The Spectrum of Involuntary Vocalizations in Humans: A Video Atlas

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ABSTRACT: In clinical practice, involuntary vocalizing behaviors are typically associated with Tourette syndrome and other tic disorders. However, they may also be encountered throughout the entire tenor of neuropsychiatry, movement disorders, and neurodevelopmental syndromes. Importantly, involuntary vocalizing behaviors may often constitute a predominant clinical sign, and, therefore, their early recognition and appropriate classification are necessary to guide diagnosis and treatment. Clinical literature and video-documented cases on the topic are surprisingly scarce. Here, we pooled data from 5 expert centers of movement disorders, with instructive video material to cover the entire range of involuntary vocalizations in humans. Medical literature was also reviewed to document the range of possible etiologies associated with the different types of vocalizing behaviors and to explore treatment options. We propose a phenomenological classification of involuntary vocalizations within different categorical domains, including (1) tics and tic-like vocalizations, (2) vocalizations as part of stereotypies, (3) vocalizations as part of dystonia or chorea,

(4) continuous vocalizing behaviors such as groaning or grunting, (5) pathological laughter and crying, (6) vocalizations resembling physiological reflexes, and (7) other vocalizations, for example, those associated with exaggerated startle responses, as part of epilepsy and sleep-related phenomena. We provide comprehensive lists of their associated etiologies, including neurodevelopmental, neurodegenerative, neuroimmunological, and structural causes and clinical clues. We then expand on the pathophysiology of the different vocalizing behaviors and comment on available treatment options. Finally, we present an algorithmic approach that covers the wide range of involuntary vocalizations in humans, with the ultimate goal of improving diagnostic accuracy and guiding appropriate treatment. © 2019 The Authors. *Movement Disorders* published by Wiley Periodicals, Inc. on behalf of International Parkinson and Movement Disorder Society.

Key Words: involuntary vocalizations; movement disorders; vocalizing behavior

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The ability to vocalize has only been a fairly recent evolutionary acquisition and was a prerequisite for the development of verbal communication in our species.[1] Our acquired repertoire of vocalizations ranges from simple sounds related to physiological reflexes (eg, sneezing) and emotional responses (eg, crying, laughing) to the intended articulation of words that are meant to express specific communicative content.[2] In all these instances, vocalizations are typically context specific and adaptive to environmental stimuli. However, the occurrence of vocalizing behaviors in the absence of these qualities typically signifies pathology and most often constitutes a major cause of distress.

Medical literature and clinical practice have historically associated abnormal vocalizing behaviors with tic disorders, as for example, Tourette syndrome (TS), of which they are also an essential part of the diagnostic criteria.[3] However, involuntary vocalizations may also be encountered throughout the entire tenor of neuropsychiatric disorders, to include movement disorders, neurodegenerative and neurodevelopmental syndromes, and functional neurological disorders. Ictal phenomena in epileptic disorders may also present with vocalizing behaviors. Although in many of these disorders, abnormal vocalizations will often be only one feature of a range of abnormal motor behaviors and clinical signs, in some cases, they may constitute the sole clinical finding. Here, their early recognition and appropriate classification are paramount for guiding diagnostic reasoning and informing therapeutic decisions. However, beyond tic disorders and TS, the clinical literature on the topic remains sparse,[2] and video-documented cases are particularly rare.

Over a period of several years, we came across a number of patients in whom abnormal vocalizations were the predominant reason for clinical presentation. Given the difficulties in the phenomenological classification of vocalizing behaviors, we here provide a clinical overview of the range of involuntary vocalizations in humans, together with 29 informative video-documented cases, to illustrate both typical and more unusual clinical examples. Our goal is to inform our colleagues from the neighboring fields of neurology, neuropsychiatry, and psychiatry on the phenomenological spectrum and diagnostic conditions associated with involuntary vocalizations, discuss their pathophysiology, and provide treatment recommendations where possible.

Methods

Data from 5 expert centers of movement disorders across Europe (Department of Neurology, Charité University Medicine Berlin, Berlin, Germany; Department of Clinical and Movement Neurosciences, Queen Square Institute of Neurology, University College London, London, UK; Department of Neurology, University Hospital Schleswig-Holstein, Christian-Albrechts-University, Kiel, Germany; Department of Neurology, University Medical Centre Groningen, University of Groningen, Groningen, The Netherlands; Clinic of Psychiatry, Socialpsychiatry and Psychotherapy, Hannover Medical School, Hannover, Germany) were pooled for this study. Cases of patients in whom involuntary vocalizations predominated in clinical presentation and for whom video material was available were first collected and reviewed. We selected the cases that exemplified distinct phenomenological characteristics of different vocalizing behaviors. We also reviewed the literature to identify the range of possible etiologies associated with the different types of

vocalizing behaviors that we included and to explore treatment options. Based on our clinical experience and the available material we gathered, we also provide practical treatment recommendations where possible. Signed patient consent was obtained for videos of all patients that we present here.

Tics and Tic-Like Vocalizations

Tics are defined as movements or sounds that resemble physiological motor behaviors, but are typically inopportune to social context and appear sudden, repetitive, and often exaggerated.[4] Tic vocalizations — commonly termed vocal or phonic tics — may include any possible sound (eg, sniffing, coughing, throat clearing, whistling, or grunting), word, or sentence and are most commonly encountered within the spectrum of primary tic disorders, as TS (Video 1A–C). In these patients, tics, including phonic and vocal behaviors, are typically preceded by premonitory urges and can be suppressed voluntarily.[4-7] Individuals with autism spectrum disorders (ASD) may also present with vocal tics, and indeed an overlap between primary tic disorders and autistic features has been reported in the medical literature.[8,9] However, in ASD premonitory urges and overall vocal tic awareness may be reduced compared to people with primary tic disorders and TS.[10] Klinefelter,[11] fragile X,[12] and Adams-Oliver syndrome,[13] as well as monosomy 9p[14] and trisomy 16p[15] are documented genetic causes of other neurodevelopmental disorders that may manifest phonic/vocal tics. Neurodegenerative syndromes may also present with phonic or vocal tics, for example, in Huntington's disease (Video 1D). Here, vocalizing behaviors such as grunting tics are often characteristic (Video 1E,F),[16-18] and although the distinction of tics from choreic sounds (also see the section on Vocalizations as Part of Dystonia, Chorea, and Other Dyskinesias) may often be difficult, some patients describe the presence of premonitory urges preceding vocal tics (case example of video 1E). Furthermore, vocal tics have been documented in patients with chorea-acanthocytosis because of VPS13A mutations[19-21] (Video 1G), Amyotrophic lateral sclerosis (ALS) frontotemporal dementia (FTD) overlap syndromes,[22] progressive supranuclear palsy (PSP),[23] and pantothenate kinase-associated neurodegeneration (PKAN).[24] Neurometabolic disorders such as Wilson's disease or phenylketonuria,[25,26] focal brain lesions,[27-34] infectious,[35-37] and other autoimmune diseases[38-41] are additional causes of vocal tics (see Table 1). Finally, phonic or vocal tics may also be drug-induced, either directly related to the acute effects of drugs[42-46] (eg, cocaine[46]) or as a long-term consequence, such as in tardive tic disorders[47-49] (Video 1H).

