



HHS Public Access

Author manuscript

Mov Disord. Author manuscript; available in PMC 2024 April 01.

Published in final edited form as:

Mov Disord. 2023 April ; 38(4): 551–557. doi:10.1002/mds.29362.

Redefining Bradykinesia

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Keywords

bradykinesia; Parkinson's disease; atypical parkinsonism

Recognizing and characterizing bradykinesia is a critical issue in movement disorders. Bradykinesia is the core symptom for the definition of parkinsonism and for the diagnosis of Parkinson's disease (PD) and atypical parkinsonism (AP).^{1–7} To some extent, however, the debate on the terminology and definition of bradykinesia has never been settled.^{8,9} Bradykinesia literally means slowness of movement. However, the term is still used interchangeably to indicate low amplitude movement (hypokinesia) or no movement (akinesia),¹⁰ and it is often used to apply to both voluntary and spontaneous/automatic movements.^{11–13} The current bradykinesia definition in the context of diagnostic criteria for PD includes both slowness and progressive reduction in movement amplitude and velocity when performing repetitive movements, that is, decrement or sequence effect.^{3,4,10}

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Author Contributions

Author roles: 1. Research project: A. Conception, B. Organization, C. Execution; 2. Manuscript Preparation: A. Writing of the first draft, B. Review and Critique. MB: 1A, 1B, 1C, 2A AE: 1A, 2B AF: 1A, 2B GP: 1A, 1B, 2B MH: 1A, 2B AB: 1A, 2B.

Conflict of Interest

None of the authors have any potential conflicts of interest to disclose.

Relevant conflicts of interest/financial disclosures: Nothing to report.

The current concept of bradykinesia, including potentially distinct motor abnormalities, is reflected in the evaluation scales routinely used in clinical practice.^{14,15}

The current bradykinesia definition has some clinical and pathophysiological inconsistencies.^{10,16,17} In this paper, we first review the historical background of the definition of bradykinesia and related terms. We then summarize the various inconsistencies that undermine the current bradykinesia definition and its applicability in different conditions and propose redefining bradykinesia. We finally emphasize the usefulness of a new bradykinesia definition and discuss the potential implications of this proposal.

Historical Background on Terminology

The terminology of bradykinesia, hypokinesia, and akinesia has long been debated.⁸ Interestingly, none of these terms were first introduced with specific reference to parkinsonism. The term bradykinesia was introduced in 1907 to describe slowed voluntary movements in dystonia.¹⁸ About 20 years later, the so-called “bradykinetic syndrome” has been described in PD and other conditions to specifically refer to slowness at the beginning and during the execution of voluntary movements.¹⁹ The term hypokinesia was introduced in 1878²⁰ as opposite to hyperkinesia.⁸ In the context of PD, hypokinesia was used for the first time in the early 1900s, with reference to the lack of “associated, successive, reactive and expressive movements.”²¹ Later on, it was suggested that hypokinesia could be adopted only when an amplitude decrement was present.²² The term akinesia was introduced in the second half of the 19th century to describe decreased motion, including paralysis. Subsequently, the term akinesia has been mainly adopted in psychiatric patients,²³ and it was used for the first time in PD only in 1870, to indicate the “paralysis” occurring in the advanced disease stages.²⁴

In the early 1990s, Gibb and Lees defined bradykinesia as “slowness of voluntary movement with a progressive reduction in speed and amplitude of repetitive actions.”^{3,25} Accordingly, the current diagnostic criterion for PD⁴ defines bradykinesia as “slowness of movement AND decrement in amplitude or speed (or progressive hesitations/halts) as movements are continued,” due to two main clinical assumptions: (1) these signs are generally present during the examination of a PD patient and (2) in parkinsonism caused by PD, a decline in either speed or amplitude or both is seen as repetitive movements are continued, a feature usually not observed in other parkinsonisms.⁴ The concept of bradykinesia is even more complicated in AP.¹⁰ The diagnostic criterion for progressive supranuclear palsy (PSP)⁵ indicates akinesia, not bradykinesia, as a core feature and used this term synonymously with parkinsonism. The criteria for multiple system atrophy (MSA)¹ diagnosis require bradykinesia to be associated with rigidity and tremor, without specifying what features would qualify as bradykinesia. Finally, the criteria for corticobasal syndrome (CBS)² use bradykinesia and akinesia interchangeably. Only the clinical criteria for dementia with Lewy bodies (DLB)⁶ refer to bradykinesia as slowness of movement and decrement in amplitude or speed as in PD.

