Neurology training for non-Neurologists in West Africa:
Focus on Parkinson’s Disease and other Neurodegenerative Disorders

ACCRA
GHANA
SEPTEMBER 15-18 2013

Ghana Institute of Management and Public Administration (GIMPA)

Posture, Gait and Balance Disorders in Parkinson’s disease

Marianna Amboni
Centro Malattia di Parkinson e disturbi del movimento, CEMAND, Salerno, Italy
IDC Hermitage-Capodimonte Napoli, Italy
Outlines

- Overview on postural deformities in PD

- Gait and balance disorders and the interplay between gait and cognition in PD
Postural deformities in Parkinson’s disease

Karen M Doherty, Bart P van de Warrenburg, Maria Cecilia Peralta, Laura Silveira-Moriyama, Jean-Philippe Azulay, Oscar S Gershank, Bastiaan R Bloem

Figure 2: Sagittal plane deformities (A: camptocormia, B: antecollis) and coronal plane deformities (C: Pisa syndrome, D: scoliosis)
Camptocormia: Definition

Camptocormia
Marked (minimum 45°) flexion in the sagittal plane originating in the thoracolumbar spine, almost complete resolution in the supine position

Figure 3: Camptocormia in the standing, seated, and supine positions
Management

Rehabilitation interventions

- Manipulative physiotherapy
- Hydrotherapy
- Use of neck collars, lumbar support belts, and orthotic device

Other interventions

- Adjusting PD medication
- Botulinum toxin therapy
- STN DBS, Gpi DBS or PPN DBS
- Spinal deformity surgery
- Anticholinergic agents

---

Table 1. Conditions associated with camptocormia

<table>
<thead>
<tr>
<th>Conditions</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CNS disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Organic</td>
<td></td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td>19, 27, 33, 60</td>
</tr>
<tr>
<td>Dystonias</td>
<td>2, 14, 28, 38</td>
</tr>
<tr>
<td>Abdominal segmental dystonia</td>
<td>37</td>
</tr>
<tr>
<td>Multisystem atrophy</td>
<td>34, 35, 65</td>
</tr>
<tr>
<td>Alzheimer’s disease</td>
<td>36</td>
</tr>
<tr>
<td>Basal ganglia disorders</td>
<td>14</td>
</tr>
<tr>
<td>Essential tremor</td>
<td>14</td>
</tr>
<tr>
<td>Viljuisk encephalomyelitis</td>
<td>26</td>
</tr>
<tr>
<td>Psychiatric</td>
<td></td>
</tr>
<tr>
<td>Gilles de la Tourette syndrome</td>
<td>28</td>
</tr>
<tr>
<td>Conversion disorder</td>
<td>41</td>
</tr>
<tr>
<td>Psychogenic</td>
<td>65</td>
</tr>
<tr>
<td>Oppositional defiant disorder</td>
<td>41</td>
</tr>
<tr>
<td>Neurosis</td>
<td>14</td>
</tr>
<tr>
<td><strong>PNS disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Primary myopathies</td>
<td></td>
</tr>
<tr>
<td>Myotonic dystrophy type 1</td>
<td>16, 21</td>
</tr>
<tr>
<td>Myotonic dystrophy type 2</td>
<td>39</td>
</tr>
<tr>
<td>Axial myopathy</td>
<td>7, 84</td>
</tr>
<tr>
<td>Dysferlinopathy</td>
<td>24</td>
</tr>
<tr>
<td>Nemaline myopathy</td>
<td>20, 26</td>
</tr>
<tr>
<td>Mitochondrial myopathy</td>
<td>40, 47</td>
</tr>
<tr>
<td>Congenital myopathy</td>
<td>40</td>
</tr>
<tr>
<td>XMPMA</td>
<td>85</td>
</tr>
<tr>
<td>Secondary myopathies</td>
<td></td>
</tr>
<tr>
<td>Hypothyroid myopathy</td>
<td>43</td>
</tr>
<tr>
<td>Isolated thoracic extensor myopathy</td>
<td>48</td>
</tr>
<tr>
<td>Inflammatory myopathy (PM)</td>
<td>18, 40, 44</td>
</tr>
<tr>
<td>Dermatomyositis</td>
<td>40, 44</td>
</tr>
<tr>
<td>Chronic axial myositis</td>
<td>41</td>
</tr>
<tr>
<td>Focal myositis\1</td>
<td>45, 46</td>
</tr>
<tr>
<td>Inclusion body myositis</td>
<td>3, 26</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>47</td>
</tr>
<tr>
<td>Myopathy with nemaline rods</td>
<td>20</td>
</tr>
<tr>
<td>Facioscapulohumeral muscular dystrophy</td>
<td>26</td>
</tr>
<tr>
<td>Neurogenic</td>
<td></td>
</tr>
<tr>
<td>Amyotrophic lateral sclerosis</td>
<td>26, 48, 50, 75</td>
</tr>
<tr>
<td>CIDP</td>
<td>51</td>
</tr>
</tbody>
</table>

