

Psychomotor disadaptation syndrome

A new clinical entity in geriatric patients

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Psychomotor Disadaptation Syndrome (PDS) is characterized by backward disequilibrium in a sitting and standing position, reactional hypertonia, an alteration of postural reactions, gait modifications, and fear of falling. PDS may be the result of a loss of postural reserves, reaching a frailty threshold that does not allow subjects to maintain an adequate functional level. This clinical picture may be associated with frontal-subcortical dysfunction in which microangiopathy could play a key role. PDS may be triggered by numerous factors, such as cardiovascular and metabolic diseases, falls, and bed rest. In addition to medical and neurological evaluations, a specific rehabilitation program is one of the most important aspects of the management of patients showing PDS.

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Over the past two decades, we have observed a clinical picture in which older patients present with severe mobility impairment. The original clinical signs seen in this picture did not correspond with any previously known neurologic disease. In this article, we describe this entity as a geriatric syndrome that corresponds with the observable decompensation of motor functions, particularly during

postural activities, such as the transfer from sitting to standing, and the standing position.

In patients with this syndrome who do not show any neurologic disease, the motor impairments are related to loss of postural abilities. Postural function is largely affected by aging itself.^{1,2} In addition to physiologic aging, the cumulated effects of diseases and a sedentary lifestyle may reduce the func-

tional reserves of motor and postural abilities, reinforcing the notion of “use it or lose it.” This loss (or low level) of functional reserves can explain the physiologic instability in motor functions that is associated with a permanent risk of decompensation. We have defined this acute or progressive form of functional insufficiency as the Psychomotor Disadaptation Syndrome (PDS). PDS is characterized by backward disequilibrium, gait abnormalities, and fear of falling.

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This syndrome is characterized by postural impairments and neurologic signs that are frequently associated with a psychological feature.^{3,4,5} In severe cases, the functional impact is observed in postural and walking abilities as well as during transfers. In a previous study, there was a correlation between PDS and impaired activity in everyday life; 92% of the PDS patients showed high levels of dependency (table 1).⁵

Postural impairments. Backward disequilibrium is the main postural feature in sitting and standing positions and is always present. Backward disequilibrium is defined by the backward position of the trunk while sitting, and a tendency to fall backward in the standing position. This feature has been described in standing position in the severe form of Parkinson’s disease, but is not observed early on. Backward disequilibrium is the result of a backward

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projection of the center-of-mass to the posterior boundary of the base of support (figure 1). In severe cases, we sometimes observe failure to reach an erect position because the center-of-mass is projected outside the base of support. When the standing position is successful, forward trunk bending, knee flexion, and toe clenching can be observed as adaptive patterns that allow the patient to maintain the center-of-mass in the base of support.

In the sitting position, the buttocks are commonly located on the anterior side of the seat while the trunk rests on the back of the armchair in patients without severe kyphosis. In severe cases, the transfer from sitting to standing is impossible without assistance because the subject cannot bend the trunk forward.

Neurologic signs. Two abnormalities have been reported as characteristics of this syndrome.

First, patients with PDS have an increase in muscle tone that counteracts movement execution. This hypertonia appears during the movements achieved by the patient and during clinical passive exam. This hypertonia is called *reactional* because it appears when a limb is mobilized passively by an investigator, and it decreases when confidence and relaxation are obtained. This reactional hypertonia is variable and heterogeneous along the range of the passive movement; this is distinguished from the cogwheel rigidity observed in Parkinson's disease, which is homogeneous and continuous along the entire movement range. The clinical examination of PDS subjects has shown that reactional hypertonia is present in up to 95% of cases.⁵ Unlike Parkinson's disease, hypertonia of PDS does not affect the face muscles so PDS patients do not have the typical PD blank stare. Additionally, hypertonia of PDS does not respond to antiparkinson medications.

Second, patients with PDS have an alteration in postural reactions that is revealed during an examination with the patient standing. This clinical in-



Figure 1. Psychomotor disadaptation syndrome is characterized by postural impairments including backward disequilibrium as pictured here. Backward disequilibrium is the result of a backward projection of the center-of-mass to the posterior boundary of the base of support.

Photograph courtesy of the authors.

vestigation of postural reactions should include both reactive postural responses and protective reactions. Reactive postural responses are tested through a slight push applied to the trunk in order to assess the quality of

muscular responses, particularly in the hip and ankle. The normal response in older subjects has been described as a hip strategy that consists in trunk mobilization in the sagittal plane to control the disequilibrium⁶. If the postural

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strategy has disappeared or is not very efficient, the subject needs to use a stepping reaction. In all cases, this protective reaction is tested both forward and backward using a stronger push, which tends to make center-of-mass projections leave the base of support.