Critical Issues with the Current Definition of Bradykinesia

Clinical Inconsistencies

First, the conceptualization of bradykinesia according to the current criteria associates this specific movement disorder with parkinsonism.⁴ This peculiarity does not apply to other disorders of movement, that is, dystonia, chorea, myoclonus, and tremor, which may instead underlie a wide spectrum of etiologies with no a priori restriction.^{26,27} In this regard, it should also be acknowledged that the current definition of bradykinesia does not necessarily apply to all cases of parkinsonism. Clinical experience indicates that bradykinesia features can be variable in patients, even in those who have a clear diagnosis of PD.

Another important issue of bradykinesia concerns AP. Although a decline in velocity or amplitude is usually seen with ongoing voluntary movements in PD, this feature is uncommon in other parkinsonisms.^{4,10,28} PSP is usually characterized by a marked reduction in velocity and amplitude without a sequence effect.^{28,29} Note that in PSP, or PD patients when there is marked hypokinesia, the amplitude of movement may be reduced, and the number of taps appears to increase during the task, a phenomenon also referred to as tachykinesia.¹³ In these cases, however, the speed at which the individual movements are performed is nonetheless slow. Thus, the current definition of bradykinesia, which includes a sequence effect, does not necessarily fit all cases of parkinsonism. Again, studies in patients with MSA, CBS, or DLB are too limited to allow even preliminary conclusions for these conditions.²⁸ However, it should be emphasized that these other degenerative parkinsonisms have motor signs that hamper the motor assessment, for example, the presence of bradyphrenia in DLB, myoclonic jerks in MSA, and apraxia in CBS.^{30,31}

If we broaden the field of observation and include non-parkinsonian neurological disorders, several clinical studies specifically refer to the presence of bradykinesia in these disorders.¹⁷ Examples of these conditions include motor neuron diseases, psychiatric conditions, other neurodegenerative diseases, and even some hyperkinetic disorders like dystonia (for which the term was originally coined), chorea, and essential tremor (ET),¹⁷ where slowness of movement has even been included among the so-called “soft signs” needed for the diagnosis of “ET-plus.”^{26,32} In these conditions, experimental studies indicate that bradykinesia can be explained by pathophysiological mechanisms that differ from those observed in parkinsonism.^{10,16,33–36} We can therefore question whether its use is appropriate.¹⁷ In particular, the sequence effect is often absent in most non-parkinsonian conditions, although this has not been adequately investigated.¹⁷

In addition, there is a discontinuity between the current bradykinesia definition and the bradykinesia assessment in patients using the MDS-UPDRS.¹⁴ The assessment of spontaneous and voluntary movements in the MDS-UPDRS scale is properly kept separate. Again, concerning voluntary movements the scale requires separate examination for speed, amplitude, hesitations, halts, or decrement (eg, sequence effect). However, it is assumed that the various movement abnormalities have the same weight on the global score. This implies that a subject can be classified as bradykinetic irrespective of the specific abnormality that is identified to score MDS-UPDRS 1 in the specific items, that is, even when there is no evidence of the sequence effect on examination. Finally, concerning

the patient evaluation, the clinical criteria emphasize the presence of limb bradykinesia for parkinsonism definition.⁴ However, clinical and experimental evidence documented that bradykinesia may affect other body regions. Involvement of the face, voice, or walking is missing from the current bradykinesia definition.⁴ For example, although progressive shortening of step length or festination has been reported in PD, and often, though not always, precedes a “motor block” (another term for hesitations/halts), also known as freezing of gait,^{37,38} the sequence effect of voluntary facial movement or the voice has not been documented.^{14,15,39}

