The Eyes and Proprioception
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Editor’s Note: This article, principally authored by O. Alves Da Silva, MD, is an invited paper. When I first became aware of Dr. Da Silva’s work with prisms and proprioception, I encouraged him to share his experiences as a pioneering ophthalmologist in Lisbon, Portugal. Many of our readers will be aware of pioneering optometric involvement in this field. It was not Dr. Da Silva’s charge to review this, as has been amply done elsewhere (see here for example: http://bit.ly/2Z6bYv0). For additional background, see the editorial in this issue.

ABSTRACT
The proprioceptive system has an extensive influence on the maintenance of human health. When the proprioceptive system is dysfunctional, the central nervous system does not recognize the correct status of tonicity of the muscles at rest or in movement, does not integrate correctly the information that comes from sensory receptors, and has difficulty in modulating multisensorial integration, with consequences in motor behavior and cognitive functions.

This results in a wide range of proprioceptive abnormalities which are clinically related, are treated together, and are termed as Postural Deficiency Syndrome (PDS) or more recently Proprioceptive Dysfunction Syndrome.

The author has personally observed more than 40,000 patients suffering from this condition during the last 40 years and devised an active prism therapeutic protocol that is based on the knowledge that small modifications of the muscular tonus of the oculomotor muscles can change the tonus of the axial paravertebral muscles.

This paper describes PDS diagnosis and provides explanation of the updated active prism protocol aimed toward vision professionals.

INTRODUCTION
This paper will introduce you to a relatively unknown clinical entity that affects millions of people and may have an ophthalmologic basis for its treatment. This condition is historically called Postural Deficiency Syndrome (PDS) or in a more recent nomenclature Proprioceptive Dysfunction Syndrome (PDS). The understanding of its pathophysiology and rationale for treatment entails a broad knowledge of neuroscience and neuro-ophthalmology. It also requires reconsideration of how oculomotor muscles can interfere with body postural tonus and cognitive brain functions. Our emphasis is on newer concepts about visual function and motor actions, and how they can affect multi-sensory brain integration. We share how the evolution in
our thinking and approach has lead toward a paradigm shift in the application of prism and complementary interventions for PDS.

**What is proprioception?**

Proprioception is the sense of self, the body's conscious awareness of movement and position. It's the sense necessary for brain integration of the corporal limits, and necessary for conscious and subconscious knowledge of the relation between each segment of the body, and between the body and environmental space around it.

The idea that humans are limited to five senses comes from the Greek philosopher Aristotle. This is a 2500 year-old concept that is scientifically outdated. Proprioception has been revealed as the sixth sense for more than 100 years.

Taking this a step further, the French scientist J.P. Roll refers to proprioception as our first sense, “the sense that makes sense of our senses”, signaling its importance. It is believed to be present in all animal species and is necessary to keep them alive. Even during the earliest stages of human evolution, normal proprioception was an essential sense. Faulty proprioception can lead to a loss of precision in movement or misperception of environmental dangers, with severe consequences for survival mechanisms. While accurate proprioception is not a condition of survival in modern times in most regions of the world, its deficits contribute to a range of motor and cognitive dysfunctions in our society that have serious consequences in quality of life, as will be illustrated in this paper.

**What is the Proprioceptive System?**

As with our other sensory modalities, proprioception should be conceived as a system rather than as an isolated sense. The origin of this concept can be traced to Sherrington, who described it extensively in his treatise on physiology in 1906.

The proprioceptive system has its neurologic afferents in the muscle spindles, extrafusal axon terminations and mechanoreceptors present in muscles, joint and organ capsules, ligaments and enthesis (the connective tissue between tendon or ligament and bone). The proprioceptive information can be transmitted to the central nervous system by conscious and subconscious pathways.

The sensory information that becomes conscious is transmitted by the ascending sensory tracts of Goll and Burdach. These pathways convey fibers for position sense and fine cutaneous sensation (touch, vibration, fine pressure and two point discrimination). They are crossed pathways that travel from their receptors through the spinal cord dorsal root, medulla nuclei, midbrain and thalamus until they reach the primary somatosensory area of cerebral cortex.

The subconscious sensory information is conveyed by ascending tracts, crossed and uncrossed, that go through the dorsal spinal cord and medulla oblongata, terminating at the cerebellum. The most well known of these ascending tracts are the anterior and posterior spinocerebellar tracts and the cuneocerebellar tract that convey information from the lower and upper body.

But we must not forget the other two important subconscious tracts most relevant for our purposes, the spinotectal tract and the spinoreticular tract. The spinotectal tract provides information for spinovisual reflexes and brings about movements of the eyes and head toward the source of stimulation. The spinoreticular tract provides cutaneous and muscular afferent information for the reticular formation.

The reticular formation in the brainstem is a heterogeneous structure with no clear boundaries between its nuclei, but is highly organized. It is home to numerous nuclei and ascending and descending tracts, with projections between them that are involved in modulating sensory and motor functions. The reticular formation is also home to groups of cells that produce neurotransmitters that
are sent to different areas throughout the central nervous system to modulate sensory perception, motor activity and behavioral responses. Reticular formation neurons also establish circuits within the motor nuclei of the cranial nerves, modulating the motor activity of the gastrointestinal system, respiratory system and cardiovascular system. The same neuronal population also contributes to orofacial motor responses by coordinating the activity of the trigeminal, facial and hypoglossal nuclei. Also found in the reticular formation are the descending pathways that help to maintain posture and muscle tone through the descending reticulospinal tract.

