

# **Parkinson's disease** and **parkinsonism** in **the elderly**

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# Contents

	<i>List of contributors</i>	<i>page</i> vii
	<i>Foreword</i>	ix
<b>1</b>	<b>A glossary of terms</b>	1
	Jolyon Meara	
<b>2</b>	<b>Diagnosis of parkinsonism in the elderly</b>	4
	Robert L. Rodnitzky	
<b>3</b>	<b>Parkinson's disease and parkinsonism in the elderly</b>	22
	Jolyon Meara and Bimal K. Bhowmick	
<b>4</b>	<b>Drug-induced parkinsonism in the elderly</b>	64
	Jean P. Hubble	
<b>5</b>	<b>Essential tremor in the elderly</b>	80
	Rajesh Pahwa and William C. Koller	
<b>6</b>	<b>Gait apraxia and multi-infarct states</b>	98
	Richard Liston and Raymond C. Tallis	
<b>7</b>	<b>The epidemiology of Parkinson's disease and parkinsonism in elderly subjects</b>	111
	Jolyon Meara and Peter Hobson	
<b>8</b>	<b>Health and social needs of people with Parkinson's disease and the worldwide organization of their care</b>	122
	Peter Hobson	
<b>9</b>	<b>The drug treatment of Parkinson's disease in elderly people</b>	134
	Theresa A. Zesiewicz and Robert A. Hauser	

<b>10</b>	<b>Rehabilitation in Parkinson's disease and parkinsonism</b>	165
	Christopher D. Ward	
<b>11</b>	<b>Rehabilitation, nursing and elderly patients with Parkinson's disease</b>	185
	Sally Roberts	
<b>12</b>	<b>Rehabilitation, physiotherapy and elderly patients with Parkinson's disease</b>	198
	Hilary Chatterton and Brenda Lövgreen	
<b>13</b>	<b>Rehabilitation, occupational therapy and elderly patients with Parkinson's disease</b>	217
	Jackie Hughes	
<b>14</b>	<b>Rehabilitation, speech and language therapy and elderly patients with Parkinson's disease</b>	226
	Sheena Round	
	<i>Index</i>	240

## Diagnosis of parkinsonism in the elderly

Robert L Rodnitzky

The diagnosis of parkinsonism depends on recognizing its component clinical features. Parkinsonism includes Parkinson's disease (PD) and all the varied conditions with clinical features resembling those of PD. To identify patients with parkinsonism correctly it is important to be able to recognize the cardinal clinical features of PD, namely akinesia, lead-pipe rigidity, rest tremor, and postural instability. The next critically important step is to determine whether they suggest PD or one of the other non-PD causes of parkinsonism (see Table 2.1). This latter distinction will enable effective treatment strategies to be devised and a meaningful discussion of prognosis and genetic implications to be undertaken. The entire process of identifying parkinsonism and assigning a specific clinical diagnosis is particularly challenging in the elderly because many of the motor changes associated with normal ageing resemble parkinsonism. Additionally, several medical conditions that are common in this age group can result in parkinsonism that may incorrectly be considered evidence of PD.

### The clinical signs of PD

Of the cardinal motor signs of PD, akinesia is perhaps the most disabling. Slowness, difficulty in initiation, and a reduction in the amount or amplitude of voluntary movement (Rodnitzky and Uc 1997) characterize akinesia (see Table 2.2). A great variety of clinically recognizable signs result from akinesia. A lack of facial expression attended by reduced blink rate is one of the most apparent manifestations of akinesia. Additional findings are diminished arm swing on one or both sides of the body, difficulty arising from a chair, a slow, short stepped gait, en bloc turning, and soft, poorly articulated speech (hypophonia). The clinical signs of akinesia are so striking that their presence alone has been considered by some to be sufficient to establish a diagnosis of PD (Quinn 1995). The more usual view is that additional motor findings are necessary to establish a diagnosis of PD. Rigidity is another common finding in patients with PD (see Table 2.3). Rigidity is felt by an examiner as an increased resistance to passive movement of joints in the fully relaxed limb.

