope for global health—including mental health. *Lancet.* 2011; 378:1613–1615. [PubMed: 22008421]
24. Collins PY, Patel V, Joestl SS, March D, Insel TR, et al. Grand challenges in global mental health. *Nature.* 2011; 475:27–30. [PubMed: 21734685]
25. Larsen HH, Ritchie JC, McNutt MD, Musselman DL. Pulmonary Embolism in a Patient with Catatonia: An Old Disease, Changing Times. *Psychosomatics.* 2011; 52:4387–4391.
26. White DA, Robins AH. Catatonia: harbinger of the neuroleptic malignant syndrome. *Br J Psychiatry.* 1991; 158:419–421. [PubMed: 1674666]
27. Philbrick KL, Rummans TA. Malignant catatonia. *J Neuropsychiatry Clin Neurosci.* 1994; 6:1–13. [PubMed: 7908547]
28. Philbrick KL, Rummans TA. Malignant catatonia. *J Neuropsychiatry Clin Neurosci.* 1994; 6:1–13. [PubMed: 7908547]

Figure 1.
Identification and Management of Catatonia

Table 1

14-item Bush-Francis catatonia screening scale [17].

Excitement	Stereotypy
Immobility/stupor	Mannerisms
Mutism	Verbigeneration
Staring	Rigidity
Posturing/catalepsy	Negativism
Grimacing	Waxy flexibility
Echopraxia/echolalia	Withdrawal