This highly complex neuronal organization is evidence that all senses are related to each other in the central nervous system, not only by simple reflex arcs but in a much more elaborate way that interfaces with cerebellar and cortical pathways. This is the basis of the neurologic concept of multisensorial integration, a CNS process by which information from different sensory systems is combined to influence perception, decisions, motor actions and behavior. It is known that spatiotemporal concordant cross-modal stimuli, for example sounds and images, evoke enhanced responses. On the other hand, spatially disparate stimuli can suppress some responses. Multisensory integration has been shown to enhance and speed the detection, localization, and reaction to biologically significant events. Neuropsychological studies also demonstrate its importance in signal disambiguation in hearing, speech and cognitive functions such as learning.18

The proprioceptive system, from its peripheral sensory components to the central nervous system pathways, appears to be an important modulator of sensorimotor activity. This concept provides a new basis for understanding how sensory information from peripheral and spinal inputs can be integrated with information from the brain and cerebellum, both higher cognitive functions and subconscious activities, and is responsible for modulating activity of pre-motor networks and ensure coordinated motor output.

Proprioception therefore extends far beyond muscular tonus, equilibrium, joint movement and postural control, as is usually presented in health sciences education. We can summarize by stating that the Proprioceptive system has three overlapping major functions:

1) **Regulation of muscular tonus.** Posture and body movement is mediated by proprioception through the afferent information it receives from all sensory receptors, and by the efferent information it sends to the extrapyramidal motor tracts.

2) **Egocentric spatial localization.** By integrating and modulating the information that comes from sensory receptors, the proprioceptive system informs the brain about the relative position of the sensory organs, the relation between each body segment, and the relative position of the body in the surrounding environment.

3) **Modulation of multisensory information.** Proprioceptive information known to be transmitted in the multisensorial deep layers of the superior colliculus in the midbrain is believed to have a role in modulating multisensory integration. This modulation has consequences in motor behavior and higher cognitive functions.

**What is Postural Deficiency Syndrome?**

Postural Deficiency Syndrome (PDS) was described by the physiatrist H. Martins da Cunha, M.D. in 1979 as a clinical entity encompassing multiple symptoms and signs related to proprioceptive dysfunction.18 The Postural Deficiency Syndrome (PDS) was originally described as “a clinical entity of functional nature due essentially to an alteration of the ideal biomechanics and to a deficiency of the proprioceptive and visual information”.20,22 Martins da Cunha stated that the patients affected by this condition had in common a...
stereotyped body posture with an asymmetrical paravertebral and thoracic hypertonia associated with an asymmetrical lower limb position and a permanent gaze deviation.

Over the last 40 years the understanding of this condition, of its clinical manifestations, and its underlying neurological pathways has evolved. This condition is presently understood as a proprioceptive dysfunction with three main characteristics:

1. An impairment of the ocular, stomatognathic and postural tonic equilibrium.
2. An impairment of egocentric spatial localization.
3. A perceptual impairment disturbing the multisensorial brain integration.

Therefore, a more comprehensive understanding of this clinical condition leads to the current nomenclature, designating this condition as Proprioception Dysfunction syndrome (PDS), maintaining its acronym.

Since the proprioceptive system is transverse to all of the other body systems, the clinical manifestations of its disturbance cannot be localized to just one area of the body.

In addition, patients do not typically present with all of the possible symptoms. For example, patient A may present with upper body muscular pain as the predominant symptom. Patient B may present with the predominant symptom as lack of balance, and patient C may present cognitive impairment as the predominant symptom. However, patients A, B and C are diagnosed using the same proprioception dysfunction diagnostic protocol and they respond to the same proprioception dysfunction treatment protocol. Before discussing the most common signs and symptoms of PDS as related to each of the three main PDS characteristics, some background is in order.

How Ophthalmologic Disturbances were Crucial to the Understanding of PDS

The first author’s (OAS) involvement with this medical condition began two years prior to its publication, when Martins da Cunha asked him to study the ophthalmological aspects of his PDS patients who did not seem to fit the pattern of what was expected to be found. Martins da Cunha was seeking the answer for three ophthalmological questions:

- The first question to be answered was why these patients had difficulty in correctly localizing the arrow related to the figures in the Maddox Wing Test.
- The second question was why postural reprogramming exercises normalized this visual test.
- The third question was why PDS patients presented with their primary position of gaze altered. This alteration consisted of the following: healthy people, when fixating at distance, present both eye axes parallel, and these are parallel to the sagittal plane. In PDS patients both eye axes are still parallel, but are no longer parallel to the sagittal plane of the body, because of a persistent head and trunk rotation.

The regular devices and methods used in ophthalmology at the time were unsatisfactory in answering these three questions. The first author therefore devised a new method that he called directional scotometry to search for a scientific answer. A Clement Clark synoptophore was equipped with a pair of simultaneous perception slides (G3 and G4). The slides were shown to the patient in primary position, in levoversion and in dextroversion to compare their projection. (Figure 1)

Our study showed that in people having asymmetrical body posture favoring left limb support, a pseudoscotoma occurred in which part of the image was missing, only when making a version movement toward the opposite side on dextroversion. In contrast, those with postural asymmetry favoring right limb support exhibited a pseudoscotoma only on levoversion.