---

Drugs

- Olanzapine: 57
- Donepezil: 59
- Valproate: 60, 86
- Systemic steroids: 27

Varia

- Lumbar disc herniation: 52
- Dystonia from neck trauma: 53
- Arthritis: 61
- Trauma: 61
- Malignancies: 54
- Idiopathic: 26

PM = Polymyositis; CIDP = chronic inflammatory demyelinating polyneuropathy; XMPMA = X-linked myopathy with postural muscle atrophy. § Camptocormia in Parkinson’s disease and multisystem atrophy.
Antecollis: Definition

Antecollis
Marked (minimum 45°) neck flexion (maybe partially overcome by voluntary or passive movement), unable to fully extend the neck against gravity but able to exert force against the resistance of the examiner’s hand
Mechanism and treatment of dropped head syndrome associated with parkinsonism

Genko Oyama

Differential Diagnosis

Weakness?
If there is weakness, consider:
• Myasthenia gravis
• MND, polio/post-polio syndrome
• LGMD, FSHD
• IBM, other myositis
• Amyloid, thyroid myopathy

Fixed deformity?
If the deformity is fixed, consider:
• Ankylosing spondylitis
• Vertebral pathology (eg, fracture)
• Spinal cord pathology (eg, syrinx)
• Idiopathic or degenerative scoliosis

Management

Rehabilitation interventions
➢ Intensive physiotherapy
➢ Use of neck collars and orthoses

Other interventions
➢ Adjusting Pd medication
➢ Clonazepam
➢ Botulinum toxin therapy
➢ DBS ?
Postural deformities in Parkinson’s disease

Karen M Doherty, Bart P van de Warrenburg, Maria Cecilia Peralta, Laura Silveira-Moriyama, Jean-Philippe Azulay, Oscar S Gershani, Bastiaan R Bloem

Definition of PISA syndrome (lateral trunk flexion) in PD

- More than 10° lateral flexion of the trunk
- Increasing during walking
- Not present when supine

Pisa Syndrome: Clinical features

- **Onset:** Insidious vs Subacute
- **Back Pain:** Common
- **Awareness:** absent (impaired perception of subjective vertical)
- **Worsening on walking, running... other kinematic trigger?**
- **Fully reversible deformity when supine**
PISA Syndrome while driving car

Role of kinematic stimulation: the car sign….
Pisa syndrome

Rehabilitation interventions
- Postural physiotherapy
- Use of orthotic devices

Management

Other interventions
- Adjusting Pd medication
- Spinal surgery
- Anticholinergics, Clozapine
- Botulinum toxin therapy?
- DBS?
Possible mechanisms involved in the development of postural deformity in Parkinson’s disease

(From Doherty et al, 2011)
Outlines

- Overview on postural deformities in PD

- Gait and balance disorders and the interplay between gait and cognition in PD
Gait disorders: a “vicious circle”

(Da Bloem et al, 2004)
Postural control systems involved in balance

(From Schoneburg et al, 2013)
Gait and Balance Examination in Parkinsonism
(modified from Bloem & Commissaris in Overstall Ed, 2002)

• **Gait:** Speed, armswing, arm tremor while walking, step height and length, stance width

• **Balance:** Supporting reactions (quiet stance, eyes open/closed), anticipatory reactions (lifting objects from floor), reactive postural responses (pull test)

• **Tasks multiple:** Motor task (walking while carrying a tray with two glasses), cognitive task (walking while performing a verbal fluency task)

• **Freezing of gait (FOG):** start and turning freezing, freezing during tasks
Freezing of gait

Freezing of gait (FOG) is a brief, paroxysmal event characterized by absence or marked reduction of forward progression of the feet despite the intention to walk.

