

PARKINSON'S DISEASE

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1. Introduction

Parkinson Disease is a progressive extrapyramidal syndrome which is due to a disfunction of the basal ganglions. This illness was described by doctor James Parkinson in 1817. The Parkinson Disease and the parkinsonian syndrome record a group of disorders described by tremor and alteration of voluntary movement, posture and balance.

The "Parkinsonian Syndrome" is the group of alterations in which the parkinsonism characteristic symptoms and signs develop to, but in a secondary form to another neurological illness, like for example in a vascular origin parkinson or in the Alzheimer´s disease. Therefore, meanwhile the Parkinson disease is a primary degenerative disorder which is produced in the second half of life and follows a progressive development, the parkinsonian syndrome has a natural history regarding its own cause.

There are two types of extrapyramidal syndromes:

a- Akinesic extrapyramidal syndrome type – rigid: This takes place in Parkinson disease and syndrome.

b- Hyperkinetic or hypotonic extrapyramidal syndrome type: these are, for example, chorea, athetosis, hemibalism, dystonies.

The extrapyramidal system has the following functions:

a- Control and adjustment of automatic movement sequence. When this function is damaged, tremor and dystonia are produced.

b- Regulation of muscle tone; when this function is altered, stiffness, akinesia or bradykinesia are produced.

c- Regulation of posture reactions.

d- Harmonization of the motor activity.

2. Etiology

Symptomes and signs of parkinsonismo have their origin in a change in the function in two regions on the basal ganglions: black substance and striatum (Caudate nucleus and putamen). These nuclear central masses of grey matter in the brain contain almost all dopamine of the human brain. Dopamine is a chemical substance and one of the neurotransmitter amines which transport the electrical message from one neuron to the next through synapse.

Parkinson disease is responsible for the enormous proportion of parkinsonism cases. The reason why the black substance and striatum degenerate is unknown, but it is a progressive process which lasts around 10 to 15 years, from the time it starts until death. In the worst cases, the increasing immobility leads to associated complications such as: pressure ulcers, weight loss and respiratory complications, which are the habitual death cause.

3. Clinic

This illness presents a characteristic clinic which will be announced next. Later, every point will be explained in detail.

- a- Akinesia: Lack of movement, or hypokinesia, which is the reduction in movement.
- b- Rigidity: this is the increase in muscle tone no velocity depending. Therefore it is different to spasticity.
- c- Tremor at rest: This point is very important. There can be two types of tremor as a neurological sign: intention tremor, like happens in the cerebellous syndrome, or rest tremor, like happens in the parkinson patient.
- d- The start of this illness uses to be insidious.
- e- Two types of Evolution may be: slow (less or equal to 10 years) or quick (less than 4 years).

Akinesia: Effects that produce akinesia or hypokinesia in ill parkinson patients.

- a- Difficulty to do two patterns of simultaneous movements, like for example, get up and shake hands.
- b- Delay and slowness in the beginning and execution of voluntary movements.
- c- Loss of voluntary and automatic movements, like for example, they lose the normal sequence of eye blinking, they lose the swinging of the pelvic and shoulder girdle during walk. This last effect risks the balance of the body during the walking phases.
- d- Amimia; Reduction in the face mimical gestures. "Facies in Mask". A sensory aphasia characterized by the loss of ability to communicate by gestures or signs.
- e- Fatigue in the execution of repetitive movements.
- f- Dysphagia.
- g- Slow, monotonous and low modulated voice.
- h- Slow walk, short steps, with no arm movement, difficulty to turn when walking and to turn in prone position.
- i- Alteration in writing. They use to have very small letter (micrography). This alteration is because of the disfunction in the fine coordination movements that involve writing.
- j- Slowness in the realisation of the daily activities

Stiffness: Effects that produce stiffness in the parkinson disease patients.

- a- It is shown a waxy stiffness; this is a continuous stiffness, it doesn't have knife effect, and does have the "Toothed Wheel" phenomenon. This effect appears when tremor and stiffness meet.
- b- As a consequence of stiffness, patients suffer from muscle pain, motor clumsiness, and they don't have sensitivity disorders.
- c- Biomechanically, they acquire a hypercyphotic posture at bipedestation, even at sedestation.

Tremor at rest: Identification of Parkinson tremor

- a- Uses to be rhythmic.
- b- Shows a slow oscillation; approximately of 4 to 6 cycles per second.
- c- Initially unilateral affectation of one of the hands.
- d- Reduces with activity.
- e- Reduces with sleep.
- f- Increases with emotional stress.
- g- The head doesn't use to show tremor.

4. Valuation of Parkinsonian patient

The valuation may be structured in the following way:

Valuation of the locomotor system:

Mechanical Valuation:

Joint analysis

Muscle analysis.

Neurological Valuation:

Posture

Balance

tremor.

Functional Valuation:

Daily activities

Fine mobility

walk.

Other systems:

Psychic.

Other Systems.

a- *Posture:* It uses to be a trend to posture in flexion, hyperciphotic in bipedestation which endures in sedestation.



b - *Balance:* Balance valuation must be done in bipedestation, in sedestation and in monopodal support. In these valuations, you must explore balance in front, back and side direction.

c- *Functional valuation:* Evaluation of capability to handle easy tasks, walk valuation, fine mobility valuation, incapacity level valuation. You will associate these valuations to a complete joint analysis in order to estimate possible retractions, and a muscle analysis to determinate the level of tendon shortening.

d- *Valuation of other aspects such as:* tremor, psychic state, respiratory disorders.