A final etiological category includes functional neurological disorders. Previous literature on such cases refers to repetitive sounds resembling vocal tics as tic-like vocalizations and offers clinical clues to distinguish the 2 types of behaviors.[50-53] Abrupt symptom onset, typically in

TABLE 1. Spectrum of involuntary vocalizations in humans, their descriptions, and etiologies

Vocalization	Description	Possible etiology
Tics and tic-like vocalizations	Sudden, exaggerated, repetitive, and inopportune to social context sounds (eg, sniffing, coughing, throat clearing, whistling, grunting) or words[b]	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, ASD, Klinefelter, fragile X, Adams-Oliver syndrome, monosomy 9p, trisomy 16p) — Neurodegenerative disorders (eg, HD, chorea-acanthocytosis, ALS-FTD overlap syndromes, PSP, PKAN) — Neurometabolic disorders (eg, Wilson's disease, PKU) — Focal brain lesions (eg, after head trauma, arteriovenous hemorrhage, following cardiac surgery, after temporal lobectomy, in osmotic demyelination syndrome, after carbon monoxide poisoning, postinfectious (VZV encephalitis)) — Infectious (eg, HIV, HSV, rubella virus) — Autoimmune (eg, postinfectious, MS, SLE, Behcet's disease, antiphospholipid syndrome) — Drug-induced (eg, carbamazepine, lamotrigine, bupropion, cocaine) — Tardive (eg, antipsychotics) — Functional neurological disorders
• Klazomania	Compulsive shouting episodes	— Primary tic disorders (eg, TS) — Focal brain lesions (eg, carbon monoxide poisoning) — Others (eg, depressive disorders, postencephalitic parkinsonism) — Functional neurological disorders
• Palilalia	Repetition of one's own syllables, words, or phrases 2 or more times in a row	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, ASD, trisomy 16p) — Neurodegenerative disorders (eg, Alzheimer's disease, PSP, chorea-acanthocytosis, VCP proteinopathy, PD[a]) — Focal brain lesions (eg, ischemic, hemorrhagic, after stereotaxic thalamotomy, after severe head trauma, after carbon monoxide poisoning, after respiratory acidosis, associated with extensive intracerebral calcifications, postinfectious [VZV encephalitis]) — Ictal — Autoimmune (eg, steroid-responsive encephalopathy) — Drug-induced (eg, clozapine, cefepime) — Others (eg, early-onset schizophrenia, membranous lipodystrophy, postencephalitic parkinsonism) — Functional neurological disorders (including startle syndromes, eg, Latah)
• Echolalia	Imitative repetition of sounds, words, or phrases in the absence of explicit awareness	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, ASD, Rubinstein-Taybi, fragile X, Williams syndrome, trisomy 16p) — Neurodegenerative disorders (eg, DLB, FTD, Alzheimer's disease, HD, CJD, PSP-CBS, CBD, familial progressive subcortical gliosis, chorea-acanthocytosis) — Neurometabolic disorders (eg, Wilson's disease, NPC, encephalopathy in D-lactic acidosis, after liver transplantation) — Focal brain lesions (eg, ischemic, after severe head trauma, after carbon monoxide poisoning) — Infectious (eg, cerebral malaria) — Autoimmune (eg, Hashimoto's encephalopathy, MS, NMDA-receptor encephalitis, SLE) — Drug-induced (eg, isoniazid, topiramate, ofloxacin, methoxphenidine, cocaine, designer tryptamine, phencyclidine) — Functional neurological disorders (including startle syndromes, eg, Jumping Frenchmen of Maine, Latah, Ragin' Cajuns of Louisiana) — Others (eg, encephalitis lethargica, catatonia)
• Coprolalia	Unintended utterance of obscenities and socially inappropriate and derogatory remarks	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, Kleine-Levin syndrome, fragile X syndrome) — Neurodegenerative disorders (eg, FTD, Alzheimer's disease, choreo-acanthocytosis) — Focal brain lesions (eg, after carbon monoxide poisoning) — Ictal — Functional neurological disorders — Others (eg, encephalitis lethargica)
Vocalizations as part of stereotypies	Vocalizations associated with repetitive, non-goal-directed, and distractible movement patterns	— Physiological, normal development — Neurodevelopmental disorders (eg, ASD, 15q13.3 microdeletion, Rett syndrome) — Others (eg, schizophrenia)

(Continues)

TABLE 1. Continued

Vocalization	Description	Possible etiology
Vocalizations as part of dystonia, chorea, and other dyskinesias	Phonic or vocal phenomena due to hyperkinetic movements, as chorea, dystonia, and other dyskinesias[b]	— Neurodegenerative disorders (eg, HD, chorea-acanthocytosis) — Autoimmune (eg, postinfectious) — Drug-induced (eg, antipsychotics, metoclopramide, lenalidomide)
Continuous vocalizations such as groaning, moaning, grunting, and shrieking	Continuous or repetitive groaning, moaning, grunting, and shrieking in the absence of appropriate context	— Neurodegenerative disorders (eg, Alzheimer's disease, vascular dementia, HD, PD, PSP) — Neurometabolic disorders (eg, acquired hepatocerebral degeneration) — Functional neurological disorders
Pathological laughter and crying	Laughter and crying occurring detached from emotional content	— Primary tic disorders (eg, TS) — Other neurodevelopmental disorders (eg, Angelman syndrome, partial trisomy 16p, Rett-like syndromes) — Neurodegenerative disorders (eg, ALS, FTD, Alzheimer's disease, primary progressive aphasia, MSA-C, CJD, SCA17, HD) — Focal brain lesions (eg, cerebrovascular disease, traumatic brain lesions) — Ictal (eg, gelastic seizures) — Autoimmune (eg, acute disseminated encephalomyelitis, MS) — Drug induced (eg, intravenous sodium valproate)
Vocalizations resembling physiological reflexes	Repetitive sounds such as belching, sniffing, coughing, wheezing[b]	— Physiological (eg, contagious yawning, groaning during sexual intercourse) — Primary tic disorders (eg, TS) — Neurodegenerative disorders (eg, as OFF symptom in PD) — Focal brain lesions (eg, ischemic) — Ictal (eg, seizure-ending signs, temporal lobe seizures) — Infectious (eg, herpes simplex encephalitis) — Functional neurological disorders

Others	Broad range of involuntary vocalizations not clearly belonging in any of the previous categories[b]	— Culture-bound startle syndromes
		— Functional neurological disorders (eg, exaggerated stimulus-triggered responses)
		— Ictal (eg, ictal cry, animal noises, singing, and humming)
		— Sleep related (eg, snoring, catathrenia, stridor [eg, in MSA, anti-IgLON5 disease, SCA17]),
		  — Night terrors
		  — Sleep-related hypermotor seizures
		  — REM sleep disorder in primary tic disorders (eg, TS), neurodevelopmental disorders (eg, ASD), neurodegenerative disorders (eg, PD, MSA, DLB, FTD, ALS, SCA3, xeroderma pigmentosum, HD), focal brain lesions (eg, brain stem ischemia, tumors) autoimmune disorders (eg, MS, Guillain-Barré syndrome, paraneoplastic), and others (eg, narcolepsy, epilepsy, posttraumatic stress disorder)

