INVITED ARTICLE

PARKINSON'S DISEASE

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ABSTRACT

The management of the elderly patient with idiopathic Parkinson's disease requires great attention to detail. Treatment should only be considered when the daily activities of life are affected. Medication should be commenced cautiously. Regular review is essential and co-ordination with other members of the multidisciplinary team is vital.

Keywords : Bradykinesia, depression, alimentary disturbances, postural instability, multidisciplinary approach

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Parkinsonism is a syndrome characterised by hypokinesia, tremor and rigidity. Many different disorders may produce this syndrome (Table I).

Table I
Causes of the Parkinsonian Syndrome

1) Idiopathic
2) Postencephalitic - Predominantly post-encephalitis lethargica
3) Arteriosclerotic brain disease
4) Drug-induced ie. dopamine receptor blocked by phenothiazines (especially prochlorperazine/stemetil) and butyrophenones
5) Toxins - Mercury, Carbon monoxide
6) Metabolic disturbances - Wilson's Disease
7) Degenerative condition involving basal ganglia - Progressive supranuclear Palsy, Shy-Drager dysautonomia
8) Guam Parkinsonism - Dementia - amyotrophic lateral sclerosis complex
9) Trauma

Idiopathic Parkinsonism is one of the commonest neurological conditions. The prevalence of Parkinson's disease is between 80 and 150 per 100,000 population[1].

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The prevalence is known to rise with age. Its peak presentation is in the seventh decade[2]. The disease is commoner in men than women.

The aetiology is multifactorial, many theories have been considered (Table II). There are some histological certainties. Depigmentation occurs in the substantia nigra with neuronal loss out of proportion to the age of the subject. Lewy bodies, present in the substantia nigra, are increasingly recognised as positive indicators of disease. Changes also occur in the globus pallidus, putamen and caudate nucleus. There appears to be an overlap in pathology between idiopathic Parkinsonism and senile dementia of Alzheimer's type.

Table II
Suggested Aetiology of Idiopathic Parkinsonism

a) Related to primary deficiency in hypothalamic cells producing MSH
b) Herpes simplex virus implicated
c) Accumulation of heavy metals
d) Affect of neurotoxins - effect of methylphenyltetrahydropyridine (MPTP)

NEUROCHEMICAL CONSIDERATIONS

Deficiency of dopamine in the substantia nigra was first demonstrated in the 1960's[3]. Idiopathic Parkinsonism is the result of the subsequent imbalance of dopamine and acetyl choline. It was initially thought that redressing the balance would restore normal function. Treatment was aimed at either increasing the dopamine or decreasing the acetyl choline. However, progress was not straightforward. Simply replacing dopamine did not prevent the progress of the disease; some features of the disease did not improve despite treatment and after a period of time treatment was associated with undesirable effects ie. dyskinesias.

Other transmitters are thought to be implicated. Changes in concentration of Gammaminobuytric acid (GABA), serotonin and noradrenaline have been documented. Research into the significance of these neurotransmitter deficiencies continues.

CLINICAL ASPECTS

The well known characteristic triad of idiopathic Parkinsonism is tremor, rigidity and akinesia.

Tremor is the presenting complaint in approximately 70% of cases. The typical resting tremor is not constant, affects distal muscles and is usually initially unilateral, involving the upper limb and spreading to the ipsilateral lower limb. It is exaggerated during stress, excitement or fatigue.

There is also a co-existing action tremor which replaces the resting tremor during movement. The action tremor is faster

and gives rise to the phenomenon of cogwheeling on passive contraction of agonist and antagonist muscles.

The tremor of idiopathic Parkinsonism has to be differentiated from senile tremor, metabolic tremor, cerebellar disorders, basal ganglia disorders and tardive dyskinesia.

Limb rigidity in idiopathic Parkinsonism is plastic - it occurs during passive stretching of flexors and extensors. It may occur unilaterally causing confusion with a cerebrovascular accident and it may occur centrally only affecting the muscles of the neck causing confusion with musculoskeletal conditions. Other conditions give rise to similar rigidity so close observation for additional differentiating features is required. Elderly with locomotor difficulties attributed to diffuse cerebral disease may demonstrate rigidity. The rigidity in patients with diffuse cerebral arteriosclerosis is usually more marked than the rigidity of idiopathic Parkinsonism.

Akinesia or bradykinesia is probably the most disabling component. The poverty of automatic movement and the slowness in initiating voluntary movement compromise all aspects of normal activity. The bradykinesia results in the mask-like facies, the micrographia, the monotonous speech and the loss of arm swing.

In addition to the traditional description other features of the disease are noteworthy.

Patients with idiopathic Parkinsonism have a higher risk of developing dementia than age matched controls[4]. The risk applies mainly to those that develop the disease after the age of 60. Forgetfulness, repetitiveness and reduced creativity are thought to be the results of diffuse lewy body development not only in the basal ganglia but also in the neocortex.

