

PARKINSON DISEASE

Paralysis Agitans

Year TWO

GENERAL INFORMATION

Parkinson disease, which is also known as paralysis agitans, Parkinsonism and shaking palsy, is one of the more common causes of a tremor in elderly people.

About 60,000 Australians suffer from the condition (1% of those over 65), and 10% are under 50 years. The mean age of onset is 60 years.

It is named after the English physician James Parkinson (1755-1825) who first described the condition in the medical literature.

SYMPTOMS - HISTORY

Early signs of the disease are a failure to swing the arm when walking, deterioration in handwriting, and poor balance.

Later symptoms are a constant tremor, general body stiffness, loss of facial expression, a stiff way of walking and lack of coordination.

The intelligence and mental powers of victims are not affected in the early stages of the disease, and this causes great frustration, particularly when speech may be impaired. Patients may become depressed, anxious and emotionally disturbed.

COMMON SYMPTOMS

- Disturbed balance
- Bradykinesia (abnormally slow movement)
- Muscular rigidity
- Tremor at rest (Rhythmic sinusoidal movement of limbs and/or head at rest)
- Clumsiness
- Falls unexpectedly

LESS COMMON SYMPTOMS

- Vertigo
- Abnormal fatigue
- Dementia (advanced stages only)

OTHER SYMPTOMS

- Mood disorders
- Insomnia
- Retarded ejaculation
- Facial weakness
- Urinary incontinence
- Libido reduced
- Nocturia
- Excessive sweating

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RISK FACTORS

Age is the main risk factor. Although it can affect adults in their 20s, it ordinarily starts in middle or late life.

Men are more likely to develop Parkinson disease than women.

Both genetic and environmental factors have been theorised but are unproven.

Theoretical triggers include trauma, other illness, or exposure to an environmental toxin (eg. pesticide, herbicide). Farmers using these chemicals seem to have a higher incidence of the disease.

Reduced oestrogen levels may increase the risk of Parkinson disease and menopausal women who receive little or no hormone replacement therapy (HRT) and those who have had total hysterectomies may be at higher risk. Menopausal women using HRT appear to have a decreased risk.

Low levels of folate in mice triggers severe Parkinson-like symptoms.

SIGNS - EXAMINATION

COMMON SIGNS

Extrapyramidal gait (Slow, rigid gait, no arm swinging)

Festination (Quick, shuffling gait with trunk bent)

Hypertonicity (Involuntary resistance is encountered when limb is passively moved. Muscles are firm to touch)

Lead-Pipe Rigidity (Heavy passive stiffness of limb throughout range)

Parkinsonian Facies (Rigidity of facial muscles that gives a characteristic loss of facial expressiveness)

Cogwheel Rigidity (Resistance to passive movement diminishes in jerky steps)

LESS COMMON SIGNS

Tardive Dyskinesia (Impairment of voluntary movement causing incomplete or partial actions)

Oculogyric Crisis (Varies from mild cases with abnormal uncontrolled random eye movements, to severe cases with fixed elevated gaze associated with painful extension of the neck which may be so severe that the occiput nearly touches the thoracic vertebrae and the airway may be compromised)

INVESTIGATIONS - PATHOLOGY, RADIOLOGY ETC.

No blood or other test is diagnostic. The diagnosis is made on clinical grounds.

CT scans (special x-rays) may reveal changes in certain parts of the brain, as may electroencephalograms (EEG), which measure the electrical brain waves. Magnetic resonance imaging (MRI) and positron emission tomography (PET) scans are now being used in some centres.

Using Levodopa to treat Parkinson's disease and observing effect on patient is most useful diagnostic tool.

PARKINSON DISEASE

POSSIBLE CAUSES

Parkinson disease is a neurodegenerative disorder affecting dopaminergic neurones.

The cause is the presence of Lewy bodies which are tiny abnormal spheres found in nerve cells in the brain. They alter the function of the cell and can be seen when affected cells are examined under a microscope. There is a genetic contribution to Parkinson disease as a genetic mutation is responsible for the protein that forms Lewy bodies.

What happens in the brain to cause the symptoms is understood. When a muscle contracts, the opposite muscle must relax. For example, when you bend your finger, the muscles on the palm side of the finger contract, while those on the back of the finger must relax. This coordination occurs in the brain. In Parkinsonism, the brain cells that control this coordination have degenerated so that smooth control of movement is lost.

Parkinson disease tends to start in the enteric nervous system, olfactory bulb and medulla where it causes minimal symptoms (eg. sleep disorders, constipation, hyposmia). Only when it spreads to the cortex and substantia nigra do significant symptoms occur.

TREATMENT - MANAGEMENT

A number of drugs (eg. amantadine, levodopa, pergolide) are available to control the symptoms and possibly slow the progress of the disease, but it is a matter of trial and error to determine which medications will help any particular patient. None of them cure Parkinson disease - they aim to control it. Levodopa acts to replace the missing chemical in the brain that causes the disease. It is sometimes combined with other medications that increase the effectiveness of the levodopa and reduce side effects (eg. pergolide). Bromocriptine also acts to control Parkinson disease. It is, strangely enough, also used to stop the production of breast milk and treat a bone overgrowth disease called acromegaly. Side effects include a reduced tolerance to alcohol.

Other medications that may be used in treatment include pramipexole, ropinirole, selegiline and procyclidine.

Physiotherapy is also very important. In rare cases, brain surgery, in which part of the brain is destroyed in an attempt to block nerve pathways that cause the constant tremor, is performed.

In the future, stem cell implants may cure the condition. This has already been shown experimentally in mice. Electronic nerve stimulators placed in the brain and/or spine have also been used in selected patients.

Parkinson Disease Treatment summary

Ther: Selegiline OR levodopa/carbidopa OR levodopa/benserazide
AND bromocriptine OR pergolide OR bntropine OR trihexyphenidyl OR
amantadine
ADD benzhexol OR biperiden OR orphenadrine OR procyclidine OR tolcapone
ADD apomorphine IM for severe episodes
All patients:
Physiotherapy
Consider neurosurgery in selected patients. Experimental procedures include
pallidotomy, stimulators, stem cell transplants and neurotrophic factors
Prec: All drugs act synergistically. Use controlled release (CR) preparations when
possible.

PROGNOSIS

There is no cure, but medications allow some patients to lead normal lives. The disease process progresses steadily over many years, rarely causing death, but causing otherwise normal people to become invalids, totally dependent on others for everyday tasks.

ADDITIONAL INFORMATION

Differential diagnoses

Other conditions that may mimic Parkinson disease :-

- Carbon monoxide poisoning
- Heavy metal poisoning
- Cerebral tumours
- Cerebral degenerative disease
- Alcoholism
- Drugs (legal and illegal)
- Encephalitis
- Arteriosclerosis.

CURIOSITY

James Parkinson, after whom this condition is named, was not only a notable English physician, but was very multi-talented, as he was also recognised as a geologist, paleontologist, and political activist.

TOTALLY, COMPLETELY AND UTTERLY USELESS INFORMATION

The brain is 2% of the body's weight, but uses 20% of the body's energy.

Any student who would like a copy of a neurology textbook called "Rationale for the Brain - a guide to the diagnosis of diseases that may cause neurological symptoms", can download the book for free from:

http://www.medwords.com.au/MW_Rationales.html

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