

Catatonias:From Diagnosis to Management

ABSTRACT :

Catatonias is a complex psychomotor syndrome characterized by a range of motor, behavioral, and speech abnormalities. It can occur in the context of psychiatric disorders such as mood disorders, schizophrenia, autism spectrum disorders, and intellectual disabilities, as well as in various medical and neurological conditions including encephalitis, encephalopathies, metabolic disorders, and drug withdrawal. Early recognition and management are crucial to prevent serious psychiatric, organic, and vital complications.

Diagnosis is primarily clinical, based on DSM-5-TR criteria, requiring at least three key features such as stupor, catalepsy, mutism, negativism, posturing, stereotypies, or echolalia/echopraxia. Differential diagnoses include obsessive-compulsive disorder, autism spectrum disorder, selective mutism, conversion disorder, tics, and drug-induced movement disorders.

Management involves a comprehensive evaluation of psychiatric and non-psychiatric etiologies, severity assessment using tools like the Bush-Francis Catatonia Rating Scale, and appropriate investigations to rule out organic causes. First-line treatment is benzodiazepines, particularly lorazepam, with rapid and effective response in many cases. For resistant or malignant forms, electroconvulsive therapy (ECT) is highly effective, and in some cases, non-invasive neurostimulation techniques such as repetitive transcranial magnetic stimulation (rTMS) may serve as alternatives.

Concomitant management of complications related to immobility, malnutrition, and autonomic dysfunction is essential. Early diagnosis, availability of skilled personnel, and access to appropriate therapeutic modalities are critical factors determining prognosis. Advances in neurostimulation have significantly improved outcomes in catatonic patients.

I. Introduction

Psychiatric emergencies in the Moroccan context represent a challenge for the patient, their relatives, as well as for healthcare providers and psychiatric and general hospital structures. Examples include agitation, suicidal crises, panic attacks, catatonia, and many other emergencies, for which early and adequate management determines not only the psychiatric prognosis but also the organic and vital outcomes.

Among the emergencies most feared by psychiatrists and emergency physicians is catatonia, whose early and appropriate diagnosis and management improve both the psychiatric and vital prognosis of the patient (1,2).

This work aims to provide a detailed overview of catatonia management, taking into account the pharmacological agents available in Morocco.

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41 **II. Definition**

42 Catatonia is a psychomotor syndrome encompassing abnormalities of motor behavior that may
43 last minutes, hours, often days, and sometimes years (3–5).

44 The clinical picture of catatonia is remarkably rich. Observed features may include mannerisms,
45 grimacing, posturing, agitation, stereotypies, fixed gaze, and ambivalence (opposing or
46 contradictory attitudes) (1,2).

47 During patient interviews, mutism, echolalia, echopraxia, and verbalization can be observed.
48 Withdrawal and negativism are also commonly seen (3,4).

49 Physical examination may reveal wax flexibility, rigidity, catalepsy, or grasp reflex,
50 along with oppositional behavior (2).

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52 **III. Positive Diagnosis: DSM-5-TR Criteria**

53 To diagnose catatonia, **at least three** of the following criteria must be present:

- 54 1. **Catalepsy:** maintenance against gravity of postures imposed by the examiner
- 55 2. **Waxy flexibility:** slight and constant resistance to passive movement applied by the
56 examiner
- 57 3. **Stupor:** absence of psychomotor activity, with no interaction with the environment
- 58 4. **Agitation:** not influenced by external stimuli
- 59 5. **Mutism:** absence or near-absence of verbal response (exclude if secondary to a
60 known aphasia)
- 61 6. **Negativism:** opposition to or lack of response to instructions or external stimuli
- 62 7. **Posturing:** active maintenance, against gravity, of a spontaneously assumed posture
- 63 8. **Mannerisms:** caricatures of ordinary actions marked by bizarreness or solemnity
- 64 9. **Stereotypies:** purposeless, repetitive, and abnormally frequent movements
- 65 10. **Grimacing facial expressions**
- 66 11. **Echolalia:** repetition of the examiner's words
- 67 12. **Echopraxia:** imitation of the examiner's movements (1,2)