ALS, amyotrophic lateral sclerosis; ASD, autism spectrum disorder; CBD, corticobasal degeneration; CBS, corticobasal syndrome; CJD, Creutzfeld-Jacob disease; DLB, dementia with Lewy bodies; FTD, frontotemporal dementia; HD, Huntington's disease; HIV, human immunodeficiency virus; HSV, herpes simplex virus; MS, multiple sclerosis; MSA-C, multiple system atrophy–cerebellar type; NPC, Niemann Pick type C; PD, Parkinson's disease; PKAN, pantothenate kinase-associated neurodegeneration; PKU, phenylketonuria; PSP, progressive supranuclear palsy; REM, rapid eye movement; SCA, spinocerebellar ataxia; SLE, systemic lupus erythematosus; TS, Tourette syndrome; VCP, valosin-containing-protein; VZV, varicella zoster virus.
[a]May occur in association with peak-dose levodopa or as side effect of deep brain stimulation.
[b]Some sounds are mediated by supraglottic structures without involvement of the larynx.

adulthood, absence of premonitory urges, lack of suppressibility, and atypical response to anti-tic medication, alongside the presence of further functional movement disorders and medically unexplained symptoms, are indeed characteristic red flags that should prompt the consideration of a functional etiology. However, even with these helpful aids, correct diagnostic labeling and etiological distinction may often be challenging, particularly in cases in which both tics and tic-like movements or sounds may co-occur.[4,54]

Klazomania

The term *klazomania* (after the Greek word for "crying") was first coined in 1925 by Benedek, a German psychiatrist, who described a patient with postencephalitic parkinsonism and involuntary attacks of compulsive paroxysmal shouting.[55] The shouting behaviors were described as extremely loud and not related to the ongoing mental state of the patient. They occurred in bouts and could last for several hours. Syllables, vowels, single words, and sometimes noises, described in the original report as "carnivorous" animal sounds, were noted.[55] Palilalic behaviors (see below) were also described in this patient, who was able to briefly suppress the involuntary vocalizations with forceful breathing. Inappropriate shouting is also a well-documented feature in TS[56] (Video 1I) and functional neurological disorders (Video 1J). Other associations of klazomania include depression[57-59] and carbon

monoxide poisoning.[60,61] Clearly, as in the distinction of tics from tic-like vocalizations, beyond phenomenological observation of vocalizing behaviors, historical information and the presence of additional clinical features are crucial to distinguish between the different etiological categories.

Palilalia

Palilalia is the involuntary repetition of one's own phrases, words, or syllables 2 or more times in a row.[62] Typically, palilalic utterances decrease in volume with the increasing number of repetitions.[63] Sometimes, the repetitions are also uttered with an accelerating speed.[62,64]

In 1908, Souques first described palilalia in a patient with an ischemic stroke of the right hemisphere.[62] Since then, palilalia, which is typically documented in up to a third of patients with TS[56,65-67] (Video 1A), was reported in patients with other neurodevelopmental disorders such as ASD[68] or trisomy 16p[15] and neurodegenerative disorders, such as PSP,[69,70] dementia of the Alzheimer's type,[71] valosin-containing-protein proteinopathy,[72] or chorea-acanthocytosis[73] (Video 1G). In patients with typically advanced parkinsonism, palilalia may also be observed either irrespective of their medication status[74] or in association with peak doses of levodopa[75] and as a side effect of bilateral stereotaxic thalamotomy, most likely as the effect of lesions.[76] Focal brain lesions, typically affecting thalamic and/or midbrain structures, may also lead to the expression of palilalic behaviors.[34,60,62,76-85] A family with extensive intracerebral calcification was reported to present palilalia,[86] and indeed patients with Fahr syndrome, an etiologically heterogeneous disorder,[87] will often present this clinical sign. Further, palilalia was reported as ictal,[88] autoimmune,[89] and drug-induced phenomenon (eg, with clozapine[90] or cefepime[91]).

Like klazomania, palilalia was also observed in patients with encephalitis lethargica and postencephalitic parkinsonism.[62,64,92-94] Other reported etiologies are early-onset schizophrenia[95] and membranous lipodystrophy.[96] Finally, palilalic behaviors may also be encountered in functional neurological disorders[52] and in culture-bound startle syndromes (eg, Indonesian Latah; also see below).[97] Of note, palilalia should be distinguished from stuttering, a disorder with dysfluency of speech and repetition of sounds, syllables, or words (eg, Video 1A, palilalia vs Video 1K, stuttering).[98,99]

Echolalia

Echolalia is the automatic imitative repetition of sounds, words, or phrases in the absence of explicit awareness.[100] Although echolalia constitutes a physiological neurodevelopmental phenomenon, its unremitting persistence or reemergence may point to pathology.[100]

As with the majority of involuntary vocalizing behaviors, the prevalence and exact characteristics of echolalia in different disorders remain understudied.[101] However, it is most commonly reported in TS[66,102] and ASD.[103-106] Patients with other neurodevelopmental disorders,[15,107-110] including fragile X[108,109] and Williams syndrome,[110] and neurodegenerative syndromes (eg, dementia with Lewy bodies,[111] various tauopathies,[112-117] HD,[118] Creutzfeldt-Jakob disease (CJD),[119] and chorea-acanthocytosis[73]) may also present with echolalia. Neurometabolic disorders, such as Niemann-Pick type C (Video 1L) and Wilson's disease[120] or encephalopathic syndromes,[121-123] as well as brain lesions due to focal or diffuse cerebrovascular damage,[114,124] carbon monoxide poisoning,[60] and severe head trauma[85] were also associated with echolalic behaviors. Infections (eg, cerebral malaria[125]) and autoimmune disorders such as N-methyl-D-aspartate (NMDA)–receptor encephalitis,[126] systemic lupus erythematosus,[127] and others[128,129] may also present with echolalia. Drug-induced echolalia was noted with isoniazid,[130] topiramate,[131] ofloxacin,[132] the NMDA-receptor antagonist methoxphenidine,[133] cocaine,[134] designer tryptamine,[135] and phencyclidine ("angel dust," "crystal").[136] Other underlying causes of echolalia are encephalitis lethargica,[94] catatonia,[137] functional neurological disorders (Video 1M[101]), and endemic startle syndromes such as the Jumping Frenchmen of Maine,[138] Latah,[97] and the Ragin' Cajuns of Louisiana.[139] Indeed, in this latter group of etiologies, echolalic behaviors are characteristic.