**Table 2.1** Stages in the diagnosis of parkinsonism**Stage 1**

- Is there clinical evidence of parkinsonism?
- Akinesia must be present for this diagnosis.

**Stage 2**

- What type of parkinsonism is present?

**Table 2.2** Clinical features of PD**Akinesia**

- Reduction in spontaneous voluntary motor activity, slow movements, difficulty with sequential and concurrent motor acts, abnormal early fatigability and reduction in amplitude of movements

**Table 2.3** Clinical features of PD**Lead-pipe rigidity**

- Abnormal resistance which remains constant throughout the range of movement and is felt when passively stretching muscles around a joint in a relaxed subject

**Cog-wheel rigidity**

- A ratchet type of fluctuating resistance felt at the wrist in synchrony with tremor bursts

Resistance is unchanged throughout the range of movement of the joint and can be distinguished from spasticity, in which resistance is greatest at the onset of passive movement and then suddenly gives way (clasp knife phenomenon). Often, a ratchet like quality (cog-wheeling) is present as the joint is moved especially when tremor is present. Subtle rigidity can be enhanced by utilizing reinforcement techniques such as instructing the patient to execute repetitive forceful movements in the contralateral limb. True rigidity must be distinguished from gegenhalten in which patients with diffuse encephalopathy or frontal lobe dysfunction exert a force opposite in direction to the examiner's attempted passive movement.

Rest tremor is one of the most easily recognizable signs of PD (see Table 2.4). It usually appears at a frequency of 3–6 Hz when the limb is fully supported and motionless. It also appears in the hands when the arms are suspended at the sides during walking. Typically, the tremor is reduced or totally disappears during action. In PD, tremor is often unilateral at the onset of the illness and remains asymmetrical even though ultimately spreading to the contralateral limbs. The presence or absence of rest tremor is a major consideration when attempting to determine

**Table 2.4** Clinical features of PD**Rest tremor**

- Classically a 4–5Hz oscillation involving the distal portion of the upper limb of ‘pill rolling’ type. Atypical rest tremor can occur and rest tremor is often accompanied by postural and kinetic tremors. The head and trunk are usually spared

whether a patient has PD or another form of parkinsonism. Rest tremor is present in the great majority of patients with PD, but in only a smaller percentage of those with other forms of parkinsonism. The distribution of tremor is also important in helping to establish a diagnosis of PD. The tremor of PD commonly begins in the hands and is slightly less common in the lower extremities and mandible. It almost never affects the head or the muscles of articulation. When present, a ‘pill rolling’ tremor at rest involving the thumb and index finger very strongly suggests a diagnosis of PD or drug-induced parkinsonism. Postural instability has a great number of potential causes other than PD, particularly in the elderly. Patients manifesting this dysfunction are at increased risk of falling since they are unable to generate normal reflex movement to counter even the slightest perturbation to their posture. The clinician can safely demonstrate an absence of postural reflexes by standing behind the patient and applying a brisk backward directed push on the sternum.

Episodes of freezing, also referred to as ‘motor blocks’, most commonly involve gait. The patient’s feet appear ‘glued’ to the floor when attempting to initiate gait, during turns, or when approaching a real or imagined obstacle such as a narrow passageway or an entrance to a room. Whether this phenomenon represents a severe form of akinesia or is physiologically separate is not known. It is common in late stage PD, but in some other forms of parkinsonism it can be an early, or even a presenting clinical sign (Giladi et al. 1991). Several guidelines have been suggested for utilizing clinical signs to establish a diagnosis of clinically probable PD. Definite diagnosis strictly requires postmortem confirmation of PD. Most guidelines require a certain number of the cardinal motor signs of parkinsonism to be present to make a diagnosis of PD in life. For example, the UK Parkinson’s Disease Society Brain Bank criteria (Hughes et al. 1992a) requires the presence of akinesia plus *one* other clinical sign from among rigidity, rest tremor, and postural instability. Koller (1995), on the other hand, suggested that any two of three motor findings from among rigidity, akinesia, and tremor is sufficient to establish a clinical diagnosis of PD. While these criteria increase diagnostic accuracy, they are far from infallible. Several studies have suggested a high level of diagnostic inaccuracy compared to postmortem findings, even when the clinical diagnosis of PD is made by experienced neurologists (Rajput et al. 1991a, Hughes et al. 1992a, de Rijk et al. 1997).