Further, we verified that there was a consistent relationship between the results of
directional scotometry and the limb support
the patient presented. For example, when
we verified a pseudoscotoma at 20° degrees
in levoversion but no pseudoscotoma in
dextroversion at 20° and 30° degrees, this
was always related to a strong right lower
limb support. Conversely, when there was a
pseudoscotoma at 20° degrees in dextroversion
and no pseudoscotoma in 20° or 30° degrees of
levovation, the patient was always supported
on his left lower limb.

Therefore, we concluded that in studying
the patient’s oculomotricity by this method
it was possible to identify a pattern of dis­
ease in PDS patients, and establish a clinical
classification system with diagnostic and
therapeutic value. The classification system
developed includes six different types of PDS
based on predominant limb support, postural
changes in head extension and rotation, and
synoptophore results.

This represented major progress because
until then, just two types (right and left type)
could be identified by Martins da Cunha,
based only on clinical postural observation.
Ultimately this finding would pave the way
for developing our ophthalmologic treatment
protocol. (Table 1)

**Diagnosis and Classification of PDS**

Analyzing the classification system (Table
1) we must consider the strong influence of
clinical observation on how the diagnostic
classifications evolved. We call PDS “mixed”
when the patient stands with divergent feet, and
the side of preferential stance may shift from one
moment to the other. On the “mixed” type you
will have pseudoscotomas in both levovation
and dextroversion. “Pure” PDS occurs when
there is a clear lateral preference in plantar
stance and you can identify the predominant
support. In pure cases, the pseudoscotomas
occur on only one side of version, on either
levovation or dextroversion.

Within the “mixed” PDS type you can identify
two different patterns. The “mixed pure” will
present with what we call a concordant postural
head change, limitation of rotation and head
tilt to the same side. The “mixed pure” PDS
patients have pseudoscotomas for symmetrical
angles of version.

In the “mixed predominant” PDS type, the
head postural changes are non-concordant
and the pseudoscotomas are also bilateral, but
occur for asymmetrical angles of version.

For each type, the terms “right” or “left” are
added as subtypes according to the laterality
of the findings.

Historically this was how the classification
was built, from postural observation to
oculomotor findings, but presently we know
that the synoptophore analysis is the most
accurate way to identify each type of PDS and
prescribe the appropriate treatment.

Nevertheless, clinical examination is abso­
lutely necessary, and one must master the
following clinical tests in order to establish
the diagnosis, and to document the postural
changes and spatial localization disorders
characteristic of this syndrome.
<table>
<thead>
<tr>
<th>Plantar stance</th>
<th>Head extension</th>
<th>Head Rotation</th>
<th>Synoptophore</th>
<th>Prismatic lenses</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mixed</strong></td>
<td></td>
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<tr>
<td>Divergent feet, side of preferential stance may shift</td>
<td>Head exam is concordant: Limitation of rotation and head tilt in extension on the same side “farther and shorter” to the same side</td>
<td>Pseudoscotomas for symmetrical angles of version</td>
<td>2 Prisms of asymmetrical power RE 125° LE 55° Higher powered prism on the side of head limitation</td>
<td></td>
</tr>
<tr>
<td><strong>Mixed</strong></td>
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<tr>
<td>Divergent feet, plantar stance may shift but with preference for <strong>right side</strong></td>
<td>Limitation of rotation and head tilt in extension to the right</td>
<td>20°/20° or 30°/30°</td>
<td>RE- 3Δ 125° LE- 2Δ 55°</td>
<td></td>
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<tr>
<td><strong>Mixed</strong></td>
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<tr>
<td>Divergent feet, plantar stance may shift but with preference for <strong>left side</strong></td>
<td>Limitation of rotation and head tilt in extension to the left</td>
<td>20°/20° or 30°/30°</td>
<td>RE- 2Δ 125° LE- 3Δ 55°</td>
<td></td>
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<tr>
<td><strong>Mixed</strong></td>
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<tr>
<td>Divergent feet, side of preferential stance more clearly defined</td>
<td>Head exam is not concordant: Limitation of rotation for one side and head tilt in extension to the other side</td>
<td>Pseudoscotomas are asymmetrical occur in higher angle of version to the side of preferential plantar stance</td>
<td>1 Prism RE 125° or LE 55° Prism on the side of higher angle of version and preferential plantar stance</td>
<td></td>
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<tr>
<td><strong>Mixed</strong></td>
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<tr>
<td>Divergent feet, plantar stance with preference for <strong>right side</strong></td>
<td>Head exam is not concordant</td>
<td>20 ° dextroversion 30 ° levoversion</td>
<td>RE 125° 2Δ or 3Δ</td>
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<tr>
<td><strong>Mixed</strong></td>
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<tr>
<td>Divergent feet, plantar stance with preference for <strong>left side</strong></td>
<td>Head exam is not concordant: Limitation of rotation for one side and head tilt in extension to the other side</td>
<td>20 ° dextroversion 30 ° levoversion</td>
<td>LE 55° 2Δ or 3Δ</td>
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<tr>
<td><strong>Pure</strong></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Clear lateral preference in plantar stance, stance foot in sagittal position</td>
<td>Head exam is not concordant: Limitation of rotation for one side and head tilt in extension to the other side</td>
<td>Pseudoscotoma only to one side of version the opposite side of plantar stance</td>
<td>1 horizontal prism RE 180° LE 0° Prism on the side of plantar stance, On the opposite side of pseudoscotoma</td>
<td></td>
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<tr>
<td><strong>Pure</strong></td>
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<tr>
<td>Right plantar stance <strong>Right foot</strong> is sagittal</td>
<td>Head exam is not concordant</td>
<td>20 ° levoversion</td>
<td>RE 180° 2Δ or 3Δ</td>
<td></td>
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<tr>
<td><strong>Pure</strong></td>
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<tr>
<td>Left plantar stance <strong>Left foot</strong> is sagittal</td>
<td>Head exam is not concordant</td>
<td>20 ° dextroversion</td>
<td>LE 0° 2Δ or 3Δ</td>
<td></td>
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</table>
A. Clinical Tests Necessary for Classification