Coprolalia

The exact definition of coprolalia in the medical context has been tortuous. Essentially, coprolalia denotes the involuntary utterance of obscenities.[52] Intent is an important classifier in coprolalic behaviors, and unfortunately it remains unclear how to objectively distinguish coprolalia from common swearing. In TS, the unintended expression of coprolalic behaviors is encountered in about one-fifth of patients.[140] Typical coprolalic behaviors in TS are characterized by the utterance of single short—in the English language, 4-letter—words with a different pitch or tone from ongoing speech.

There have been only a few reports of patients exhibiting coprolalia in other neurological conditions, such as neurodevelopmental disorders (eg, Kleine-Levin[141] and fragile X syndrome[12]), neurodegenerative syndromes (eg, FTD,[142] Alzheimer's disease,[143] and chorea-acanthocytosis[144]), after focal brain lesions[60], in encephalitis lethargica[94], or as ictal phenomenon.[145] A final category includes functional neurological disorders, and often these patients may be misdiagnosed with TS, although their clinical characteristics may largely differ.[52] Indeed, different from coprolalia in TS, functional coprolalic behaviors often comprise short sentences with obscene content. Most importantly, many of these behaviors are also context dependent (see Video 1N). A previous history of medically unexplained

symptoms and further documented functional neurological signs are typically present.[52]

Vocalizations as Part of Stereotypies

The precise definition of stereotypies and their exact phenomenological distinction from other repetitive motor behaviors, for example, tics, is difficult.[146] The term denotes a repetitive, often continuous, non-goal-directed movement pattern that is typically distractible.[146] As with echolalic behaviors, stereotypies are also part of physiological development that often abate within the first years of life.[146] Although the persistence of stereotypic vocalizations may still be part of normal development,[147] in many cases it signifies pathology, and indeed stereotypic utterances are part of the diagnostic criteria of ASD (Video 2A).[3] One large case series of 83 patients with Rett syndrome described phonic stereotypies with repetitive sounds, words, or phrases in only 6% of patients.[148] We recently observed loud stereotypic vocalizations in a patient with 15q13.3 microdeletion syndrome (Video 2B) and late-treated cases with phenylketonuria. Further, stereotypic vocalizations have been documented in patients with schizophrenia.[149]

Vocalizations as Part of Dystonia, Chorea, and Other Dyskinesias

Involuntary sounds may also be part of dystonic and choreic disorders. For example, lip-smacking sounds (Video 3A) and panting and gasping (Video 3B) are characteristic presentations of drug-related, usually tardive syndromes. Most recently, we documented a case with generalized dyskinetic movements and loud utterances following treatment with lenalidomide (Video 3C). A similar case, albeit without video documentation, was also recently reported.[150] In chorea-acanthocytosis, beyond the presence of tic vocalizations, sounds such as belching, spitting, clicking, sniffing, grunting, sucking, blowing, gasping, sighing, or monosyllabic utterances may be observed.[151] In HD, lip-smacking and grunting (also see below) are frequently reported. In a large cohort of patients with Sydenham's chorea, 8% presented with simple vocalizations (tongue clicking, throat clearing, sniffing) not preceded by premonitory sensations, but in association with facial chorea in most of the patients.[152] It was proposed that the sounds are generated by involuntary choreic activation of pharyngeal and laryngeal muscles.[152]

Continuous Vocalizations Such as Groaning, Moaning, Grunting, and Shrieking

Continuous groaning, moaning, grunting, and shrieking are most frequently associated with neurodegenerative diseases. For example, in dementias, such as Alzheimer's disease[153,154] and others[153,154], to include HD, continuous involuntary vocalizations, often labeled as vocally disruptive behavior, are part of a spectrum of behavioral symptoms that correlate with the severity of cognitive impairment[155] and may be exacerbated with emotional arousal (Video 4A). Purposeless noisemaking, for example, groaning or howling in parkinsonism[156] (Video 4B), including PSP[157-159] (Video 4C), may also be encountered. We also documented continuous shrieking in a patient with acquired hepatocerebral degeneration during an episode of acute encephalopathy (Video 4D). Finally, functional neurological disorders may also present with continuous sounds, such as grunting (Video 4E) or shrieking (Video 4F).

Pathological Laughter and Crying

Laughter and crying behaviors that occur detached from emotional content were reported in patients with TS as part of tic behaviors[160] and other neurodevelopmental disorders (eg, Angelman syndrome,[161] partial trisomy 16p,[15] and Rett-like syndromes[162]). However, pathological laughter and crying is most commonly associated with neurodegenerative disorders, such as ALS,[163] FTD,[164] Alzheimer's disease,[165] primary progressive aphasia,[166] multiple system atrophy cerebellar type,[167] CJD,[168] spinocerebellar ataxia (SCA) 17,[169] and HD. Focal brain lesions in cerebrovascular disease,[170-177] traumatic brain injury,[178-180] autoimmune-mediated lesions in disseminated encephalomyelitis[181-183] or drug-induced behavior[184] are additional etiologies. Finally, recurring "automatic" laughter was also reported as part of ictal phenomena (gelastic seizures).[185,186]

Vocalizations Resembling Physiological Reflexes

Typical vocalizations related to physiological reflexes are sniffing, throat clearing, belching, and wheezing, whereby these audible sounds are mediated by supraglottic structures without involvement of the larynx. Sniffing and throat clearing are noises that are frequently encountered as habitual behaviors (eg, throat clearing in concert halls) and as simple vocal tics in patients with TS.[66] Persistent coughing as a vocal tic can be misinterpreted as disease of the upper and lower airways.[187,188] An extraordinary cause of belching was seen in a patient with parkinsonism, who suffered from a disturbance of esophageal motility with consecutive belching during OFF-periods that remitted with levodopa intake.[189] Persistent hiccups were reported after ischemic lesions of the brain stem.[190,191] Sounds such as coughing or throat clearing may also present either as ictal phenomena[192,193] or "seizure-ending signs."[194] Belching in combination with aerophagia was described in a patient following herpes simplex encephalitis.[195] Sniffing, coughing, belching (Video 5A), and hiccup-related sounds (Video 5B) were also documented in functional neurological disorders.[52,196] Other physiological involuntary

vocalizations are "contagious yawning"[197] or groaning during sexual intercourse.[198]

Others

This group encompasses involuntary vocalizations that may not clearly belong in any of the previous categories and may represent distinctive phenomena of specific etiologies. For example, patients with culture-bound startle syndromes, such as Latah (also see section on palilalia), typically vocalize following a loud external stimulus.[97] Patients with functional movement disorders may also show similarly exaggerated stimulus-triggered responses (Video 6). This type of vocalized startling differs from the classic motor startle response in hyperekplexia. In the classical hereditary forms of hyperekplexia, the latency of the stereotypic spread of muscle activation is very short, whereas in the neuropsychiatric forms the latency is longer and includes a secondary phase with vocalization.[199]

