Abnormal Gait Assignment: Classic NPH Gait

Normal pressure hydrocephalus (NPH) results from an increase in intracranial pressure (ICP) due to an abnormal accumulation of cerebrospinal fluid (CSF) in the ventricles. The increased pressure upon the cortical tissue causes a wobbly gait pattern termed classic magnetic gait. The Hydrocephalus Association's video,1 titled Classic NPH Gait Pre-Shunt Surgery, presented a patient with a classic magnetic gait pattern. The patient’s gait was analyzed for cadence, step length, and gait deviations in the patient's trunk, pelvis, hip, knee, ankle, and toes.

The video began showing the patient shuffling with his feet seemingly stuck to the floor. The patient ambulated independently and presented with short steps, stooped posture, and difficulty with tandem gait. After 20 seconds, despite his efforts, the patient experienced a neurological episode and could not continue ambulating. The patient thereafter appeared to be in the marked stage of NPH, defined as a considerably unstable gait with decreased step height, foot-floor clearance, and walking speed.2 The patient compensated by increasing step width and the foot rotation angles. Despite these compensations, the patient displayed an increased tendency to fall backwards.

By visual estimation, the patient had less than normal values in velocity (norm 74.3 m/min) and stride length (norm 1.34 m).3 The patient was expected to display a lower cadence in pre-episode (100 steps/min) than post-episode (80 steps/min), but instead showed the opposite. The severity of the episode prevented the patient from advancing, thereby reducing the cadence.

The patient's trunk exhibited a slight forward lean during the Single Limb Support task (SLS). The forward lean was as a compensation for impaired proprioception in order to increase visual input and maintain balance. This posture moved the patient's center of mass forward to increase forward progression and impacted the critical events of Loading Response, Mid-Stance and Terminal-Swing. Post-episode, the patient shifted his trunk to the left and pressed his arm against the wall for balance. This compensation helped accomplish foot clearance and limb advancement during the SLA task. The lateral trunk lean increased energy cost and decreased forward momentum.

At the pelvis, the patient exhibited posterior pelvic tilt, hikes, and lack of backward and forward rotation. The hikes and posterior pelvic tilt were a compensation to clear the swing limb and help execute the critical events during SLA. Due to the impaired motor control associated with NPH, the lack of backward and forward rotation contributed to decreased step length.

The principal aberration in the hip region occurred during Initial-Swing and Mid-Swing. During the patient’s gait, critical hip flexion was not reached and interfered with thigh advancement and foot clearance, respectively. This deviation also obstructed his limb advancement and foot clearance, the two accomplishments in SLA. Impaired motor control in the foot prevented the patient from flexing the hip
rapidly during Initial-Swing. This interfered with the accomplishment of foot clearance, which did not occur normally during Mid-Swing.

Post-episode, the patient presented with limited hip extension, which altered the accomplishment of forward progression during SLS. During Mid-Stance and Terminal-Stance, the patient was unable to progress his body over a single limb. In order to generate forward momentum, as a compensation for this lack of hip flexion, the patient utilized a stooped, forward-leaning posture to progress his body over his single.

At the knee joint, pre-episode, the patient compensated for inadequate hip flexion with just enough knee flexion for foot clearance. However, the level of knee flexion falls short of the normal peak degrees during Initial Swing, and the step length is consequently shortened. Post-episode, the patient presented with significant wobbles during Weight Acceptance (WA) and SLS. The right limb wobbled by quickly oscillating between knee flexion and extension, thus never allowing for full flexion. Knee flexion is a critical event during Pre-Swing and Initial-Swing and failed to occur at optimal levels. These impairments were likely caused by decreased proprioception and knee flexor strength. This resulted in increased energy expenditure by decreasing shock absorption and the forward momentum of the patient.

At the foot, the patient avoided normal WA by landing flat-footed and excessively everted. This compensated for weak quadriceps, but decreased shock absorption and reduced forward momentum of the tibia. During Terminal Stance and Terminal Swing, there was insufficient heel-off and MTP extension, respectively. These deviations interfered with forward progression and reduced step length and were potentially due to weakness in the plantarflexors and the toe extensors. Throughout SLA, the patient exhibited Up^3 toes to compensate with foot clearance. This deviation was caused by insufficient dorsiflexion and weak pre-tibials.

The abnormal gait analysis showed classic magnetic gait as causing problems primarily in Swing Phase of the normal gait pattern. Deviations in classic magnetic gait also interfered with normal processes in Stance Phase, such as the forward trunk lean during Loading Response and limited hip extension during Mid- and Terminal Stance. The patient also presented several defining gait characteristics: reduced walking speed, significantly shorter strides (stride length), decreased foot-to-floor clearance and a broad-based gait.

References