**The Head Extension Test**

The patient is asked to extend the head backwards, and the distance from shoulder to ear on both sides are measured. In the presence of proprioceptive dysfunction, both distances are asymmetrical, in spite of the patient having the sensation that they are symmetrical.

**The Head Rotation Test**

The patient is asked to rotate the head to the right side and to the left side as far as he can. In the presence of proprioceptive dysfunction the rotation is asymmetrical and limited.

B. Clinical Tests Elucidating Altered Muscular Tonus

H. Martins da Cunha originally proposed the use of trigger points, which are highly painful under finger pressure. When the patient wears active prisms to correct postural asymmetry, the pain is eliminated. That was, however, a more aggressive test that the first author was not comfortable using. He therefore proposed the following standard tests:

**The Mouth Opening Test**

The patient is asked to open the mouth as much as he can and this opening is measured. In the case of proprioceptive dysfunction there can be limitation in mouth opening, and in some cases very severe. The proprioceptive response to prism can be dramatic in improving this.

**The Tonic Convergence Test**

The examiner moves the tip of a ballpoint pen or small size object on the midline toward the eyes. The distance at which asthenopia is experienced, and the distance at which fusion is disrupted are measured.

In the case of proprioceptive dysfunction both distances are remote, with asthenopia occurring before 6 inches and loss of convergence occurring before 4 inches.

C. Clinical tests elucidating dysperception of spatial localization

**The Eye-Hand Localization Test (Figure 2)**

The eye-hand test, developed in our clinical practice, is described as follows: a pen mark is drawn on the cutaneous ridge of the first

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**Figure 2:** Example of eye-hand test, illustrating error and accuracy.
interdigital space of the dorsal aspect of the hand. An object, the pen, is presented to the patient by the observer within a comfortable grasping distance, on the contralateral side of the hand to be tested. The patient is asked to hit the pen with the hand mark while regarding it, as quickly and as accurately as possible, and to avoid final correction of the trajectories. The observer then verifies if the patient hit the target correctly or if the target was hit by any other finger region distant from the drawn mark.

This test is believed to evaluate sensorimotor transformation involving eye, head, hand, and postural systems. It is a functional test that probably evaluates the well described function of the superior colliculus in reaching movements and its role in orienting gaze and arms toward a target. It differs from other common pointing tests that evaluate cerebellar function, visual acuity or fine hand motricity related to another neurologic pathways.

The test has been validated using kinematic analysis of movement. It was confirmed to be altered in every type of PDS. The hand accuracy consistently improved in all cases after treatment with active prisms and there was an established relation between the improvement of eye-hand accuracy and the normalization of head tilt as well as the gain in head rotation. The eye-hand test can therefore be used as a valuable diagnostic tool as well as a treatment assessment instrument.

Other spatial localization tests can be used to show the patient and his family that there is something wrong concerning his proprioception. The following are some examples:

**Mirror test:** H. Martins da Cunha used to ask the patients to place themselves in a straight standing position in front of a mirror with their eyes closed. He then asked them to open their eyes and see the difference between their body perception and reality.

**Feet position test:** The patient is asked to close his eyes and copy the position of his feet with his hands. When opening his eyes he will usually see that his hands are pointing in different directions that his feet are.
**Tactile test:** The clinician touches a point over the patient's thorax and asks him to immediately touch himself at the same skin point. This is done quickly, pinpointing a random sequence of different skin points. Family members can observe the error in the patient’s match, sometimes as large as several centimeters of discrepancy compared to where the clinician has touched.

**Symptoms of Ophthalmic Disturbances in PDS**

As previously stated, the proprioceptive system is transversal to all sensorimotor functions of the organism and in that way an impairment in proprioception can translate to several different symptoms. For better comprehension we can organize the symptoms according to each main PDS characteristic they reveal, as shown in Table 2.

We particularly call attention to the symptoms related to pain that are frequently musculoskeletal but can also be neuropathic (for example sciatica or migraine) or organ related (for example epigastralgia), as pain in its many presentations is one of the most prevalent complaints of PDS patients.

Interestingly, the pathophysiology underlying some symptoms of proprioceptive dysfunction resulting in numbness and paresthesias is believed to stem from disturbances in vasomotricity and asymmetrical blood flow supply. H. Martins da Cunha was a pioneer in showing that asymmetrical arterial blood flow supply to the facial region could be normalized with proprioceptive treatment as documented through infrared thermography of the face.