Rajput et al. (1991a) found that only 76% of patients with a final clinical diagnosis of PD during life had evidence of the disease when examined at autopsy. Hughes et al. (1992a) examined 100 brains of patients with a final clinical diagnosis of PD and could confirm such a diagnosis in only 76% of cases. The diagnosis in the remainder included conditions such as progressive supranuclear palsy (PSP), multiple system atrophy (MSA), and Alzheimer's disease (AD). The clinicians in this study had utilized clinical criteria of their choice in arriving at a diagnosis of PD. When systematized diagnostic clinical criteria were retrospectively applied to cases in this study diagnostic accuracy improved to 82%. In clinical practice diagnostic accuracy is likely to be much lower than these figures suggest since these studies only looked at final diagnosis. However, all patients in these studies died at least 10 years ago and in the interim diagnostic awareness may have improved as is suggested by the most recent clinicopathological series (Ansoerge et al. 1997). When applying additional computer generated criteria with a high predictive value for diagnostic accuracy (asymmetrical onset, no atypical features and no other possible etiology), the diagnostic accuracy was further increased to 93%, but 32% of pathologically confirmed cases were rejected on this basis. A further study compared eight different sets of diagnostic criteria that might be applied to prevalence studies of PD and found some sets too inclusive and others too restrictive (de Rijk et al. 1997). These authors concluded that the most reasonable inclusion criteria for PD was two of the three cardinal features (tremor, akinesia and rigidity) in the absence of other apparent causes of parkinsonism. It is clear from these studies that PD can be distinguished from other forms of parkinsonism on clinical grounds alone with high, but not total, accuracy. Using the most stringent diagnostic criteria reduces misdiagnosis, but at the expense of misclassifying a significant number of true cases of PD.

Controversy still exists over the condition of 'tremor dominant' PD. In this situation tremor is an isolated finding and this can easily lead to misdiagnosis with the most common cause of isolated tremor, essential tremor. Rest tremor, accompanying the more usual postural and kinetic tremor, can be a late manifestation of essential tremor in elderly subjects (Rajput et al. 1993). Tremor dominant cases of presumed PD could usefully be assessed with an apomorphine challenge test to help distinguish PD from other causes of isolated tremor.

### **Atypical features suggesting diagnoses other than PD**

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Despite the caveat that diagnostic criteria can be made so specific as to reduce their clinical utility, the usefulness of incorporating a careful search for atypical features as an exclusionary criterion for PD deserves special mention. Atypical features not only alert the clinician to the possibility that the diagnosis may not be PD, but in a



positive sense may suggest another form of parkinsonism and lead the clinician to the correct diagnosis. In this regard it is important to understand which facets of the natural history or clinical examination are to be considered highly atypical for PD as well as which of them singly or in combination strongly suggest another clinical diagnosis within the spectrum of parkinsonism.

There is a reasonably well defined body of clinical signs and symptoms that are distinctly unusual in PD. When one or more of these signs appear in the akinetic-rigid or tremulous patient, they should prompt the clinician to question the diagnosis of PD. The following discussion describes the most important of these atypical signs and indicates which alternative diagnoses each one suggests. Several of these atypical features are much more likely to occur in elderly subjects, making their identification and proper interpretation even more important in this age group.