Another important category encompasses ictal phenomena. Ictal vocalizations (also see previous sections) may inherently cover the entire tenor of possible sounds and phonemes of humans: from the classic "ictal cry," signifying the beginning of generalized tonic-clonic seizures,[200] over echo-, pali-, and coprolalic[145,201-204] behaviors, to animal noises ("bleating of sheep," barking),[205,206] singing, and humming.[207-209] Of note, weeping, moaning, and coughing may also be encountered in nonepileptic seizures.[200]

A final category includes noisemaking during sleep. In addition to common snoring, which is the result of obstructed air movement in the upper airways leading to vibration of the soft palate and posterior faucial pillars,[210] other sleep-related sounds include strictly expiratory groaning and moaning, known as catathrenia.[211] In neurodegenerative disorders, such as multiple system atrophy (MSA)[212] or SCA17,[213] stridor during sleep is a common

feature. In anti-IgLON5 syndromes, a prominent stridor in association with REM sleep behavior disorder (RBD) does frequently occur.[214] RBD itself may also be associated with vocalizations such as laughing, talking, shouting, and swearing. It has been described in TS[215] and autism.[216] Most commonly, however, RBD occurs in neurodegeneration (eg, α-synucleinopathies,[217-220] tauopathies,[219,221,222] and others[223-228]; see Table 1). RBD was also reported as a result of focal brain lesions, particularly within the brain stem following stroke[229-231] or due to tumors,[232] and in autoimmune disorders, such as multiple sclerosis,[233] Guillain-Barré syndrome,[234] and paraneoplastic encephalitis.[235] It has also been described in association with narcolepsy,[236] epilepsy,[237] and posttraumatic stress disorder.[238] Finally, vocalizations during sleep can be related to night terrors[239] or sleep-related hypermotor seizures.[240] Figure 1 provides a diagnostic algorithm on how to etiologically approach the different involuntary vocalizations described here.

Pathophysiology of Involuntary Vocalizations

The physiology of vocalizing behaviors relies on a well-coordinated network of respiratory, laryngeal, and supralaryngeal muscles.[241] The motoneuronal pool underlying the innervation of these motor effectors is widespread between pontine segments of the brain stem (eg, for the control of jaw-closing muscles) over to motor neurons of the upper lumbar spinal cord (eg, innervation of abdominal muscles).[241] The coordination of this extensive neuronal network is accomplished by superordinate neural structures, which control and maintain the different elements of vocalizing behaviors to include vocal reflexes (eg, shrieking or crying as a result of a painful stimulus), imitative vocalizations, and human speech.[242] Extensive research in a wide range of mammals, including humans, has revealed 2 basic networks underlying vocalization behaviors with overlapping output structures.[242] A cingulo-periaqueductal network has been associated with
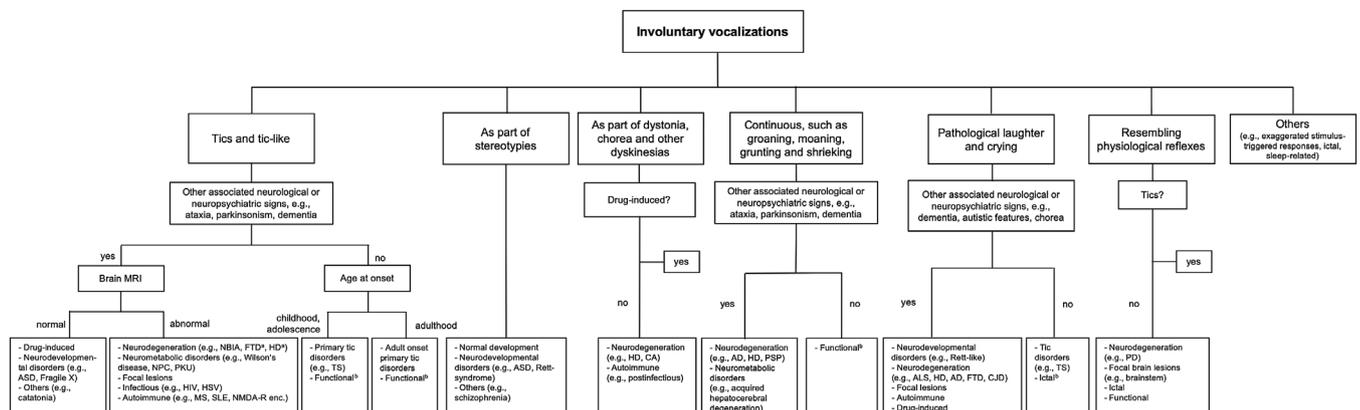

FIG. 1. Diagnostic algorithm for the approach of patients with involuntary vocalizations. [a]MRI might be normal, [b]may also occur with other neuropsychiatric or neurological signs. AD, Alzheimer's disease, ALS, amyotrophic lateral sclerosis, ASD, autism spectrum disorder, CA, chorea-acanthocytosis, CJD, Creutzfeld-Jacob Disease, FTD, frontotemporal dementia, HD, Huntington's disease, MS, multiple sclerosis, NBIA, Neurodegeneration with Brain Iron Accumulation, NPC, Niemann Pick type C, PD, Parkinson's disease, PSP, progressive supranuclear palsy, SLE, systemic Lupus erythematodes, TS, Tourette syndrome. Also refer to table 1 for complete list of etiologies associated with involuntary vocalizations.
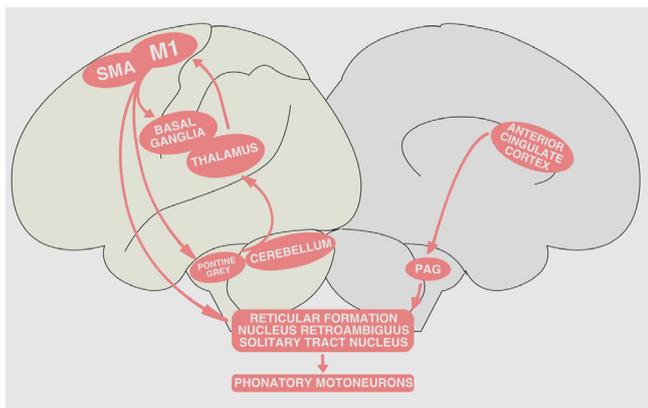
FIG. 2. Simplified representation of the 2 main functional neuroanatomical networks involved in vocalizing behaviors. Left, a motor-cortical-striato-thalamo-cortical network underlying control of learned vocalizations (eg, speaking and singing). Right, a limbic cingulo-periaqueductal network regulating gating control of patterned reflex vocalizations (eg, nonverbal emotional responses, such as crying or moaning). Adapted by references 241 and 242. PAG, periaqueductal gray; SMA, supplementary motor areas; M1, primary motor cortex. [Color figure can be viewed at wileyonlinelibrary.com]

the control of patterned vocalizations related to the gating of reflexes, such as nonverbal emotional responses (eg, crying, moaning, shrieking, and laughing). The supplementary motor area together with the motor cortex, the cortico-striato-thalamo-cortical pathways, and a wider network extending to the pontine gray and cerebellar pathways regulate fine motor control and learned vocalizations, such as the ability to speak and sing. Figure 2 provides a simplified representation of the key neural structures underlying human vocalizing behaviors.