Pathophysiological Inconsistencies

A point of criticism regarding the current definition of bradykinesia is to use a single term to denote motor alterations that depend on the impaired control of both spontaneous and voluntary movements, which are instead physiologically distinct. Furthermore, the alterations that fall under the definition of bradykinesia of voluntary movements are often tangled together despite distinct underlying mechanisms. The observation that bradykinesia and hypokinesia often coexist in the same patient is in part explained by biomechanical principles. In healthy individuals there is an almost-linear relationship between movement velocity and amplitude^{40,41}; that is, movement velocity is proportional to movement amplitude, and the relationship is approximately constant over a wide range of values,⁴¹ though the relationship can be lost in PD.⁴² Also, bradykinesia and hypokinesia improve significantly in PD when patients are treated with dopaminergic drugs or deep brain stimulation (DBS) indicating that these two motor abnormalities share a common pathophysiological background.^{43–46} However, bradykinesia and hypokinesia may vary in terms of sensitivity to change in response to dopaminergic medication or surgery, possibly due to the multi-level network effects of these treatments.^{10,45,47–49} Thus, it seems more appropriate to interpret bradykinesia and hypokinesia as separate movement abnormalities.

Other aspects to consider relate to impaired performance of repetitive and continuous movements, that is, the sequence effect. This abnormality has never been documented during spontaneous movements,^{10,39} for example, spontaneous facial expressions or arm pendulum movements during walking, and therefore the current definition of bradykinesia does not fit with spontaneous movement abnormalities in parkinsonism. Again, concerning voluntary movements, the current bradykinesia definition cannot be applied when single movements are tested because the sequence effect can be documented only during repetitive and continuous movements.^{10,16} Nevertheless, experimental findings indicate that in some cases, PD patients may not have a measurable sequence effect during finger tapping or writing.^{10,28,29,46,50,51} Some experimental evidence supports a common pathophysiological mechanism between bradykinesia, hypokinesia, and sequence effect in PD that involves β oscillations at the basal ganglia level.^{52,53} Thus, the increased power of β -band oscillations (and the overall duration of β bursts) possibly represents a fundamental pathophysiological substrate of PD, to which further mechanisms superimpose, culminating in specific motor abnormalities.^{52,53} However, there is also evidence indicating that the pathophysiological mechanisms underlying bradykinesia, hypokinesia, and sequence effect are distinct. The most convincing observation again derives from differences in response to dopaminergic drugs or DBS. Although dopaminergic replacement therapy improves movement velocity

and amplitude, there is no evidence showing that dopaminergic replacement improves the sequence effect.^{10,45,46,50,51,54} Furthermore, experimental studies using neurophysiological techniques or neuroimaging have demonstrated pathophysiological differences between PD patients with and without the sequence effect. These differences are primarily observed at the cortical and cerebellar levels, which are two of the most important nodes in the pathophysiology of PD.^{10,40,45,51,55}

Finally, progressive hesitations/halts specifically refer to increased variability in the regularity and timing of repetitive movements, particularly those characterized by the alternating contraction and relaxation of agonist/antagonist muscle groups.¹² Similar alterations are also frequently observed in cerebellar disorders. Therefore, the interpretation of possible underlying pathophysiological mechanisms remains speculative. For example, it is possible that progressive hesitations/halts in parkinsonism reflect the central timing impairment during the performance of voluntary movements or the pathophysiological involvement of the cerebellum.^{55,56}