### **Early dementia**

Dementia is common in PD, having been found in as many as 65% of patients by the age of 85 years (Mayeux et al. 1990). However, the dementia of PD seldom appears at the onset of the illness. Early dementia in the akinetic-rigid patient should prompt consideration of a variety of other syndromes with parkinsonian features, including dementia with Lewy bodies (DLB), PSP, corticobasal ganglionic degeneration (CBGD), normal pressure hydrocephalus, Creutzfeldt-Jacob disease, or AD. The confusion with AD arises from the fact that parkinsonism can appear in some patients with AD (Hughes et al. 1992a, Hulette et al. 1995). These signs are usually thought to occur late in the illness and are most likely to be seen in the elderly patient with severe cognitive impairment (Lopez et al. 1997). However, in the study of Hughes et al. (1992a) AD was a common cause of diagnostic error in PD and it appears from this study that AD, particularly when AD pathology involves the corpus striatum, can present with parkinsonism sufficient to lead to diagnostic confusion with PD. The development of mild parkinsonian features one or more years after the onset of otherwise clinically typical AD, especially in an elderly patient, should be considered to be a case of AD with parkinsonism, rather than PD with dementia. In the elderly patient, the possibility of the concurrent appearance of both PD and AD should also be given consideration, taking into account the high prevalence of both of these conditions in this age group. In this circumstance, the presence of a rest tremor or a significant improvement in parkinsonism after treatment with levodopa lends some support to a diagnosis of PD, rather than parkinsonism due to AD alone.

Certain clinical characteristics of the dementia may also be atypical for PD. Marked fluctuations in cognitive impairment consisting of periods of confusion alternating with lucidity, for example, should suggest DLB (Byrne et al. 1989, Mega

et al. 1996). The differentiating characteristics of the dementia associated with AD and those found in PD have been well studied. The dementia of AD is more likely to be associated with abnormalities of memory and language and the presence of anosognosia while that of PD is more commonly characterized by impairment of visuospatial and executive functions (Mohr et al. 1990, Starkstein et al. 1996, Mahieux et al. 1998).

Early hallucinations have implications similar to those of early dementia. Hallucinations occurring in PD typically appear late in the course of the illness and are almost always associated with the chronic use of antiparkinsonian drugs. Hallucinations occurring prior to the initiation of such drugs or with the first administration of these agents strongly suggests another diagnosis, particularly DLB. This phenomenon points out the importance of accurately distinguishing between PD and DLB. Compared to PD, DLB patients are much more likely to suffer behavioural side effects from dopaminergic or anticholinergic agents.

### **Early falls and postural instability**

Falling in PD is typically the result of impaired postural reflexes, postural hypotension or severe large amplitude dyskinesias. Severe freezing with inability to check forward propulsion of the upper trunk is another possible cause. These causes of falling typically appear in late stage PD and seem to occur earlier with late onset PD. However, a variety of other conditions with parkinsonian features may present with early falling.

The condition among those most likely to present with falling is PSP (Jankovic et al. 1990). In these patients, the gait abnormality is quite different from that seen in PD. In PSP patients there is akinesia associated with axial rigidity and nuchal dystonia, often in extension, vertical supranuclear gaze palsy and impaired postural reflexes. This combination results in frequent and early falling. The early appearance of gait freezing in PSP, which sometimes antedates the other motor signs of this condition, is also a major contributing factor to the occurrence of early falling. Postural instability is much more common early in the course of MSA than in PD. These patients may have marked akinesia with a loss of postural reflexes, sometimes associated with truncal dystonia. Wenning et al. (1997) in a review of 203 pathologically proven cases of MSA found that 38% had presented with ataxia. The term 'lower body parkinsonism' has been used to describe a severe isolated gait disorder associated with diffuse cerebral vascular disease (Fitzgerald and Jankovic 1989). This form of vascular parkinsonism is characterized by isolated involvement of the lower extremities and severe freezing of gait, often leading to falls. Normal pressure hydrocephalus can present with an early and predominant gait disorder associated with frequent falls. In this condition the gait is characterized by inability to lift the feet from the floor, short shuffling steps, imbalance while walking,

difficulty turning, and gait ignition failure (Marsden and Thompson 1997, Graff-Radford and Godersky 1997, and see Chapter 6).

