Management of Apraxic Gait in a Stroke Patient

Puangpeth Jantra, MD, Trilok N. Monga, MD, Joel M. Press, MD, Bradley J. Gervais, BS


There is little information available regarding management of apraxic gait. We present a 61-year-old man with a five-year history of right-sided cerebrovascular accident, apraxic gait, difficulty in walking, and frequent falls. A CT head scan revealed moderate cerebral atrophy, a small lacunar infarction. The patient was unable to initiate walking, was bed ridden and housebound. Traditional gait training and balance exercises failed to improve his gait. Two straight canes were modified by fixing florescent horizontal projections approximately two inches up from the tip of the cane. The patient became independent with safe ambulation after practicing for approximately three weeks and was discharged home.

KEY WORDS: Apraxic gait; Management; Stroke

Apraxia is defined by Gerschwind as “a disorder of learned movements which cannot be accounted for by weakness, sensory loss, incoordination, inattention, or comprehension deficit.” In 1920, Liepmann classified apraxia into three categories: 1) ideomotor apraxia, 2) Limb-Kinetic apraxia, and 3) ideational apraxia.

Ideomotor apraxia is characterized by failure to perform previously learned activities. It is the most common type of apraxia. Three types of ideomotor apraxia have been described: 1) limb apraxia, including apraxia of gait; 2) trunkal apraxia; and 3) buccofacial apraxia.

Meyer and Barron in 1960 defined the apraxia of gait as the loss of ability to use the lower limbs in the act of walking and noted that this loss cannot be accounted for on the basis of sensory impairment or motor weakness. Meyer concluded that any disease affecting the mesial aspect of the frontal and parietal lobes may produce gait apraxia.

There is little information available regarding management of apraxic gait. We report a stroke patient who improved in his walking with the use of two modified canes.

CASE REPORT

A 61-year-old male patient presented with a five-year history of difficulty in walking. The patient had a right-sided cerebrovascular accident in 1983 with mild residual left-sided weakness; at that time, he was able to ambulate safely with a straight cane. He was diagnosed to have frontal lobe apraxia in 1984, and since then, his gait gradually had deteriorated, and he presented with a history of frequent falls. He was bedridden and housebound upon admission to the Rehabilitation Medicine Service in 1988. His history included controlled hypertension, stable congestive heart failure, and ethanol abuse.

Physical examination revealed that the patient was alert, oriented, and had normal speech. Facial expression was flat. There were no visual field deficits. He had signs of mild left-sided hemiparesis with no sensory deficit, and the deep tendon reflexes were normal. There was no evidence of cerebellar dysfunction, tremor, or rigidity. No perceptual deficits were noted except for the apraxic gait. Although there was good motor recovery, the patient was unable to initiate walking and to draw a circle with either foot. He was independent in activities of daily living. No apraxia was noted in the upper limbs.

A CAT scan of the head showed moderate cerebral atrophy and low density area in white matter, most likely representing small lacunar infarction. Carotid Doppler Study revealed normal hemodynamics and a small focal plaque at the bulb of the left common carotid artery. Echocardiogram showed a mural thrombus.

Management

A neurologist and a cardiologist were consulted; there was no evidence of either hydrocephalus or Parkinson’s disease, and it was believed that the patient was at a high risk for anticoagulant therapy.

The initial rehabilitation program included traditional gait training, general exercise program, and safety education. The patient did not show improvement. The use of ankle weights for the purpose of increased proprioceptive feedback also proved ineffective. The gait training program was then modified; the patient was asked to step over grey and red colored stripes and paper sheets placed on the floor. Using cues from the stripes and paper sheets, he was able to initiate walking. Walking was noted to be better when the colored stripes were used. However, as soon as the cues were removed, his ability to walk would regress, and there was no carry-over from one session of gait training to the next. It was also noted that the patient had more difficulty in initiating the gait with his left foot.
It was obvious that the patient needed ongoing cueing if he was to improve his ability to walk independently. As color coding for right and left, back and front have previously been recommended for ongoing cognitive cues in patients with dressing apraxia, it was decided to provide the patient with two modified straight canes. The canes were modified by attaching horizontal projections at a point approximately two inches up from the tip of the cane (figs 1, 2, 3). The projections were painted orange with fluorescent paint. The patient was asked to step over these projections during the swing phase of his gait cycle. Using this technique, he was able to initiate gait, maintain walking, and learned to ambulate safely. Although he needed further instructions in making turns, he was able to negotiate stairs and used the modified canes on the bottom and top of the stairs.

The patient progressed to where he needed just one modified cane for safe ambulation. The discarded cane was from the right hand. The patient had no difficulty in managing the left hand cane despite very mild weakness on that side. He was discharged home after a 23-day stay. Prior to his discharge, the family was instructed to supervise the patient for outdoor ambulation and to mark colored stripes over the floor inside the house so that the patient could walk independently without the cane.

Follow-up at one year postdischarge revealed that the patient remained independent within the house without canes and for limited community walking with one modified cane in his left hand.

DISCUSSION

The severity of gait abnormality and inability of this patient to ambulate could not be explained upon the mild residual left-sided weakness. Neither was the clinical picture compatible with Parkinsonism. It was concluded that gait apraxia was the cause of the patient's problem in walking.

There is little information available regarding management of apraxic gait. In dressing apraxia, cueing with color coding of the sleeves has been tried. Meyer, in 1960, used a mechanical walker mounted on the wheel for training of a patient with apraxic gait. This patient did not show any significant improvement.

Estanol, in 1981, reported that one has to identify a stimulus that would help the patient initiate and maintain walking and that one may have to try various stimuli in a given patient. In our patient, stimuli that could successfully initiate a response were either stepping over the colored stripes...
on the floor or over spread out paper sheets. However, there was no carry-over from one session to the next. Eventually the patient was able to receive the cueing from the colored horizontal projections of the inverted canes and became independent with safe ambulation.

CONCLUSION

We have presented a patient with a history of cerebrovascular accident resulting from a lacunar infarction. The patient was diagnosed to have apraxia of gait. Independent and safe ambulation was achieved by cues received from painted horizontal projections attached to the straight canes and by marking colored stripes over the floor. This case illustrates the importance of identifying a stimulus to provide effective cueing in a functional and safe manner. This case further illustrates that without appropriate rehabilitative intervention, the patient would have remained housebound and bedridden. To our knowledge, such an approach has not been previously reported.

References