Change in serotinin levels has been suggested as the cause of depressed mood commonly found in affected individuals. Patients appear particularly vulnerable to depression at times of rapid deterioration in motor symptoms. Suspicion of dementia coexisting with Parkinsonism requires careful evaluation to ensure that pseudodementia is not ignored. Anti-depressants have numerous side effects (cardiac arryhthmias, constipation, postural hypotension). ECT may be helpful. Support groups are beneficial.

Psychotic behaviour in an individual with known Parkinson's disease may be related to drug withdrawal. Reduction of anticholinergics or dopamine should be a gradual process.

The alimentary disturbances experienced by an affected individual are often ignored. Drooling of saliva can be very embarrassing for the patient. It is a potential cause of dehydration. It usually indicates advanced disease with severe akinesia. It is not caused by hypersecretion.

Dysphagia may result from delay in initiation of swallowing with irregular epiglottal movements and predisposes the individual to recurrent aspiration pneumonias. Food and drink should only be consumed with the patient in the upright position. Constipation is common. The sedentary lifestyle, loss of salivary fluid, diminished fluid and food intake and anti-cholinergic medication are all predisposing factors. The constipation may be so severe that the patient may develop obstruction.

Micturition problems should be enquired about. Patients may experience difficulty negotiating the physical environment and adjusting clothing. They may complain of frequency of micturition or may be found to have urinary retention.

Postural disorders are common. The patient may suffer from physical instability. Standing unaided may be difficult. Falls may be frequent due to lack of defensive righting reactions. The latter may either be due to postural reflex changes or to the hypokinesia.

DIAGNOSTIC DIFFICULTIES

Clinical presentation may be difficult to interpret as so many features could be mistaken for ageing changes - the individual with Parkinson's disease is almost a caricature of the ageing process.

Certain presentations may cause diagnostic difficulties (Table III).

Table III

Differential Diagnosis

Tremor	-	Senile tremor, tremor of metabolic diseases, cerebellar disorders, basal ganglia disorders, tardive dyskinesia	
Rigidity	-	Unilateral	- Cerebrovascular accident
		Diffuse	- Generalised cerebral arteriosclerosis
		Neck/Shoulders	- Osteoarthritis
Bradykinesia	-	Hypothyroidism, dementia, depression	
Dysphagia	-	Carcinoma oesophagus	

TREATMENT

Once the diagnosis of idiopathic Parkinsonism is made, full assessment and documentation is required. Response to therapy can only be monitored by accurate documentation. Numerous parameters require consideration. Facial expression, posture, speed of walking, step length, tremor, speech, writing, dexterity and ability to perform activities of daily living such as dressing, eating, washing, bathing, etc. need to be ascertained at each visit.

Accurate documentation and advice to the patient to keep a daily diary may be of benefit when the treatment regime is under review.

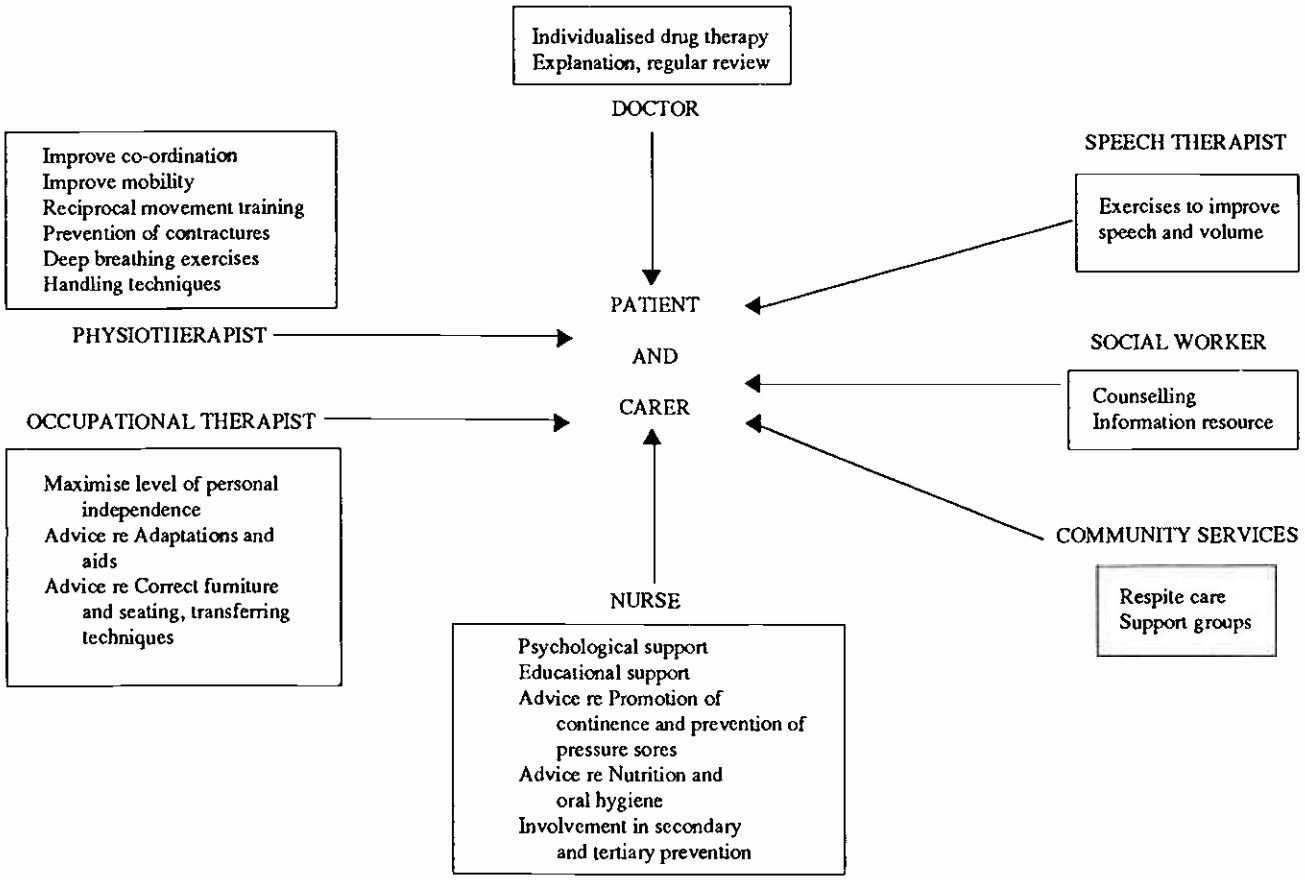
Bowel and bladder problems should improve with the introduction of anti-Parkinsonian therapy. If symptoms persist despite good control of akinesia and rigidity then further investigation might be warranted.