### **Severe autonomic dysfunction**

Severe autonomic dysfunction early in the course of the illness is not typical of PD. Late in the course of PD patients may develop mild to moderate symptoms of autonomic insufficiency such as constipation, urinary incontinence, orthostatic hypotension, impotence, or impaired lacrimation (Goetz et al. 1986, Beattie et al. 1993). Anticholinergic drugs used to treat PD may contribute to the appearance of constipation or bladder dysfunction, while dopaminergic agents can cause or exacerbate hypotension and, to a lesser extent, constipation. The possibility of MSA should be considered in patients with evidence of early autonomic dysfunction in the absence of other diseases and drug treatments known to effect the autonomic system (Magalhaes 1995). In MSA autonomic dysfunction can predate signs of parkinsonism by several years. In one study of MSA autonomic signs antedated motor symptoms by one to two years in a quarter of cases (Wenning et al. 1994). Two techniques, electromyography of the urethral sphincter (Pramstellar et al. 1995) and formal urodynamic studies (Bonnett et al. 1997) are available to objectively distinguish the autonomic dysfunction of MSA from that of PD. The urethral sphincter is invariably denervated in MSA patients with incontinence, but not in PD patients with similar symptoms. In PD, urodynamic studies reveal an urgency to void without chronic retention, associated with detrusor hyperreflexia and normal urethral sphincter function. In MSA there is often chronic urinary retention, a hypoactive detrusor muscle and lower urethral pressures.

### **Poor or transient benefit from drug treatment**

Dopaminergic drugs, especially levodopa, usually improve the signs of PD. The vast majority of PD patients benefit from levodopa therapy. In one series of pathologically proven cases of PD, 94% had responded to levodopa during life (Rajput et al. 1990). The response rate to levodopa in other causes of parkinsonism is much lower. As many as 65% of MSA patients have been reported to respond, at least initially, to levodopa (Hughes et al. 1992b), although in most studies the response is closer to one third (Rajput et al. 1990). Even among those with an initial response, fewer than 5% may continue to benefit in the advanced stages of the illness (Wenning et al. 1997). In PSP, a levodopa response rate of 38% has been reported (Nieforth and Golbe 1993). Patients with multisystem degenerative disease who are initially responsive to levodopa commonly experience a rapid disappearance of benefit within one to two years. In CBGD early benefit from dopaminergic agents is much less common than that seen in PSP or MSA. In other causes of parkinsonism such as vascular parkinsonism and normal pressure hydrocephalus, lack of

significant response to levodopa is the rule, but rare exceptions have been reported (Mark et al. 1995). On balance, these observations suggest that total refractoriness to therapeutic doses of levodopa therapy, in the absence of malabsorption, is a strong point against the diagnosis of PD. On the other hand, responsiveness to levodopa at the onset of illness can be considered a mild point in favour of a diagnosis of PD, but does not reliably distinguish between PD and several other forms of parkinsonism.

### **Striking asymmetry of motor signs**

Marked asymmetry is unusual in PD, although mild asymmetry is quite common. When mild to moderate asymmetry does exist in PD, it tends to become less apparent as the illness advances. Profound tremor or rigidity on one side of the body with minimal or no symptoms on the contralateral side suggests a variety of diagnoses other than PD. Marked unilateral limb rigidity is often seen in CBGD (Schneider et al. 1997). A severe and strictly unilateral rest tremor raises the question of structural pathology such as an infarction involving the contralateral cerebellar outflow pathways. This anatomic localization is further suggested when the tremor is worse upon assuming a posture and during action than it is at rest. The syndrome of hemiparkinsonism hemiatrophy results in unilateral levodopa-responsive parkinsonism associated with ipsilateral body atrophy and contralateral brain atrophy, presumably related to brain injury early in life (Giladi et al. 1990). This condition should not be a major source of diagnostic confusion in the elderly since it typically appears before the age of 50 years old. Unilateral parkinsonism of acute onset or parkinsonism associated with pyramidal tract signs should suggest the possibility of an isolated infarction involving the contralateral brain stem or basal ganglia (de la Fuente Fernandez 1995).