68 In parallel with psychiatric assessment, it is important to search for autonomic signs of possible
69 **malignant catatonia:**

- 70 • Tachycardia or bradycardia
- 71 • Hypo- or hypertension
- 72 • Rapid or slow breathing
- 73 • Sweating
- 74 • Hyperthermia > 38.5 °C or hypothermia < 35 °C (1)

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76 IV. Differential Diagnosis

- 77 1. **Obsessive–compulsive disorders (OCD):** compulsive
78 rituals may involve repeated behaviors and slowness due to indecision (6)
- 79 2. **Autism spectrum disorders (ASD):** stereotyped behaviors involve compulsive and
80 ritualized actions, as well as mannerisms (5,7)
- 81 3. **Intellectual disability:** stereotyped behaviors occurring in environmental stress contexts
82 (5)
- 83 4. **Selective mutism:** occurring in the context of an anxiety disorder without
84 communication deficits (6)
- 85 5. **Acute stress disorder:** occurring in a traumatic context (6)
- 86 6. **Conversion disorder:** fluctuating presentation, suggestibility,
87 physiologically inconsistent (6)
- 88 7. **Chronic tics:** multiple motor and vocal tics (6)
- 89 8. **Gilles de la Tourette syndrome:** complex motor and vocal tics, including echolalia,
90 echopraxia, and grimacing (6)
- 91 9. **Developmental coordination disorder:** bizarre, complex motor sequences (6)
- 92 10. **Stereotypic movement disorder** (6)
- 93 11. **Adverse effects of antipsychotics:** akathisia, parkinsonism, dyskinesia, acute dystonia
94 (1)
- 95 12. **Serotonin syndrome** (1)

96

97 V. Etiologies

98 1. Psychiatric Etiologies

99 Psychiatric causes are the most frequent (75–80%) (1,2):

- 100 • **Mood disorders:** bipolar disorder, unipolar disorder
- 101 • **Schizoaffective disorder**
- 102 • **Schizophrenia**
- 103 • **Autism spectrum disorder** (5,9)
- 104 • **Intellectual developmental disorder**
- 105 • **Acute stress disorder**

106 In children and adolescents, catatonia is particularly associated with autism spectrum disorders
107 and early-onset psychotic disorders (2,8–10).

108 2. Non-Psychiatric Etiologies (20–25%)

109 a. Encephalitides

- 110 • **Infectious:** HSV1, Syphilis, HIV, HBV, HCV
- 111 • **Inflammatory:** multiple sclerosis flare, neurolupus
- 112 • **Spongiform:** Creutzfeldt–Jakob disease, fatal familial insomnia
- 113 • **Neoplastic**

- 114 • **Autoimmune:** Hashimoto's encephalitis, limbic encephalitis with anti-NMDAR
115 antibodies (1)

116 b. Encephalopathies

- 117 • **Toxic:** alcohol, cocaine, ecstasy, hyponatremia, drug-induced
118 • **Metabolic:** ketoacidosis, severe hyponatremia
119 • **Deficiency-related:** vitamin B12, folate
120 • **Neurometabolic disorders:** Niemann–Pick disease type C, Wilson disease,
121 hyperhomocysteinemia, porphyria, xanthomatosis, urea cycle disorder (1,2)

122 c. Other Causes

- 123 • **Epilepsies**
124 • **Braintumors**
125 • **Dementias**
126 • **Traumatic brain injury**
127 • **Cerebral infarctions:** parietal, temporal, or thalamic lobes
128 • **Withdrawal states:** benzodiazepines, clozapine, anticholinergics, alcohol (1)
129 • **Genetic syndromes:** DiGeorge syndrome (1)

130

131 VI. Management

132 Catatonia is not a disease itself but a syndrome requiring thorough investigation of its causes.
133 Associated symptoms may vary, justifying close clinical observation and
134 regular physical examinations (1,2).