The protective reactions of the arms are assessed in front of a wall to trigger forward displacement of the arms. In severe forms of PDS, the patient falls forward with no protective reactions of the arms; typically, Parkinson's patients retain this protective arm reaction. The lack of such protection may explain the face injuries observed frequently in hospitalized older adults with PDS; Parkinson's patients rarely incur these kinds of injuries. In our

study, none of the patients with PDS showed normal reactive postural responses, while normal stepping forward reactions were found in less than one-quarter of the patients, and normal stepping backward reactions in only 11% of cases (table 1).⁵

Gait disorders. The gait modifications that characterize PDS include: hesitancy in initiation, small steps with a shuffle-like gait pattern ("marche à petits pas"), increase in the support base and in the duration of double support, slowing of walking speed, trend toward retropulsion, knee flexion. These signs may be observed in many neurologic diseases⁷, but features of the support base can help to distinguish PDS from Parkinson's disease: In PDS, the sup-

port base is increased, whereas in Parkinson's disease, it is reduced.

These gait abnormalities must not be considered as specific components of PDS, but rather as a consequence of

The functional impact of PDS makes rehabilitation an essential component of patient management

a poor level of performance in dynamic equilibrium.

Psychological feature. Fear of falling, which is strongly linked to low self-confidence, is usually associated with PDS.

Associated findings. Certain signs, such as protein-calorie malnutrition and cognitive impairment,⁵ were found to be associated with PDS (table 1). Moreover, the measure of aerobic capacity from the "Get Up and Go" test showed an increase in energy expenditure with a much higher energy cost in PDS compared with other age-matched control subjects.⁸

Pathophysiology

The vicious spiral that affects postural and motor function in PDS can be considered a model of the process of abnormal aging. Postural function is affected by the aging process and may be worsened by chronic diseases.² Physiologic systems that are critical for postural control systems are altered at every level: sensory inputs, neural pathways for motor control, musculoskeletal integrity, and central processing. Loss of reserves in these different systems occurs gradually, and compensations among the different levels allow the individual to maintain the efficiency of balance until a threshold of decompensation occurs.

We postulate that PDS can occur

Table 1 Neurological signs, functional state and associated manifestations of PDS in a population of 73 patients

Functional Katz index*	
A, B, and C	8%
D, E, F, and G	92%
MMSE **	18.8 ± 5.5
BMI (kg/m²)**	21.9 ± 3.0
Reactional hypertonia	96%
Akinesia	71%
Reactive postural responses†	
0	82%
1	8%
2	0)
Protective reactions††	
Normal stepping forward reactions	23%
Normal stepping backward reactions	11%

* Patients in grades A,B, and C can be described as being relatively independent, whereas the other patients needed help from others.

** mean ± SD.

† Quality of reactive postural responses was assessed as follows:

- 0) lack of reaction
- 1) weak responses, which were inadequate to maintain equilibrium when a slight push was applied to the trunk
- 2) normal strategy.

†† Protective reactions were tested using a stronger push, which tended to make center of mass projections leave the base of support. They were considered as normal when the patient was able to step quickly enough to avoid the fall.

Source: Created for Geriatrics by F. Mourey, PhD, P. Manckoundia, MD, I. Martin-Arveux, MD, B. Tavernier-Vidal, MD, and P. Pflitzenmeyer, MD based on data from reference 5.

through one of two mechanisms:³ 1) a progressive reduction in functional reserves reaches a threshold at which point the patient is unable to maintain adequate functional compensation; 2) the cumulative effects of both progressive loss of postural reserves and an acute stress factor; the combination of the two components results in a breakdown of the entire compensatory process.

Numerous CNS diseases can be associated with PDS (table 2). Several authors have suggested that lesions of the periventricular fibres connecting the cerebellum and motor cortex, impaired central somatosensory integration, and the disruption of long-loop reflexes are associated with postural and gait impairment.^{9,10} Subcortical structures seem to be predominantly involved in PDS, possibly through chronic white matter ischemia or acute cerebral infarcts.¹¹

It remains unknown why postural and gait abnormalities are dominant while other dysfunctions, such as disinhibition or mood lability and apathy, usually reported in fronto-subcortical dysfunction, are less frequent. Perhaps, in PDS, alteration of the basal ganglia (especially related to motor slowing) may be more prominent than that of other anatomic regions implicated in frontal-subcortical dysfunction.