### **Absence of rest tremor**

The failure to demonstrate rest tremor does not exclude the diagnosis of PD, but rest tremor is more common in PD than in most other forms of parkinsonism. In two series of pathologically confirmed cases of PD, rest tremor was found to have occurred in 76% (Hughes et al. 1992c) and 100% (Rajput et al. 1991b) of patients during life. However, the incidence of rest tremor in parkinsonism not due to PD in these studies was between 31% and 50%. In autopsy proven cases of MSA only 39% had rest tremor (Wenning et al. 1997), while in 11 autopsy confirmed cases of CBGD only 18 had resting tremor during life (Schneider et al. 1997). It is important to mention that a tremor of the head should not be considered a rest tremor. More commonly, a head tremor is a postural tremor, reflecting the continued activity of axial postural muscles when in the upright position, or a dystonic tremor. In the former instance, a diagnosis of essential tremor should be considered and in the

latter instance, cervical dystonia is the likely cause of tremor. In those patients suspected of having PD, but who do not manifest rest tremor, it is useful to engage the patient in mildly stressful activities, such as difficult mental arithmetic, in order to uncover a latent rest tremor.

### **Other features less common in PD**

A variety of other findings cast doubt on a presumptive diagnosis of PD. The presence of more than one similarly affected first degree relative, while not impossible in PD, is unusual. Families with autosomal dominant parkinsonism have been reported but are exceedingly rare (Golbe et al. 1996). Rather, conditions with a known genetic basis for familial occurrence should be considered, such as Wilson's disease, Machado–Joseph disease and adult onset Hallervorden–Spatz disease. In a patient with tremor dominant disease (see above) a positive history of familial tremor may suggest the diagnosis of essential tremor (see Chapter 5). Other than essential tremor, these heritable causes of parkinsonism are unlikely to present in the elderly. Patients with Huntington's disease, an autosomal dominant condition, can manifest signs of parkinsonism at any age, although isolated parkinsonism (the Westphal variant) typically appears as a juvenile form prior to the age of 25 years old. Recent neuroleptic drug therapy precludes a definite diagnosis of PD. Although drug-induced parkinsonism typically remits within a matter of weeks after withdrawal of the offending agent, it can sometimes persist for up to one year (see Chapter 4). A history of oculogyric crisis suggests the possibility of post encephalitic parkinsonism rather than PD.

There are several other clinical signs that may appear in a patient with parkinsonism which are not only atypical for PD, but by themselves strongly suggest another specific diagnosis. These signs, and the conditions they suggest, include supranuclear gaze palsy or eyelid apraxia (PSP), ataxia or other signs of cerebellar dysfunction (MSA), prominent myoclonus (Creutzfeldt–Jakob disease), alien limb phenomenon, or limb apraxia (CBGD) and a marked response to anticholinergic therapy (drug-induced parkinsonism).

### **Conditions commonly mimicking PD in the elderly**

Certain conditions mimic PD so commonly in the elderly that their differentiating features deserve special mention. These conditions have several overlapping clinical features with PD, but for most of them, the relative severity of these signs and their time of appearance in the course of the illness differ from that seen in PD. More importantly the nature of associated neurological signs and symptoms in those conditions usually allow the correct diagnosis to be made.

### Vascular parkinsonism

Parkinsonian symptoms can occur as a result of a number of different vascular pathologies. Riley and Lang (1996) identified four categories of cerebral vascular disease that have this potential: multi-infarct disease, *état crible* (multiple dilated perivascular spaces), Binswanger's disease (subcortical arteriosclerotic encephalopathy) and single focal infarctions or haemorrhage. These pathologic states become increasingly common with advancing age. It is important to distinguish them from PD so that appropriate therapy can be considered. For vascular parkinsonism, risk factors such as hypertension, hyperlipidemia, and coagulation abnormalities must be identified and reversed, whereas in true PD, specific antiparkinsonian drug therapy will be indicated. A variety of clinical signs and several facets of the clinical course help distinguish vascular parkinsonism from PD. In vascular parkinsonism, a subacute or acute onset of symptoms is sometimes seen, and similarly, the progression of the illness may occur in a stepwise fashion (Hurtig 1993). Rest tremor is rare (Inzelberg et al. 1994) and is almost never the predominant motor sign as can be the case in PD. Pyramidal tract findings such as hyperreflexia and Babinski signs are common in vascular parkinsonism, as are pathological laughing and crying. Rigidity, when present in vascular parkinsonism, is much more likely to be clasp knife in nature secondary to spasticity, rather than the typical lead-pipe variety associated with PD. In patients with widespread bilateral vascular disease, especially that involving the frontal lobes, paratonic rigidity (*gegenhalten*) may be present and can be distinguished from the rigidity of PD. Many patients with vascular parkinsonism present with a characteristic isolated impairment of ambulation consisting of a hesitant, shuffling gait with a preserved arm swing (Elbe et al. 1996). These patients have extremely poor postural stability and frequent falls are much more common than in PD (Trenkwalder et al. 1995). Parkinsonian symptoms such as hypophonia or facial masking are seldom present (Thompson and Marsden 1987). Patients with vascular parkinsonism demonstrating this predominant involvement of gait and balance with less involvement of the arms and face, have been described as manifesting the syndrome of 'lower body parkinsonism' (Quinn 1995). One of the most important differentiating features between PD and vascular parkinsonism is that there is seldom a significant clinical response to levodopa in patients with the vascular syndrome. Occasional striking, though rare, exceptions to this observation have been reported (Mark et al. 1995). As will be discussed below, brain imaging may help confirm the diagnosis of parkinsonism related to isolated cerebral infarctions or widespread subcortical vascular insults.