When contemplating treatment, drugs should not be seen as the only available form of therapy. Physical therapy and psychotherapy are also important options that need consideration (Fig 1).

Anti-cholinergics were advocated as the initial line of management, however they may be problematic when prescribed for the elderly. In most patients their effects are only moderate, reducing tremor and rigidity to some effect but with little action on bradykinesia. They may help with sialorrhoea. Their side-effects may prohibit their usefulness. Co-existing urinary retention, cardiovascular disease, hepatic or renal impairment may prevent usage. Gastro-intestinal disturbances, blurred vision, tachycardia, nervousness, mental confusion and psychiatric disturbances may develop and necessitate discontinuation of treatment.

Levadopa administered in conjunction with an extra-cerebral dopa-decarboxylase inhibitor (carbidopa or benzerazide) is the mainstay of treatment, improving bradykinesia and rigidity more than tremor. Peripheral side-effects are reduced because of the presence of the inhibitor. Nausea, agitation, postural hypotension, tachycardias, abnormal voluntary movements and psychiatric symptoms are the more common problems. Treatment should be initiated with low doses and gradually increased by small increments. Titration is required, the final dose being a compromise between increased mobility and dose-limiting side-effects. Intervals between doses may be critical and should be chosen to suit the needs of the individual patient.

Fig 1
Management Guidelines



In the elderly patients, it is suggested that the initial dose should be 62.5 (Levadopa 50mg, benserazide 12.5mg) once daily. This should be increased to twice daily and subsequently three times daily at three day intervals. Further increases should be considered on an individual basis balancing functional improvement against risk of side effect.

The most frequent dose-limiting side effects of levadopa are involuntary movements and psychiatric complications. Treatment should not be discontinued abruptly. As the patient ages the maintenance dose may need to be reduced.

As the distress progresses, the individual may be troubled by various "on-off" effects. These are characterised by fluctuations in performance with normal performance during the 'on' period and weakness and akinesia during the 'off' period.

Selegiline is a monoamino-oxidase-B inhibitor recently introduced which may be of benefit in patients developing end of dose akinesia. Before initiating Selegiline therapy the levadopa dosage needs to be reduced by approximately 20%. Selegiline should be commenced as a 5mg dose in the morning. This dose can be increased to 10mg if clinically indicated. Side-effects are similar to levadopa.

Bromocriptine acts by direct stimulation of surviving dopamine receptors. Its use is often restricted by side effects particularly hypotension. Abnormal involuntary movements and confusional states are common and the dosage of levadopa and bromocriptine should be balanced for optimal effect. Elderly patients should not receive more than 1mg daily initially. The dose should be increased at weekly intervals to a maximum of 5mg total daily dose.

Amantadine may help with akinesia, rigidity and tremor. Its effect is usually only temporary and it should be used with caution in the presence of cardiovascular, hepatic or renal disease. It may cause insomnia, dry mouth, peripheral oedema and toxic confusional states.

Non-drug therapy consists of assessment and treatment by physiotherapist, occupational therapist and occasionally speech therapist. Advice will be given with regards appropriate exercises that can be performed at home. Exercises can aid walking style. Occupational therapy would concentrate on maximising independence in activities of daily living and giving advice regarding adjustments at home to improve the quality of life for patient and carer.

Self help groups may be beneficial to the whole family. Understanding the disease helps sufferers cope with its effects. Carers have an opportunity to share their frustrations.

The management of Parkinson's disease requires an understanding of the disease process, the pharmacology of the available drugs, in particular, a knowledge of the different dyskinesias associated with levadopa therapy and an awareness that mental and social needs also require attention.

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