Unfortunately, despite the advances in the field of vocalizations, most pathological phenomena reported here remain scientifically understudied. However, phenomenological observations and cross-species comparative behavioral and neuroanatomical studies, including lesions and chemical and electrical stimulation protocols (reviewed in reference 242) provide important insights into the neuronal structures involved in the different pathologies we present here. Research in tic and tic-like vocalizations implicates 2 key pathophysiological mechanisms for repetitive vocalizations. First, local disinhibition within the cortico-basal ganglia-thalamo-cortical loops that control motor behavior is suggested to lead to amplified output gain. This has been demonstrated in primate and rodent models of tic-like behaviors[243,244] and was further supported by neuropathological studies in patients with TS.[245,246] A single study examined the neuronal locus of disinhibition to produce repetitive grunting sounds, labeled as tic-like behaviors in monkeys, and highlighted the characteristic involvement of the nucleus accumbens and the anterior cingulum, as part of the cingulo-periaqueductal network, underlying these behaviors.[247] As tic vocalizations range from simple nonverbal utterances, such as sniffs or grunts, to words and complete sentences, it is

likely that structures of both the cortico-striato-thalamo-cortical and the limbic cingulo-periaqueductal networks are involved in the generation of vocal tics. In turn, pathologically increased output gain, including vocalizations, is further selectively reinforced through enhanced stimulus-response learning via dopaminergic input — here, vocalizing tics receive behavioral salience.[248] The efficacy of antidopaminergic medication (also see below) to treat tic vocalizations corroborates the pathophysiological role of reinforcement learning. Most importantly, disinhibition and enhanced reinforcement learning may either be the result of a neurodevelopmental disorder, as in primary tic disorders, or due to brain damage, as in frontal lobe syndromes or neurodegeneration (also see Tics and Tic-Like Vocalizations section).

It remains unclear why in certain conditions, as in HD, for example, vocal tics such as grunting may often be very specific. In one account, the most commonly employed motor programs would also have the highest probability of being part of tic behaviors. For example, in primary tic disorders, patients mostly exhibit their tics at the motor effectors, which they most commonly employ in their daily living (eg, blinking). In light of the phenomenological overlap between choreic involuntary vocalizations, which may also lead to expiratory gasping, sniffing, or grunting, this view predicts that patients with choreic grunting would also have a high probability of developing grunting tics. Indeed, a clear clinical distinction between choreic grunts and grunting tics may in many cases be notoriously difficult (Video 1E,F vs Video 1G).

Different to tics, vocalizations as part of stereotypies remain less well explored. Certain clinical facts, as for example the absence of a premonitory urge in stereotypies and their typically continuous nature, imply distinct functional neuroanatomical correlates, even though the cortico-basal ganglia-thalamo-cortical circuitry has also been involved.[249-251] The fact that stereotypies can frequently be observed in both humans and animals during confinement and sensory isolation also highlights the significance of self-stimulation in their emergence and maintenance.[252,253]

The pathophysiology of involuntary sounds as part of dystonia, chorea, and other dyskinesias, is intrinsically related to the nature of the involuntary movements[254-256] and is beyond the scope of this article. Indeed, the vocalizing sounds are the result of involuntary activation of structures related to the respiratory and vocal apparatus, but do not, we posit, involve higher-order neural processes that produce patterned behaviors such as speech. Beyond the few neurodegenerative choreic disorders we have included, most syndromes we have identified are drug-induced, and indeed extensive literature exists about the pathophysiology of drug-induced movement disorders, including vocalizations (for example, reviewed in references [257] and [258]). Video 3C demonstrates lenalidomide-induced vocalizations as part of a choreodystonic syndrome. Only one similar case has been

documented previously.[150] Although the exact mechanism of action remains unclear, we do wish to note the unusual and dramatic side effect of this medication.[259]

One particular category includes continuous vocalizations, including groaning, moaning, grunting, and shrieking. We previously published a case (Video 4C) in which we highlighted the role of distinct neural generators in vocalizing behaviors. We postulated that continuous groaning could be the result of ongoing activation of the cingulo-periaqueductal circuit, described above (also see Fig. 2), as a result of either enhanced excitation, reduced top-down inhibition, or both.[159] Given the common denominator of many of the disorders we report here linked to frontal lobe damage, we suggest that loss of inhibitory control over a subcortical cingulo-periaqueductal circuit involved in the generation of nonverbal utterances could lead to these types of behaviors.[159,242] Additional factors, such as enhanced limbic drive and dysfunction of the serotonergic system,[260] may further strengthen and/or perpetuate these behaviors. We suggest that the pathophysiology of pathological laughter and crying also falls within this pathophysiological category — with the exception of gelastic seizures as ictal phenomena, which are typically associated with hypothalamic hamartomas.[261,262] Epileptic activity of the frontal lobes, including the anterior cingulate cortex,[263-266] but also parietal[267] and temporal lobes,[268] has also been reported to give rise to gelastic seizures.

Within the group of vocalizations and sounds that resemble physiological reflexes, the most common etiologies are indeed tics (also see above) and functional neurological disorders. The pathophysiology of functional neurological disorders, including movement disorders, has been reviewed before.[269-271] It is important to note that tics, vocalizations as part of stereotypies and vocalizations as part of a functional disorder, are typically distractible. This highlights that for these particular vocalizing behaviors, superintending centers related to attention and potentially motivation can alter the output gain based on environmental context.

In ictal vocalizations, the behavioral abnormality depends on the cortical locus of abnormal neuronal excitation. For example, seizures over the temporal lobe typically elicit various types of different vocalizations, such as animal noises, coprolalia, throat clearing, and belching.[192,193,201,205] Similar vocalization behaviors have also been described for epileptic discharges over mesiofrontal brain areas, including the supplementary motor area and the anterior cingulate cortex.[242,272]

Finally, vocalizations in REM sleep disorder are suggested to result from dysfunction of the nucleus subcoeruleus and/or the reticular formation, whose glutamatergic, GABAergic, and glycinergic projections fail to inhibit spinal motor neurons, and thus muscle atonia is no longer induced.[273] Neurodegenerative disorders, such as PD,[274] MSA, DLB, and PSP,[275] with abnormalities in REM sleep behavior typically affect these structures, and

indeed the reticular formation is a key structure for the activation of the motor neuronal pool involved in vocalizations (Fig. 2).