During FOG patients generally feel their feet glued to the ground.

FOG is frequent in patients with advanced Parkinson’s disease (PD), and has been found in up to 26% of early stage PD patients (Giladi et al, 1992; Giladi et al, 2001).
Freezing of gait (FOG)

FOG generally appears in gait initiation, in turning, in walking through a narrow passage, while performing another task, rarely when the patient tries to reach a destination or in open space.

(From Snijders et al., Mov Disord 2008)
FOG and motor state

✓ The relationship between FOG and dopaminergic medication is quite complex.

✓ Based on the dopaminergic responsiveness, it is possible to identify:

1. Treatment responsive FOG, the most common form (Schaafsma et al, 2003)

2. Non-responsive FOG which is indifferent to changes in dopaminergic medication (Bloem et al, 2004; Narabayashi H, 1980)

3. Drug-induced FOG, the rarest form (Ambani et al, 1973; Espay et al, 2012)
FOG during off

FOG during on
Drug-induced FOG
Management of FOG

**Pharmacological approach**
- Levodopa increase/decrease
- Monoamine oxidase type-B inhibitors

**Rehabilitation approach**
- Cueing strategies
- Use of cane with laser light visual cue
- Use of devices with auditory cues

**Other interventions**
- STN DBS
- PPN DBS
- Botulin toxin in legs muscles
Freezing of Gait and Executive Functions in Patients with Parkinson’s Disease

Marianna Amboni, MD,1,2 Autilia Cozzolino, MD,1 Katia Longo, MD,1 Marina Picillo, MD,1 and Paolo Barone, MD, PhD1*

1Department of Neurological Sciences, University “Federico II.” Naples, Italy
2Istituto di Diagnosi e Cura “Hermitage Capodimonte,” Naples, Italy

TABLE 2. Cognitive evaluationa

<table>
<thead>
<tr>
<th></th>
<th>FOG+ (n = 13)</th>
<th>FOG− (n = 15)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbal fluency</td>
<td>29.19 ± 5.97</td>
<td>37.50 ± 9.43</td>
<td>0.011</td>
</tr>
<tr>
<td>FAB</td>
<td>14.0 ± 2.16</td>
<td>15.95 ± 1.33</td>
<td>0.008</td>
</tr>
<tr>
<td>TPCT</td>
<td>5.85 ± 2.70</td>
<td>7.73 ± 1.33</td>
<td>0.024</td>
</tr>
<tr>
<td>Stroop II</td>
<td>36.55 ± 8.68</td>
<td>44.67 ± 4.73</td>
<td>0.004</td>
</tr>
<tr>
<td>Stroop III</td>
<td>18.24 ± 5.3</td>
<td>21.42 ± 3.82</td>
<td>0.077</td>
</tr>
</tbody>
</table>

Values are mean ± SD.
aScores are age and education adjusted.
FAB: frontal assessment battery; TPCT: ten-point clock test.
The innovative model linking cognition and gait

Possible line of intervention

Cognitive impairment

Gait abnormalities

Dementia

Falls/immobility

Integrative tools for risk estimation

(from Amboni, Barone & Hausdorff, 2013)
Summary

✓ Postural deformities are frequent and disabling complications of PD; they include camptocormia, antecollis, Pisa syndrome, and scoliosis. Early detection of postural abnormalities could help to prevent fixed irreversible deformities.

✓ FOG is a common and disabling symptom in PD patients; it appears to be closely interconnected with falls.

✓ The relationship between FOG and dopaminergic medication is quite complex, suggesting that the pathophysiology of this phenomenon is more complex than the pathophysiology of the classic motor symptoms of PD.