### Normal pressure hydrocephalus

This condition typically presents with the clinical triad of dementia, urinary incontinence, and a gait disorder (Graff-Radford and Godersky 1997). It can occur at any

age, but in adults the majority of cases present after the age of 60 years (Krauss et al. 1997). Although it is usually the gait disorder of normal pressure hydrocephalus (NPH) that results in diagnostic confusion with PD, a variety of other parkinsonian features can also be seen in this condition. In a study of 90 adults with NPH, Krauss et al. 1997 found that 81% had some evidence of akinesia. In the same study, rest tremor and rigidity were relatively rare, each occurring in only 14% of patients. The gait disorder of NPH is commonly referred to as an apraxia of gait, although this label is physiologically incorrect, given the underlying motor abnormality and akinesia seen in this condition. Typically, patients with NPH walk with slow short steps and demonstrate impaired ability to lift their feet off the walking surface (Sudarsky and Simon 1987). Like vascular parkinsonism, the gait in these patients is often impaired out of proportion to upper extremity and facial dysfunction. Aside from this unusual distribution of symptoms, differentiation from PD is aided by a history of antecedent events known to predispose to NPH (meningitis, subarachnoid haemorrhage, serious cranial trauma) and the early appearance of both dementia and bladder dysfunction. Whereas improvement with levodopa therapy is mild and infrequent, removal of 40–50 ml of CSF by lumbar puncture may result in a marked, albeit transient, improvement of symptoms.

The diagnosis of NPH should not be seriously considered unless ventriculomegaly in the absence of cortical atrophy is demonstrated by brain imaging. In properly selected patients, the neurological abnormalities associated with NPH, including the gait disorder, the generalized akinesia and cognitive impairment, can be improved by placement of a cerebrospinal fluid shunt (Black et al. 1985).

### **Essential tremor**

This condition is characterized by a postural tremor that worsens with action (see Chapter 5). It appears predominantly in the arms and to a lesser extent in the lower extremities. The head is commonly involved and there may be an associated voice tremor. Essential tremor (ET) is a common disorder with a prevalence in the general population estimated to be as high as 1.7% (Larsson and Sjogren 1960). More importantly, it is much more common with advancing age. Prevalence rates have been reported to be as high as 13% in individuals between the ages of 70 and 79 years old (Rautakorpi et al. 1982). In individuals experiencing the onset earlier in life, tremor amplitude becomes progressively greater over time. Accordingly, the tremor itself becomes much more apparent in the later years of life.