Treatment Options

Within the range of the different involuntary vocalizations, the treatment strategy depends on the vocalization type and the underlying etiology. However, beyond the treatment of tics, therapeutic interventions in other types of vocalizations are mostly based on case series and single case reports. For tic vocalizations, as in the example of primary tic disorders, there are 3 main therapeutic venues: (1) behavioral treatments, including habit reversal training and its expansion, the comprehensive behavioral intervention for tics (CBIT) (for a review, see reference 276; (2) pharmacological interventions, such as antipsychotics, dopamine-depleting agents, α2-agonists, and more recently cannabinoids[277-279] (3) surgical interventions for refractory cases, such as deep brain stimulation.[280] In addition, local injections of botulinum toxin might also alleviate symptoms.[281] Single case reports have indicated that other medications might also be helpful. For example, fluoxetine was used to control laughing tics in TS.[160] However, the efficacy of these treatments remains understudied. An important caveat is the treatment of tic-like behaviors in functional neurological disorders in which behavioral therapies should be preferred over pharmacological agents.[50-52,54,282]

In klazomania, particularly in the presence of depression and anxiety, benzodiazepines showed some therapeutic promise in 1 case, whereas quetiapine, risperidone, aripiprazole, amitriptyline, and sertraline were ineffective.[57] Electroconvulsive therapy was also reported to be effective in 2 patients with klazomania and depression.[57,58]

Treatment reports specifically targeting pali-, echo-, and coprolalia are particularly rare. Palilalia in vascular dementia was responsive to the antidepressant trazodone.[283] In some cases of echo- and coprolalia, benzodiazepines led to the alleviation of symptoms.[94,123] The amphetamine-related drug fenfluramine was efficient in the reduction of echolalia in 10 patients with ASD.[107] Echolalia in a case with a left temporoparietal hemorrhage and a case with a diagnosis of Rubinstein-Taybi syndrome improved after behavioral therapy.[107,284] We believe that behavioral therapy should be a first-line option in patients with repetitive vocalizing behaviors, such as pali-, echo-, or coprolalia, but also in cases with vocalizations as part of stereotypies. However, in some of these cases, particularly in the presence of additional behavioral abnormalities, pharmacological augmentation may be necessary.

In vocalizations as part of dystonia, chorea, or other dyskinesias, the most common etiology is drug-induced. In these cases, the causing agent should be removed if possible, or dosage should be reduced. In addition, the prescription of dopamine-depleting agents might be helpful.[2]

TABLE 2. Treatment options for involuntary vocalizations

Vocalization	Treatment option
Tics and tic-like vocalizations	Behavioral therapy — HRT, CBIT — ERP Pharmacological treatments — Antipsychotics (eg, aripiprazole, risperidone, olanzapine) — α2-Agonists (eg, clonidine, guanfacine) — Dopamine-depleting drugs (eg, tetrabenazine) — Cannabinoids — Botulinum toxin — Others (eg, baclofen, topiramate) DBS Electroconvulsive therapy (for klazomania)[a]
Vocalizations as part of stereotypies	Behavioral therapy (eg, CBT) Pharmacological treatments — Antipsychotics (eg, haloperidol, risperidone, olanzapine) — Antidepressants (eg, SSRI such as fluoxetine, SNRI such as sertraline, citalopram) — Botulinum toxin
Vocalizations as part of chorea, dystonia, and other dyskinesias	Pharmacological therapy — Reduce offending agent if possible — Dopamine-depleting drugs (eg, tetrabenazine, deutetrabenazine)
Continuous vocalizations such as groaning, moaning, grunting, and shrieking	Behavioral therapy — Practical interventions (eg, relieve discomfort, provide orientation, avoid excess attention to vocalizing behavior) Pharmacological therapy — Benzodiazepines (eg, lorazepam) — Antipsychotics (eg, risperidone) — Antidepressants (eg, tricyclic such as doxepin, SSRI such as paroxetine, citalopram, SARI such as trazodone) — β-blockers (eg, propranolol)
Pathological laughter and crying	Pharmacological therapy — Antidepressants (eg, tricyclic such as doxepin, SSRI such as paroxetine, citalopram, and other such as venlafaxine, mirtazapine, or reboxetine) — Dopaminergic drugs (eg, levodopa) — NMDA-receptor antagonists
Vocalizations resembling physiological reflexes	Behavioral therapy

CBIT, comprehensive behavioral intervention for tics; CBT, cognitive behavioral therapy; DBS, deep brain stimulation; ERP, exposure and response prevention; HRT, habit reversal training; NMDA, N-methyl-ᴅ-aspartate; SARI, serotonin antagonist and reuptake inhibitor; SNRI, serotonin-norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor.
[a]Two case reports.

Although being a huge burden in hospitals and nursing homes, specific treatment for continuous vocalizing behaviors, such as those associated with neurodegeneration, is poorly investigated. A detailed assessment about whether other circumstances such as physical or mental suffering (pain, discomfort, fatigue, frustration, depressed mood, deprivation, etc.) could elicit or precipitate the vocalizing behavior is recommended. The recognition and removal of these factors could lead to a remission of vocalizing behaviors. In addition, behavioral interventions such as avoidance of positive reinforcement of vocally disruptive behavior could be helpful.[285] Pharmacological approaches include tranquilizers, antipsychotics, anticonvulsants, antidepressants, and beta-blockers, however, with mixed responses.[154] In the absence of randomized, controlled studies, the antidepressants paroxetine,[286] citalopram,[287] trazodone,[288,289] and doxepine[290] were shown to reduce vocalizing behavior in single cases and case series. Although reported to be the most effective,[155] benzodiazepine[285] intake should be monitored with caution to maintain functionality and mobility. In cases with concomitant aggression, antipsychotic medication could be helpful, and in patients with comorbid depression or anxiety, the usage of antidepressants is preferable.[285] Pathological crying after brain injury was reported to be well controlled with paroxetine and citalopram in a large case series.[291] Intractable hiccups responded well to inhaled cannabis in a patient with AIDS.[292] Table 2 provides a comprehensive overview of treatment options in involuntary vocalizations.

Conclusion

We here presented the wide range of involuntary vocalizations in humans, together with 29 video-documented cases to exemplify their phenomenology. Based on these cases and on the extensive literature review, we provide a diagnostic algorithm to guide clinicians in approaching patients with involuntary vocalizing behaviors (Fig. 1), discuss their pathophysiology, and provide treatment options, where available. We do recognize that some of the behaviors that we document reflect sounds emitted from supraglottic structures, rather than true vocalizations generated from the vocal cords, and have clearly documented the differences between these phenomena. Also, we are aware that the classification of some of the vocalizations we present as involuntary (eg, tics) may be open to criticism. However, we do suggest that several of their qualities, for example, their inflexible, repetitive, and socially inopportune character, as well as their perception as unwanted and often distressing phenomena, guarantee a minimal involuntary component. Our algorithmic approach may not cover every possible clinical presentation of involuntary vocalizations and its respective etiology. Nevertheless, we do hope that it provides a clear framework to guide clinicians in their diagnostic considerations. This, in turn, will translate to improved pathophysiological understanding and appropriate management of these paradigmatic neuropsychiatric patients.