5. Physical Therapy Treatment

The goal in this disease is helping the patient to keep his independence as long as possible, and give advice to family and assistants how to manage the person affected by the illness whenever the activity progresses and incapacibilities arise. At this point, the family must adquire certain specific characteristics because some of them may increase with emotional stress, as described in the clinic.

The treatment will start early in the evolution of the disease and will be last all his life, with the schedulling of house exercises and the stablished medical surveillance.

These patients have lost automatism, therefore it is important to achive extensive, repetitive, rhythmic and armonic movements

We can organize the treatment in the following way:

1. Cinesitherapic Activities.

1.a- To prevent and treat retractions:

We try to prevent them by passive and active assisted mobilisation. If retractions are already stablished, we will use thermotherapy, passive cinesitherapy reenforcing shortened muscle elongation, concentric work of the antagonist muscle to the shortened and the use of progressive splints.

2. Control of posture trend at flexion.

In order to achieve it, we will do:

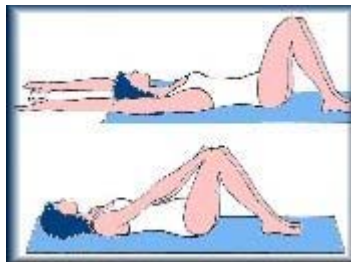
Concentric exercises of trunk extension.

Concentric exercises of hip ABD.

Concentric exercises of knee extension.

Concentric and alternative exercises of dorsal and plantar ankle flexion.

We must bear in mind that in these patients a ventralisation of the center of gravity is produced. Therefore, the flexor patterns increase over the extensors. Following this deduction, it is so important to reduce these patterns as to inhibit them. Then these exercises are better to be done with the patient on a prone position due to the fact that this position itself inhibits already the flexor patterns. On the other hand, the displacement of the gravity center that they suffer doesn't matter any more in this position. Normally in the prone position, tremor is reduced.



3. Exercises for self-autonomy:

3.a- Exercises to be done on the bed or on the floor:

These exercises are done by changing from one decubitus to another, paying attention when turning the initial rotation of the neck and helping the patient himself with his upper limbs to turn.

Movements of one upper limb and one lower opposite limb, imitating the scapular pelvic dissociation that must be produced during walk.

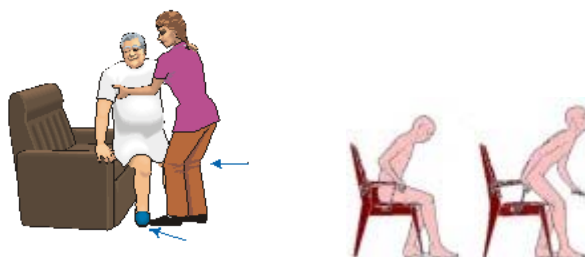
Rhythmic, symmetric and simultaneous mobilisations of 2 upper limbs, of 1 lower limb and of 2 lower limbs simultaneously.

From the floor to the bed, we do sit-up exercises or elevation from prone position, assisting himself with his lower limbs.

3.b- Sedestation exercises:

These exercises must be done once the patient controls the mentioned exercises of the former section.

Sitting-down and getting-up exercises, verifying that he does flexion on the trunk to move the gravity center, helping himself with his upper limbs. In case the patient isn't able to do so, he can be assisted in the transit from sedestation to bipedestation.



Active exercises of flexion, extension and rotation of neck and trunk. These exercises must be done in a rhythmic way. We even enumerate the movements in order to stimulate their integration.



Balance stimulating exercise to the pushing in sedestation. In order that the patient keeps a good front, back and side balance in bipedestation, he must do these exercises in sedestation before.

3.c- Bipedestation exercises:

As before, it is strongly recommended that you perform these exercises after achieving the previous ones.

Posture control exercises at the mirror. With these exercises, we work posture symmetry mostly in frontal plane. If there is possibility to have more mirrors, we will do the same in the saggital plane, so that the patient is able to look up at himself and modify his posture.



Sit-up exercises from the floor or sit-down exercises on the floor. If he can stand them, the patient can do them helping himself with the upper limbs, at different rythims and speeds.

Balance exercises, progressively reducing the support base until it becomes a monopodal support.

Static walk exercises with coordinated swinging movements of upper and lower limbs, simulating walking back and forwards, walk overpassing small obstacles.

Weight tranfer exercises and turns on its vertical axis.

3.d- Walking exercises:

Very long steps must be done, with a lot of knee flexion and extension. Generaly, these are very exagerated steps.

You should avoid stucked postures. When the patient is about to start the walking activity and he remains stucked, we should give him a push or a stimulus to start the pace. We can also avoid so by making him follow symmetric signs on the floor, like for example, painted footprints.



To practise walking backwards and sideways, avoiding obstacles.

6. Personal Considerations:

As it happens in all the degenerative pathologies, when we face a patient with these characteristics, it is necessary that the enthusiasm to make them functional people is heavier than other reasons. This pathology doesn't have to be associated to a cognitive degrade. And this produces a problem in the patient that we must reduce, since him or her are aware of everything his or her body is experiencing. We must transform its clinic in something possitive with the medical and physiotherapeutic activities and occupational therapy. We must translate these actions into an improvement in their life quality, which is a right every human being has, with independence of our base clinical situation.

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