In patients showing reduced reserves in fronto-subcortical function, many acute factors may trigger PDS. In all cases, the clinician must look for any factor that may induce lesions in the subcortical area, particularly pathologic processes that may be associated with a decrease in cerebral blood flow. Among these numerous causes, acute cardiac failure, recent arrhythmia, and fall in blood pressure must be investigated. Besides these dominant pathologic features, any factor that may alter neuronal metabolic state (eg, dehydration, inflammatory states, anemia, hypoxemia, hypoglycemia) must be ruled out or promptly treated (table 3). Moreover, psychotropic medica-

Table 2 Chronic diseases associated with PDS

Degenerative diseases
Parkinson's disease and Parkinsonism Multiple system atrophy Progressive supranuclear palsy Lewy body disease
Vascular lesions
Binswanger's disease Subcortical vascular lesions
Normal pressure hydrocephalus
Intracranial neoplasm
Particularly lymphomas of nervous system
Infectious diseases
Meningoencephalitis HIV infection
Chronic depression
Source: Created for Geriatrics by F. Mourey, PhD, P. Manckoundia, MD, I. Martin-Arveux, MD, B. Tavernier-Vidal, MD, and P. Pfitzenmeyer, MD.

tions may increase the risk of PDS.

In addition to organic causes, falls by themselves may precipitate full-blown PDS in individuals previously displaying compensated postural dyscontrol. In these cases, pure motor forms without behavioral disorders are more often observed; some aspect of this pure motor form may resemble the post-fall syndrome.¹²

Finally, we can suggest that inactivity has a deleterious effect in general and may exacerbate the occurrence of PDS.⁴ In support of this, we have noted that PDS often is triggered by immobility and bed rest in very old people. In these patients, besides the known effects of inactivity on muscle weakness and cardiovascular response to exercise, we hypothesize that immobilization contributes to a loss of postural strategies and motor automatism.

For all the reasons we have developed above, we think that PDS should not be considered as a disease, but as a

Table 3 Acute factors associated with PDS

Cardiovascular conditions
Hypotension Cardiac arrhythmia Heart failure
Metabolic diseases
Dehydration and electrolyte imbalance Anemia Hypoxia Hypoglycemia Hyperthermia
Drugs
Antipsychotic drugs Benzodiazepines Analgesics (opiates)
Falls
Bed rest
Source: Created for Geriatrics by F. Mourey, PhD, P. Manckoundia, MD, I. Martin-Arveux, MD, B. Tavernier-Vidal, MD, and P. Pfitzenmeyer, MD.

geriatric syndrome, which may be associated with numerous degenerative or neurovascular diseases.

Management of PDS

The usual medical procedure, which tries to distinguish a specific disease through differential diagnosis, may not be useful in the clinical approach to PDS. Because it is not a diagnosis but a constellation of symptoms (or syndrome), the recognition of PDS must be followed by consideration of all of the conditions or diseases that may provoke this functional disadaptation. For example, in our clinical practice, we have observed PDS in patients showing severe Parkinson's disease. In these cases, backward disequilibrium and an alteration of postural reactions can occur abruptly following different acute conditions such as falls, bed rest associated with acute illness or surgical procedures, or the worsening of cardio-

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vascular diseases. In the severe state of Parkinson's disease, physicians must reconsider not only the treatment of Parkinson's disease, but also the medical treatment of all conditions that have triggered PDS.

The functional impact of PDS makes rehabilitation an essential component of patient management. Because older patients are at increased risk of functional impairment arising from acute illness and surgery, a program of early rehabilitation should be initiated to prevent or reverse this decline as well as prevent PDS. Once PDS has developed, the main goal of rehabilitation is to correct postural insufficiency. Balance training should integrate specific exercises to regulate anomalies of the projection of the center-of-mass and correct backward disequilibrium. Automatic movements, such as rolling from side to side, sitting up, and transferring, must be relearned through repetitive exercises.

To improve the benefits of these exercises, physicians, nursing staff, and physical and occupational therapists must coordinate their interventions through a multidisciplinary team approach. Once postural instability has improved, gait exercises and learning to rise from the floor after falling should be introduced. Concomitant strengthening exercises facilitate the postural retraining and motor-programming process. In a previous study, we demonstrated that a specific rehabilitation program for in-bed mobility and lying-to-standing transfers can prevent severe functional disability in patients with PDS who have a fear of falling.⁴

Conclusion

We believe that Psychomotor Disadaptation Syndrome should be added to the list of geriatric syndromes. This entity, which is characterized by postural and motor disabilities and associated with many neurologic disorders, may be triggered by numerous factors, such as cardiovascular and metabolic diseases, falls, and bed rest. Early medical management of underlying disor-

ders and timely, syndrome-specific rehabilitation are determining factors for prognosis. ■

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