Why Redefine Bradykinesia

A new bradykinesia definition would have important practical implications. First, it would allow us to harmonize the concept of bradykinesia with other disorders of movement, where a certain disorder is not necessarily linked to a specific etiology.^{26,27} Again, it would help avoid terminological contradictions that result in confusion in the characterization of patients' motor alterations both in clinical practice and in scientific work. In this regard, considering the various alterations separately, rather than incorporating them into a single term, would make it possible to better characterize a clinical phenotype, ideally with the aid of neurophysiology or novel technologies. Redefining bradykinesia would allow a better evaluation of patients and a more accurate description of the effect of therapies, which is variable in relation to the specific motor abnormality observed.¹⁰

Toward a New Definition of Bradykinesia

We suggest redefining bradykinesia by following three major principles.

Distinguishing voluntary and automatic/spontaneous movements.

Although bradykinesia and its associated features would refer to voluntary movement abnormalities, an alternative term is necessary to specifically refer to automatic/spontaneous movement abnormalities. We propose the term “oligokinesia” to describe the latter phenomena (Table 1).

Dissecting the “bradykinesia complex.”

Bradykinesia-related terms are best used in their original etymological meaning¹⁰ (Table 1). In line with this reasoning, normal movement is indicated by the term eukinesia (Fig. 1). The term bradykinesia would specifically refer to the slowness of voluntary movements, which, however, may be accompanied by associated features, depending on the motor task performed. These include (1) hypokinesia, (2) sequence effect, and (3) hesitations/halts, in variable combinations. Note that in this work, we have used the term

sequence effect instead of decrement. Decrement specifically refers to the progressive decrease of a movement parameter; however, the deterioration of motor performance in PD during repetitive movements may also manifest itself in the form of an increase in the time required to execute the motor performance.¹⁰ We, therefore, believe that the correct term to use, which is all-encompassing, is sequence effect and not decrement. Whenever present, the various movement abnormalities should be separately specifically reported in the description of the clinical phenotype. Therefore, if slowness of movement is present, then there is “bradykinesia.” If a sequence effect is also present, this would be referred to as “bradykinesia with sequence effect.” Additional elements, for example, hypokinesia, hesitations/halts, or oligokinesia, can also be added to complete a full movement description (see Axis I later). The term akinesia should be used for conditions in which the assessment is marred by the patient’s inability to perform voluntary movement, and therefore a more detailed phenomenological description is not possible.

Establishing a dual-axis approach.

Redefining bradykinesia according to the aforementioned principles implies that the phenomenological description of the patient is valid, regardless of the underlying etiology. As is already the case with other movement disorders,^{26,27} this description can fit in the first of two distinct axes for the approach to bradykinesia: Axis I, which describes the major phenomenology of bradykinesia and related terms in a given patient, and Axis II, which addresses the etiology (Table 2). Axis I may include (1) type of movements (voluntary vs. spontaneous movement and single vs. repetitive movements), (2) major movement features, (3) body distribution (limbs and/or axial districts), and (4) possible associated features (isolated and/or combined with other neurological disorders). Axis II may instead describe the possible etiology underlying the motor abnormality, that is, (1) parkinsonism, (2) non-parkinsonian conditions (due to a known specific cause), or (3) unknown etiology.

Tentative Implications and Conclusions

In this paper we addressed the general structure of a new approach to defining bradykinesia, and we analyzed the phenomenological characteristics of bradykinesia (Axis I). We suggest that when there is a combination of motor alterations, that is, bradykinesia with sequence effect and any additional features, the clinical picture is highly suggestive of parkinsonism. In contrast, isolated bradykinesia is a non-specific finding that may be present in various non-parkinsonian conditions of known or unknown etiology. Further studies will be needed to further clarify the relationships between Axis I and Axis II for the purpose of bradykinesia definition. The combination of these two sets of descriptors may provide meaningful information on any bradykinetic patient, avoid the inclusion of distinct motor alterations with different pathophysiological backgrounds in one single term and the use of inconsistent terminology, and serve as a basis for the development of better research and treatment strategies.