Other significative symptoms are those related to cognitive dysfunction that can impair the learning process, irrespective of intelligence levels and good visual acuity. These symptoms are best explained by the disturbance of multisensorial brain integration that occurs as result of a faulty proprioception. These symptoms include attention deficit, hyperactivity and poor concentration. Dyslexia, dysgraphia and dysorthographia also present as symptoms with great impact in PDS patients that can be improved through the treatment protocol.

We focus next on the signs of ophthalmological disturbances found in PDS patients.

**Signs of Ophthalmic Disturbances in PDS**

The ophthalmic signs of PDS are diverse and complex, as are the neuronal pathways they rely on. The following is a survey of ophthalmic signs and symptoms associated with PDS.

**Visual scotomas** are evidenced through computerized campimetry (Visual Field Test), with areas of relative and absolute scotomas located para-centrally and in the periphery. The distribution or pattern of these scotomas does not correspond to any specific neurological condition and is sometimes incorrectly interpreted as normal tension glaucoma. The scotomas represent a deficit in visual perception consistent with processing delays that occur in the auditory system of patients with PDS.

**Monocular diplopia** can occur when one image of the target is linked to the fovea, but another view is linked to an anomalous or eccentric fixation point.

**Pseudoscotomas** appear when an error of proprioceptive information related to the position of the ocular globe leads to a discrepancy between objective and subjective gaze relative to the target location.

**Binocular Diplopia** is typically due to reduced capacity of sensory fusion, and often represents a central fusion disruption as can occur in acquired brain injury.

**Convergence insufficiency** is the inability to sustain fusion at near. This is not an eye muscle problem in PDS patients, as both eyes have normal adduction ability of the medial recti. When we test each eye in isolation, asking the patient to close one eye and follow an approaching point with the other eye, there is total adduction with the near point of fixation to the nose. However, when the same PDS patient is asked to follow an approaching
point with both eyes open, the break point occurs well beyond the normal near point of convergence. This indicates a disturbance in binocular perception rather than weakness of the medial recti. Disruption in fusion occurs because of the mismatch between image localization of each eye, related to a faulty perception of the muscular tonus of one eye relative to the other eye.

**Near exotropia** with exodeviation and associated diplopia at near, but with no deviation and with single vision at distance.

**Oculomotor dyscoordination** with the patient presenting inability to coordinate the eyes in most gaze positions. Symptoms of diplopia and incomitant ocular deviation often occur. This may be associated with nystagmoid movements that, although rare, have been recorded on video.

**Ophthalmic migraine** during which micropsia or a relative scotoma can occur as with an aura. These altered visual perceptions are frequently followed by headache.

**Sensation of ocular globe retraction**, during which there is a sensation of feeling that the eyes are being pulled backward on inward.

**Error of spatial localization of objects**, with figures and visual points of reference resulting in straight lines being perceived as curved. Examples are a picture hanging straight that is perceived as crooked; a vertical wall being perceived as leaning; and while reading, lines in a text perceived as jumping. The Rey complex figure test shows that in some PDS patients faulty visual perception also occurs in two dimensional transformations involving copying and memory reproduction.\(^7,33\)

**Errors of egocentric localization.** We call egocentric localization the ability in locating one’s body in space. In essence, errors of spatial and egocentric localization in PDS patients are evidence of a mismatch between what the subject feels as his body spatial position and the real localization of his body in space. There is perceptual impairment of the real relation between each segment of the body, and between the body space and environment space around it. This can be exhibited in difficulties with left and right orientation. It can be manifest in frequent falls or ankle sprains due to difficulties in locating one’s body in relation to the ground. It can be exhibited with difficulty in motor planning and execution, such as precisely and automatically estimating the reaching distance to grab an object. This can lead to clumsiness, bumping into the furniture, poor sports performance, accidents while handling instruments, or while driving.

These errors vary with the direction of gaze and include the inaccurate perception of the distance between one’s own body and surrounding objects, which appears to be less than what it really is.

The neuro-ophthalmologic explanation for perceptual impairment due to proprioceptive dysfunction is as follows. How does the brain know where surrounding objects are in relation to the observer, and where the eyes are in relation to the rest of the body? It is not the retina that informs the brain about the exact location of the objects that surround the observer, in relation to the observer. The retina is a receptive field that provides information for topographic organization of the visual fields in the visual cortex. Each point of the image seen activates the same point within the cortical visual map irrespective of eye, head or trunk position. The retina has allocentric localization ability but does not provide egocentric localization information. The exact location of an object in relation to an observer is given by the perception of the position of the eyes in relation to the rest of the observer’s body, and by the perception of the position of the eyes in relation to the object.

The brain knows where the eyes are because of the perception of the difference in tonus between the oculomotor muscles. When looking straight ahead, there is tonic equivalence between the lateral and medial recti in each eye. When looking laterally, one of
these muscles has higher tonus than the other, as one is contracted and the other is relaxed. It is the perception of higher or lower tonus that indicates to the brain the location of the target object. By changing the muscular tonus of extraocular muscles, egocentric spatial localization can be changed. Furthermore, changing the perception of tonus of distant structures as the neck muscles or the Achilles tendon, can result in an illusory movement of the visual target, the direction of which depends on the muscles stimulated. Therefore, the correct perception of the relative tonicity of the oculomotor muscles is crucial. If there is a dysfunction of proprioception, as in PDS, this perception is altered.

**Complementary Tests**

Besides clinical examination and directional scotometry there are complementary tests that can further illustrate the neurological effects of PDS. These tests are not routinely used in the clinic but have been very useful for understanding the syndrome as a perceptual impairment disturbing multisensorial brain integration.