Legends to the Videos

Video 1. Tics and tic-like vocalizations. (A–C) Vocalizations in TS. (A) Multiple vocal tics including whistling,

grunting, sighing, palilalia ("ja, ja, hallo, hallo, hallo," ie, "yes, yes, hello, hello, hello") and coprolalia ("scheiße"). Motor (facial twitches) and vocal tics (humming) started at age 12. Tics were preceded by premonitory urges and were suppressible on demand. The patient was also diagnosed with obsessive-compulsive disorder, attention deficit hyperactivity disorder, depression, and anxiety disorder. (B) Tic vocalizations including nonsensical sounds, words ("der Kampf") and phrases ("Hilfe, L... stirbt") including coprolalia. Tic behaviors first appeared at age 5, waxed and waned over time, were preceded by premonitory urges, and could be voluntarily suppressed. (C) Bout of grunting, throat clearing, and coughing tics in a patient with TS. Motor and vocal tics were present since the ages of 5 and 12 years, respectively, and waxed and waned with time. Tics were preceded by premonitory urges and could be voluntarily suppressed. Severe obsessive-compulsive and major depressive disorder were also diagnosed. (D–F) Vocalizations in HD. (D) Shrieking, sniffing and shouting tics. (E) Characteristic repetitive grunting tics and sniffing sounds. The patient described a mounting urge sensation in his larynx preceding and leading to the release of these sounds. (F) Grunting, throat clearing, and coughing tics (previously published[293]). The involuntary phenomena could be suppressed for a few seconds until an unpleasant tension and tightness led to their continuation. (G) Laughter, rasping sounds, grunting, hissing, snorting, and palilalic utterance of nonsensical words ("upsa") in monozygotic twins with chorea-acanthocytosis (previously published without video material[20]). (H) Drug-induced (risperidone and methylphenidate overdose) lip-smacking tics in a patient with schizophrenia. He was able to briefly voluntarily suppress the repetitive lip-smacking movements but experienced an increasing urge to release them. Treatment with tetrabenazine improved the repetitive behaviors. (I) Involuntary shouting (klazomania) in a patient with TS. Eye blinking was the first tic at age 10, followed by multiple waxing and waning motor and vocal tics. Over 2 years the patient presented a complex pattern of motor and vocal tics with repetitive foot stamping, flailing movements of the arms, and grimacing alongside bouts of loud shouting. Severe obsessive-compulsive disorder and self-injurious behavior (hitting his head, pressing against his eye, scratching) were also present. (J) Recurrent shouting (klazomania) in a patient with functional disorder (previously published without video material[294]). The patient first developed sudden jerks of the head, neck, and left arm combined with involuntary vocalizations such as screams, yelps, and grunts a few days after a minor traffic accident at age 33. Sudden movements and screams were not preceded by premonitory urges, were not suppressible, and were triggered by unexpected bright lights or taps, stress, and anger, but also occurred spontaneously. Neurophysiological

analysis of startle-induced behaviors showed variable patterns of muscle activation and prolonged activation latencies. (K) Stuttering in a patient with Parkinson's disease and deep brain stimulation (DBS) in DBS-OFF (K-1) and DBS-ON (K-2) conditions. (L) Echolalia ("mit mir," ie, "with me") in a patient with Niemann-Pick type C. (M) Echolalia ("Christmas," "ice cream," "bugger") in a patient with a functional neurological disorder. She presented with jerks, which first started in her right arm during a driving lesson 2 years earlier and then spread over her whole body. During the same period, she began to repeat words spoken by other people (echolalia) and imitate other people's actions (echopraxia). Movements and vocalizations, although sometimes preceded by inner tension, could not completely be inhibited voluntarily. However, they were distractible. Sudden spontaneous jerking during walking was also documented (previously published[295]). (N) Repetitive continuous swearing ("functional coprolalia") in a patient with functional neurological disorder and a previous diagnosis of TS. The repetitive swearing ("Hure") occurred in bouts and over prolonged periods and was context dependent, that is, triggered only when the patient met his previous partner or discussed her. During the same period, he also developed a functional gait disorder, which he described as the inability to walk as a result of "extreme tension" that lasted for a period of 2 years and resolved spontaneously.

Video 2. Vocalizations as part of stereotypies. (A) Stereotypic vocalizations accompanied by motor stereotypies (repetitive touching of the right ear) in a patient with autism spectrum disorder, before (A-1), during (A-2), and after (A-3) treatment with botulinum toxin of the vocal cords. (B) Stereotypic shouts accompanied by motor stereotypies (flexion-extension movement of the upper extremity) in a patient with 15q13.3 microdeletion syndrome and cognitive disability, impulsivity, short stature, cachexia, and mitral valve insufficiency. The stereotypic behavior developed 4 years earlier during a stressful period. The patient reported a soothing character of the repetitive shouts and movements, which reduced a feeling of inner distress. The behavior was distractible, although the patient felt that she was not able to suppress the movements and vocalizations.

Video 3. Vocalizations as part of chorea, dystonia, and other dyskinesias. (A) Lip smacking in a patient with tardive dyskinesia. (B) Panting and gasping in a patient with tardive dyskinesia due to chronic metoclopramide intake. (C) Acute-onset hissing and shrieking in a patient with generalized choreodystonia subsequent to lenalidomide treatment for multiple myeloma.

Video 4. Continuous groaning, moaning, grunting, and shrieking. (A) Repetitive shouting associated with emotional discomfort in a patient with HD. (B) Continuous howling in a patient with parkinsonism and dementia. (C) Continuous groaning in a patient with PSP

(previously published[159]). (D) Continuous shrieking in a patient with acquired hepatocerebral degeneration during an acute encephalopathic episode (D-1) and after treatment (D-2). (E) Continuous grunting with distractibility (E-1) in a patient with a functional neurological disorder. The continuous vocalizing behavior, which was distractible and entrainable, began abruptly 4 years ago and was perceived as involuntary. It remitted during talking, eating, and drinking. (F) Continuous shrieking in a patient with functional neurological disorder (previously published without video material[294]). Repetitive inspiratory shrieking associated with facial grimacing, eye closure, and variable jerks of the head and upper extremities triggered by unexpected, loud noises or also occurring spontaneously. These behaviors, which were not preceded by premonitory urges and were not suppressible, appeared 1 year after a head injury as a result of a traffic accident. Comorbid anxiety disorder with panic attacks and forgetfulness were noted.

Video 5. Vocalizations resembling physiological reflexes. (A) Air gasping and belching as a result of functional aerophagia in a patient with functional neurological disorder. She described suffering from anxiety episodes, which led to aerophagic behaviors with subsequent gastric distention and belching. (B) Recurrent hiccup-like sounds in a patient with functional neurological disorder. These appeared abruptly following an episode of severe diarrhea after food poisoning. She could voluntarily suppress the hiccup-like sounds by bending over or pressing the arms against the abdominal wall, but otherwise felt that she had no control over them. Hiccup-like vocalizations remitted during eating and drinking.

Video 6. Other involuntary vocalizations. Exaggerated pseudo-startle response with shouting and hissing in a patient with functional neurological disorder. There was a variable pattern of muscle recruitment and vocalizations throughout examination, following acoustic and light tactile stimuli over different body areas, but also preceding those. An irregular and frequency-variable tremor of both arms, which was distractible and entrainable, was also noted. "Huffing and puffing" and other effortful behaviors were documented during neurological examination.■

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Supporting Data

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