135 1. Evaluation

136 A standardized assessment should include:

- 137 **a. Clinical Examination:** detailed history and neurological exam (1)
138 **b. Priority Investigations:** exclude abrupt withdrawal (benzodiazepines, alcohol,
139 anticholinergics) and identify organic causes (degenerative, metabolic, immunologic) (1,2)
140 **c. Symptom kinetics:** evaluate onset and prior neuropsychiatric disorders, especially in
141 older adults (cognitive decline, loss of autonomy) (1,2)
142 **d. Targeted neurological exam:** identify vigilance disorders, neuropathies,
143 abnormal movements, cerebellar or pyramidal involvement, or oculomotor paralysis (1)
144 **e. General physical exam:** cardiac and pulmonary auscultation, skin inspection, abdominal
145 palpation (hepatosplenomegaly), lymph nodes, thyroid (1)

146 2. Paraclinical Assessment

147 a. Etiological Workup

- 148 • **Laboratory:** TSH, fasting glucose, calcium/phosphate/vitamin D/PTH, vitamins B1,
149 B6, B9, B12, ammonia, homocysteine, cholesterol profile, cortisol, CRP,

- 150 serumproteinelectrophoresis, serologies (HIV, HBV, HCV, syphilis), anti-DNA, anti-
151 streptococcalantibodies (1)
- 152 • **Imaging:** MRI (preferred) to detect focal lesions,
153 degenerative/metabolic/inflammatory signs, intracranial hypertension; CT scan if MRI
154 unavailable (1)
 - 155 • **EEG:** in altered consciousness, fluctuating symptoms, or suspected epilepsy (1)
 - 156 • **Lumbarpuncture:** in suspected encephalitis, subacute neurological changes, seizures,
157 systemic inflammation, or unexplained infectious syndrome (1)

158 **b. Pre-therapeutic Assessment:** complete blood count, coagulation, electrolytes, renal and
159 liver function, CPK, drug levels, urine toxicology (1)

160 **3. Severity Assessment**

161 **Bush–Francis Catatonia Rating Scale (BFCRS):** 23 items scored 0–3; total 69 (1)

- 162 • **Malignant catatonia:** febrile catatonia with autonomic instability (tachycardia,
163 tachypnea, sweating); mortality 10–20% (1)
- 164 • **Chronic catatonia:** episodes lasting months or years (2,10)

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166 **VII. Treatment**

167 **1. Benzodiazepines**

168 First-line treatment, especially **lorazepam**, with response in hours to days, efficacy up to 80%
169 (1,2). Start with lorazepam 1–2.5 mg IV (or orally), evaluate after 30 min. Increase dose every
170 4–12 h until symptoms resolve; daily doses may reach 16–20 mg. Zolpidem may also be used for
171 diagnostic test (1).

172 **2. Electroconvulsive Therapy (ECT) and other neurostimulation methods :**

173 ECT is indicated for resistant or malignant catatonia. Pre-anesthesia evaluation and MRI to rule
174 out intracranial hypertension (9).

- 175 • **Absolute contraindication:** intracranial hypertension
- 176 • **Relative contraindications:** recent intracerebral hemorrhage, head trauma,
177 untreated retinal detachment, severe glaucoma, recent myocardial infarction,
178 unstable angina, unstable fractures

179 ECT initiation: titrate electrical current to seizure threshold or use “age-dose” method.
180 Bitemporal electrode placement recommended; lorazepam may be paused 8–12 h before session.
181 Post-ECT effects: headache, amnesia, nausea, vomiting, confusion, anesthesia-related effects
182 (9,15).

183 FDA recommendation: ECT in patients >13 years with refractory or severe catatonia,
184 including malignant forms. Pediatric efficacy: 76–80% (9,10,12).

- 185 • **rTMS** treatment could, in some cases, serve as a therapeutic alternative to avoid long-term
186 ECT.
187 • **tDCS**: noninvasive cortical stimulation using low-intensity direct current via scalp
188 electrodes (15).

189 **3. Management of Complications**

190 Prevent and treat malnutrition, dehydration, and complications of immobility
191 (thromboembolic, infections, joint issues) (1).

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193 **VIII. Conclusion**

194 Management of catatonia has improved significantly, especially with brain stimulation
195 techniques. Availability of suitable equipment, qualified personnel, and early intervention
196 remain key factors determining prognosis (1,15).

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