**Brain Mapping**

Brain mapping by computerized electroencephalography indicates that patients with Postural Deficiency Syndrome (PDS) present with higher values of absolute power (μV²) than the values of absolute power found in higher brain wave frequencies measured (Delta, Theta, Alpha, Beta 1 and Beta 2) (Figure 3a). Particularly high values are found for Delta frequency waves. When wearing the active prisms, the values of absolute power obtained for the Delta frequency become similar to those encountered in the other frequencies measured (Figure 3b).

The normal waking EEG in adults does not usually record expressive Delta waves. They should be present only during sleep, as Delta activity is inhibited by the ascending cholinergic neurons from the reticular activating system. If Delta waves appear as prominent in the waking record they usually suggest head trauma, intoxication, or demential cognitive impairment. The brain maps obtained in PDS patients, with none of the aforementioned conditions, suggest that there is a reversible dysfunction most likely involving the reticular formation. The same brain maps also indicate that the effect of active prisms is not restricted to a specific area or areas of the brain, but that the effect is associated with a diffuse change in specific low frequency waves, in particular the Delta waves.

**Directional Coordimetry**

Classic Coordimetry is the conventional test used to study ocular motor palsy with the Hess-Lancaster Screen. Classic Coordimetry is carried out in spirals, where mislocalization due to ocular motor palsy results in a skewed box diagram.
Directional Coordimetry is a modified protocol of Classic Coordimetry used to study the Postural Deficiency Syndrome (PDS). Directional Coordimetry is carried out line by line, right to left and left to right; and up-down and down-up. In PDS, shifts of localization that are not coherent relative to one another can be observed using Directional Coordimetry (Figure 4).33,7

The technique consists of showing the red lights of the Hess-Lancaster screen not one by one in nine positions as done in the classic spiral method, but line by line. It is done from left to right, and repeated from right to the left.

The first test simulates the eye movements while reading and the second test simulates eye movements while doing elementary mathematics exercises. Other directions had been tested during our research in order to show that the localization of the same point is not constant, and depends on the direction in which gaze is moving.

Note the following:
- Without active prisms there are points incorrectly localized, but the mis-localization does not correspond to Hering’s law for the pattern of displacement of the meridional points.
- The same point is localized differently depending on whether gaze direction is moving from left to right or right to left.
- With active prisms all points are accurately localized.
- Looking at the results with active prisms, it is not possible to detect any evidence of prismatic power. This means that the oculomotor system absorbed the prismatic power.

To summarize, in contrast with the results normally obtained in Classic Coordimetry, using Directional Coordimetry one can observe that active prisms correct inaccuracies in localization, but the images are not shifted by the
prismatic power. This indicates that the potency of the active prisms has been absorbed by the oculomotor system.

Computerized Visual Fields

When the first author started researching PDS at the University Hospital in Lisbon, most of the patients reported that they could see clearer immediately after putting on trial lenses with active prisms. This was not reflected in any change in visual acuity. However, computerized campimetry (Figure 5) showed visual paracentral absolute and relative scotomas. These scotomas were not indicative of any particular pattern of field loss associated with neurovisual or neurological insult or disease, and they disappeared with proprioceptive treatment. Apparently patients were seeing more clearly because their visual field had become intact.1,5

Treatment of PDS

Our treatment approach to PDS incorporates an active prism protocol reviewed in Tables 1 and 3. In addition to the prismatic lens protocol we employ a Postural Reprogramming program according to the Martins da Cunha Technique. This is done in conjunction with proprioceptive insoles, equipment to improve the ergonomics of a workstation as a reading ramp, and other proprioceptive stimulations that are beyond the scope of this article.

In cases of dyslexia and other learning difficulties, treatment also includes exercises involving cognitive training done by our psychologist, usually completed in six to ten sessions.

Active Prism Prescription Protocol

In 1955 the French scientist, neurologist and ophthalmologist J.B. Baron published a doctoral thesis12 in which he showed the effect of surgical alteration of extraocular muscles on the proprioceptive system in fish. Inducing a deviation equal or lesser than four degrees in the horizontal muscles resulted in the fish swimming in circles. When the same deviation was made in the oblique muscles the fish would swim in aberrant elliptical patterns. This occurred even in the absence of vision, leading him to conclude that there is a relationship between eye muscle tonus and paravertebral skeletal muscles based on proprioception.

J.B. Baron also showed in his practice that in some human patients having insufficiency of convergence, improvement was obtained through the use of low powered base out prism. That was quite unexpected, because it was written in all textbooks of strabismus that the direction of prism indicated to help under-convergence should be a base in rather than a base out prism.