It should be noted that the approach to the definition of bradykinesia proposed here may be applicable to both clinical and experimental settings. Although bradykinesia, hypokinesia, sequence effect, hesitations/halts, akinesia, and oligokinesia can be identified

in many patients on clinical grounds only, there is a proportion of patients in whom these abnormalities can be hard to distinguish. Laboratory methods of objective movement quantification, such as kinematic analysis, could be applied in these cases.^{57,58}

In conclusion, the proper recognition and classification of bradykinesia and related features, that is, the “bradykinesia complex,” may improve further research efforts and increase the accuracy of its use in distinguishing different parkinsonisms from one another and between parkinsonian from non-parkinsonian conditions.

Acknowledgment:

The authors wish to thank Marina Milano (marina.milano3@gmail.com) for the graphic design of Figure 1. Open Access Funding provided by Università degli Studi di Roma La Sapienza within the CRUI-CARE Agreement.

Data Availability Statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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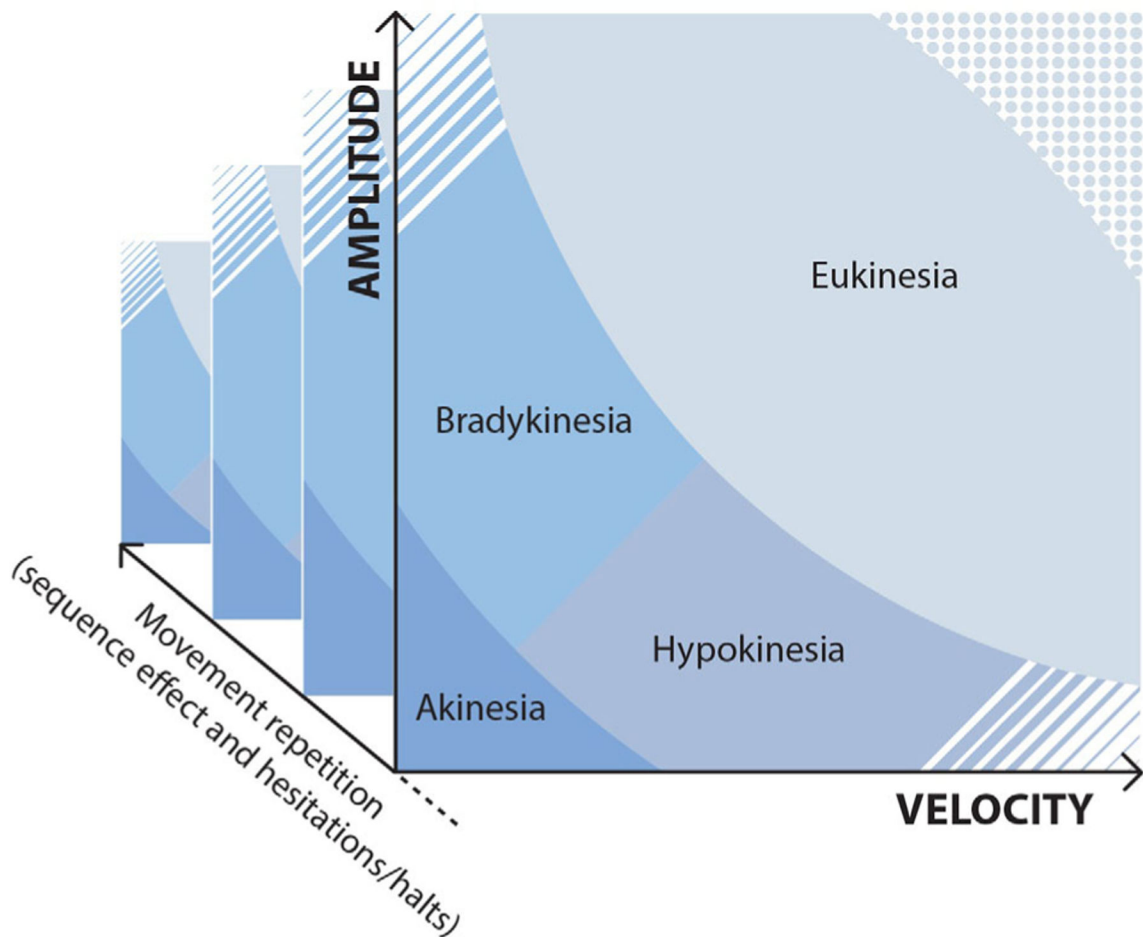