The fact that many patients responded positively to base out prism was the intriguing fact that promoted the first author’s clinical investigation. Methodical patient evaluation brought the new knowledge that these cases were the ones we classified as left pure PDS type. Clinical investigation also concluded that patients with convergence insufficiency not responding to base out prism belonged to other PDS types that responded positively to other kinds of prisms. We therefore theorized that when prisms helped, we were eliminating the underlying problem that resulted in the postural condition rather than masking a symptom. This was the basis for the establishment of the active prism treatment protocol that was developed by the first author3 and validated in the following years.6

The protocol of active prism consists of prescribing low powered prismatic lenses from 1 to 4 diopters with base out or upper temporal base, applied to one or both eyes simultaneously.24,13,6,3

As it is known, humans do not possess purely vertical eye muscles. The superior rectus muscle is at a 23 degree oblique angle. This means that to elevate the eye there must be a simultaneous action of two muscles, the superior rectus and the inferior oblique muscle. In introducing a vertical prism, we are stimulating intorsion and extorsion simultaneously, and this may produce a proprioceptive conflict between the two actions.
In accordance with the previously mentioned works of Baron, there is a direct connection between oculomotor muscles and the musculoskeletal system. Therefore, it is not surprising that by stimulating oculomotor muscles we can observe changes in the perception of muscular tonus in different segments of the body. A vertical prism, by simultaneously stimulating two antagonist eye muscles, may produce a conflict between antagonist muscles in other anatomic locations.

This is in contrast to lateral prism or oblique prism largely targeting one muscle. As an example, base out prism in front of the right eye produces a horizontal image displacement to the left. In response, the eye tries to relocate the image to the right by relaxing the right lateral rectus muscle. Base out prism in front of the left eye produces a horizontal to the right, with relocation of the image made possible through relaxation of the left lateral rectus. Prism can be also be introduced obliquely to target the response of individual muscles. Applying an upper temporal prism in front of the right eye at 125 degrees, a relaxation effect is produced on the right inferior oblique muscle. Applying an upper temporal prism in front of the left eye at 55 degrees will relax the left inferior oblique. An important point to underline is that in using a base out or an upper temporal prism we are stimulating one oculomotor muscle at a time. Consequently, in targeting these muscles, we stimulate corresponding skeletal muscles that are proprioceptively related.

We must remember that the lateral rectus is an abductor muscle and that the inferior oblique has 3 different actions, external rotation (excyclorotation), abduction and elevation. We believe that in PDS patients the postural changes are due to a non-antagonized predominance of the external rotators, abductors and extensors.

Concerning the power of the prism, we have verified that the most frequent power needed is 2 or 3 diopters when both eyes needed upper temporal prisms. When only one eye needs a prism the most commonly needed power is 3 diopters if we select a base out prism, and 2 diopters if we select an upper temporal prism. This covers more than 90 percent of the cases.

In other cases we need to use lower or higher powers in the 1 to 4 diopter range. For instance, in cases of diplopia there is often indication to reduce the prism power. In cases where we do not attain correction of asymmetry in head rotation, there is often the need to increase the power of the prism. We increase or decrease prism in these cases in 0.5 diopter intervals.

As previously mentioned, PDS is classified in 6 types according to the postural clinical exam (head rotation, head extension and lower limb position) and directional scotometry evaluated by the synoptophore.

**Active Prism Prescription by Case Type**

For each type of PDS there is a specific active prism prescription as indicated in Table 1. When there are only pseudocotomas for one side of version or pseudoscotomas that exist in asymmetrical angles, the patient only needs one prism that is applied in front of the eye that is on the side of preferential plantar stance.

When there are pseudoscotomas for symmetrical angles of levoversion and dextroversion, the patient needs two prisms of asymmetrical power, as noted in Table 1 and summarized in Table 3. The higher powered prism will be applied on the side to which head extension is shorter.

The final decision of what active prism to prescribe is based upon clinical observation of the patient. All the semiologic tests described above are performed before and after applying the active prisms, with only a few minutes of interval in between. If they are normalized with the same profile of prism, the prism may be prescribed with confidence. If not, all parameters must be reviewed.

The correct active prism is the one that simultaneously corrects all of the previous clinical parameters. The only exception for this
is the tonic eye convergence test, that does not correct immediately in every case. When there is not a full correction of the clinical signs, the decision of what power of active prism to prescribe may be further aided by performing the Maddox test at distance.

Beyond delineating our prism protocol, it is equally important to contrast it with conventional approaches to ocular or postural disturbances or misalignment. Some authors use the Maddox test or other haploscopic tests in their protocol for prism prescription. Whenever the patient reports one image higher than the other, they interpret this as a vertical heterophoria. The phoria is then believed to be the primary cause of the symptoms. The solution proposed by these authors is to compensate the supposed phoria by prescribing a vertical prism. However, if we do a cover test on these patients, we will verify that there is no objective sign of refixation movement, nor a subjective sensation of image displacement.

In our opinion this is not a vertical heterophoria, but a deficit in localization. We refer to this as heterolocalization. This is usually evident in the horizontal plane on the near Maddox test, and in the vertical plane on the distance Maddox test. This aberrant localization mimics a heterophoria, but in reality is a sign of proprioceptive disturbance. Some ophthalmologic authorities even misinterpret this as a small compensated strabismus. The Maddox test is useful in our clinic as an aid in prescribing for cases of PDS mixed pure, but not to diagnose anything.

In the conventional assessment of binocular vision and strabismus, the Maddox test at distance is used to detect heterophorias. The same test is used in the assessment of proprioception, but with a different purpose. PDS patients do not usually have true heterophoria, but have a perception of the visual image as being displaced. We can confirm that this is not a true heterophoria because there is an absence of refixation movement on the cover test.

The same is true using the Maddox wing test. On this test the arrow is actually pointing to zero, but the PDS patient sees it as if it was pointing to 14 or 16 at the scale. Once more this is not an indication of true heterophoria, as confirmed by the absence of refixation movement on the near vision cover test.

To reiterate, the displacement of the visual image without strabismus is called heterolocalization. We believe this occurs because of a misperception of the muscular tonus of the oculomotor muscles. As it is known, the brain decodes the egocentric spatial localization of an object seen based on the perception it has of eye muscle tonus.

Conventionally, practitioners tend to split prism equally between the right and left eyes. For treatment purposes of PDS, when the Maddox test is normal in patients of the mixed pure type, the power difference between right

<table>
<thead>
<tr>
<th>PDS type</th>
<th>Peripheral Pseudoscotomas</th>
<th>More commonly prescribed active prisms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pure</strong></td>
<td><strong>Pseudoscotomas only to one side of version</strong></td>
<td><strong>levoversion at 20°</strong></td>
</tr>
<tr>
<td>right</td>
<td><strong>dextroversion at 20°</strong></td>
<td><strong>RE 180° 3Δ</strong></td>
</tr>
<tr>
<td>left</td>
<td><strong>dextroversion at 30°</strong></td>
<td><strong>LE 0° 3Δ</strong></td>
</tr>
<tr>
<td><strong>Mixed Predominant</strong></td>
<td><strong>Pseudoscotomas are asymmetrical occur in higher angle of version to the side of preferential plantar stance</strong></td>
<td><strong>RE 125° 2Δ or 3Δ</strong></td>
</tr>
<tr>
<td>right</td>
<td><strong>dextroversion at 20°</strong></td>
<td><strong>LE 55° 2Δ or 3Δ</strong></td>
</tr>
<tr>
<td>left</td>
<td><strong>dextroversion at 30°</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Mixed Pure</strong></td>
<td><strong>Pseudoscotomas for symmetrical angles of version</strong></td>
<td>**RE 3Δ 125° **</td>
</tr>
<tr>
<td>right</td>
<td><strong>levoversion at 20°</strong></td>
<td>**LE 3Δ 55° **</td>
</tr>
<tr>
<td>left</td>
<td><strong>dextroversion at 20°</strong></td>
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<tr>
<td></td>
<td><strong>or levoversion at 30°</strong></td>
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<tr>
<td></td>
<td><strong>dextroversion at 30°</strong></td>
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and left active prisms may be reduced. In the other types of PDS, however, reducing the power difference measured or derived by observation may lead to a false prescription protocol.

**SUMMARY AND CONCLUSION**

The proprioceptive system has an extensive influence on the maintenance of human health. When the proprioceptive system is dysfunctional, the central nervous system does not recognize the correct status of tonicity of the muscles at rest or in motion, does not integrate correctly the information that comes from sensory receptors, and has difficulty in modulating multi-sensory integration, with consequences in motor behavior and cognitive function. This results in a wide range of proprioceptive abnormalities which are clinically related and are treated together, termed as Postural Deficiency Syndrome (PDS).

It is possible to influence body proprioception by stimulating not only direct mechanoreceptors like neuromuscular spindles, or joint or tendon neurologic terminations, but also by modulating the information from other sensory input as well. These range from visual receptors connected to the retinocolicular pathway in the uppermost region of the body to the sole plantar receivers underfoot.

The first author’s contribution to this field extends the work of Martins da Cunha by using an active prism protocol to treat postural asymmetries that produces a change in oculomotor system and modifies the visual information input. This therapeutic effect is based on the knowledge that small modifications of the muscular tonus of the oculomotor muscles can change the tonus of the axial paravertebral muscles.

Prisms have been conventionally prescribed by arbitrarily splitting the power between the two eyes, or by taking into account asymmetries exhibited in sensory projection of binocular targets. The diagnostic protocol the first author has devised goes beyond this to encompass pseudoscotomas observed on versinal movements in horizontal gaze which we have termed directional scotometry; asymmetry in head tilts and rotations; and rotation in supporting heel and foot support. Our treatment protocol then applies prisms asymetrically to each eye to effectively produce changes in these visual, head, and body asymmetries.

Our diagnostic and treatment protocol is constantly being refined. To date we have seen success in the treatment of a variety of functional types of oculomotor dyscoordination ranging from binocular disturbances such as convergence insufficiency, near exotropia and asthenopia, to unilateral visual disturbances such as monocular diplopia, paracentral scotomas of the visual field, and metamorphopsia. PDS and its treatment can extend to certain types of cognitive dysfunction, functional ataxia, dyspraxia, dyslexia, postural imbalances, and pain. Low powered active prisms, up to four diopters, can influence blood circulation as shown by arterial Doppler ultrasound.

An understanding of the significance of the visual system in PDS provides an important role for the vision professional in managing proprioceptive dysfunctions. Relatively inexpensive, non-invasive, and accurate diagnosis is possible leading to more effective treatment of PDS patients with significant impact on their quality of health and life.

**REFERENCES**


34. Sherrington CS (1906) The integrative action of the nervous system. Yale University Press, New Haven, CT, US


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Dr. da Silva was the Portuguese lead for the European project on infant videorefractive screening programs, coordinated by professor Janette Atkinson, and a founding member and first president of the Portuguese Group of Pediatric Ophthalmology and Strabismus. He was a member of ESA, and has presented his group’s research at numerous international conferences as keynote speaker.

He is a Visiting Professor in several postgraduate courses encompassing Italy, France, Spain and Portugal, and has published six books on the treatment of Proprioception Dysfunction Syndrome.