FIG. 1.

The figure schematically represents the two main dimensions of voluntary movement, namely velocity (x -axis) and amplitude (y -axis). Based on these two values, the movement can be considered predominantly bradykinetic if of reduced velocity or hypokinetic if of reduced amplitude. If both parameters are markedly reduced, the movement is defined as akinetic. Conversely, amplitude and velocity may be within a normal range (eukinesia). In some cases, velocity and amplitude may have values that exceed normal limits, as observed in some hyperkinesias, for example, ballismus. The dashed area at the lower right indicates markedly reduced amplitude but high velocity movements. The dashed area at the upper left indicates markedly reduced velocity but high amplitude movements. Dotted and dashed areas overall indicate unlikely movement values. With the repetition of the movement (z -axis), changes in motor performance can be observed in terms of amplitude and/or velocity reduction (sequence effect) or motor hesitations/halts. Note that although in this figure distinct limits are depicted to differentiate the various areas, these limits may be blurred in the reality. Again, the limits for the definition of movement abnormalities are arbitrary and may be modified in future experimental studies, though the conceptual framework of the new definition of bradykinesia elaborated here should remain unchanged.

TABLE 1

The “bradykinesia complex”

Feature	Definition	Types of movements being affected
Bradykinesia	Reduced velocity of movements	Single or repetitive, alternating and continuous movements (limbs and body axis)
Hypokinesia	Reduced amplitude of movements	Single or repetitive, alternating and continuous movements (limbs and body axis)
Sequence effect	Progressive reduction in amplitude and/or velocity	Repetitive, alternating, and continuous movements (limbs)
Hesitations/halts	Irregularities in movement timing	Repetitive, alternating, and continuous movements (limbs)
Akinesia	Inability to perform a movement	Single, repetitive, alternating, and continuous movements (limbs and body axis)
Oligokinesia	Reduction/lack of spontaneous/ automatic movements	Spontaneous blinking, spontaneous facial expressions, pendular movements of the upper limbs during walking

Note: The definitions of bradykinesia and related features (hypokinesia, sequence effect, and hesitations/halts) are summarized.

TABLE 2

Proposed classification of bradykinesia and related motor abnormalities

Axis I. Phenomenology

- | | |
|--------------------------------|--|
| • Type of movements | Voluntary (single and repetitive or continuous) vs. spontaneous and semi-automatic movement (eg, face expressions, walking) |
| • Major movement features | Bradykinesia (movement slowness), hypokinesia (reduced movement amplitude), sequence effect, hesitations/ halts (only repetitive movements), akinesia, oligokinesia (only spontaneous movements) |
| • Body distribution | Limbs and/or body axis (face, voice, trunk) |
| • Associated clinical features | Isolated or combined with other neurological symptoms |

Axis II. Etiology

- | | |
|-------------------------------|---|
| • Parkinsonism | PD, PSP, MSA, CBS, DLB, and others |
| • Non-parkinsonian conditions | Hyperkinetic movement disorders and other neurological conditions |
| • Unknown etiology | |
-

Abbreviations: PD: Parkinson's disease; PSP: progressive supranuclear palsy; MSA: multiple system atrophy; CBS: corticobasal syndrome; DLB: dementia Lewy body.