Because this condition is so common in the elderly, and because it presents with tremor, ET is frequently misdiagnosed as PD. Several clinical features help distinguish it from PD. Perhaps the most important differentiating feature is the nature of the predominant tremor in the two conditions. The tremor of ET appears predominantly when the involved body part is maintained in a fixed posture. It

persists or may be accentuated during movement. The classical tremor of PD occurs at rest. In both conditions, however, the opposite form of tremor may appear concurrently, but is almost never the predominant tremor type. Another distinguishing feature is that in ET, tremor commonly affects the head and voice, while in PD this distribution is virtually never seen. Aside from tremor, the associated neurological findings of ET and PD help distinguish the two conditions. ET is a monosymptomatic condition, tremor being the sole manifestation, whereas in fully developed PD, there may be coexistent bradykinesia, postural imbalance, and rigidity. In those cases of tremor dominant PD in which tremor may be the sole presenting sign, the correct diagnosis depends on identifying the tremor's classical circumstance of occurrence (at rest) and distribution (sparing the head and voice). An additional source of diagnostic uncertainty in the elderly patient is the possibility that ET and PD may both be present. Koller et al. (1994), in a study of 678 ET patients, found concurrent PD in 6.1%, an association that is slightly higher than would be predicted by the relatively high prevalence of the two conditions alone. In this regard, it should be noted that the late appearance of rest tremor in an elderly patient previously diagnosed as having ET is not sufficient to diagnose concurrent PD. Rajput et al. (1993) report the finding of resting tremor with no other signs of parkinsonism in three patients with confirmed essential tremor who underwent postmortem study. All three patients had additional postural and/or kinetic tremor of at least 10 years duration before the development of resting tremor. Resting tremor first developed in these three subjects when they were all older than 60 years. Similarly, cogwheel rigidity may appear in ET reflecting the presence of ongoing tremor in muscles that may not be properly relaxed. When other diagnostic criteria have failed to distinguish ET from PD, a trial of therapies that are specific for one or the other of these two conditions may prove useful. Levodopa has the potential to improve PD tremor remarkably, while it has virtually no effect on the tremor of ET. On the other hand, alcohol may dramatically improve the postural tremor of ET (Koller and Biary 1984) but has a less consistent and less impressive effect on the rest tremor of PD.

### **Senile gait**

Significant changes in the mechanics of ambulation appear in the normal elderly individual (Sudarsky 1990, Elble et al. 1992). The resultant pattern of walking has been referred to by a variety of terms, including senile gait, cautious gait, and *marche à petit pas*. This condition is characterized by a slow short stepped gait carried out on a slightly widespread base. Nutt and Horak (1997) have likened this gait pattern to that of a normal person walking on a slippery surface, and at risk for falling. Elble (1997) reviewed the kinematics of gait in older, healthy adults and found that the major changes were a slower velocity, a shorter stride length, reduced



arm swing, a more flexed knee position and reduced toe clearance at initial heel–floor contact. He also observed that these individuals spend a greater proportion of time in a double limb support stance than younger subjects. In this population, arm swing and lower extremity rotations are reduced in proportion to stride length. In addition to these abnormalities, Sudarsky and Tideiksaar (1997) noted that other kinematic investigations of the gait in this population revealed a reduction in velocity and a wider stride. These data clearly indicate that the gait of healthy aged individuals is characterized by a variety of features such as reduced stride length, diminished arm swing and axial flexion that overlaps with several of the typical clinical features of PD. The clinician must therefore not succumb to the temptation to diagnose PD or parkinsonism in an elderly individual solely on the basis of an abnormal gait and impaired mobility. Rather, additional confirmatory signs of a *movement disorder* must be sought, such as a decrease in facial expression, reduced blink rate, significant hypophonia, rest tremor, rigidity and freezing, singly or in combination. The presence of parkinsonism in addition to a gait abnormality is needed to confidently establish a diagnosis of PD or parkinsonism in an elderly individual. A corollary of this is that elderly patients presenting with a primary disorder of gait should not be assigned a diagnosis of senile gait if any of these ancillary clinical findings are present.

### **Neuroimaging and neurophysiological tests in the diagnosis of parkinsonism**

The differentiation of PD from other causes of parkinsonism is largely based on careful clinical observation and examination. However, in some instances neuroimaging or neurophysiologic studies may enhance the certainty of the clinical diagnosis. In conditions for which the causative gene has been identified and can be assayed, the laboratory may provide absolute confirmation of the presumptive clinical diagnosis.

Neurodiagnostic aids are almost never of significant benefit in confirming a diagnosis of PD. Neither magnetic resonance imaging (MRI) of the head nor computed tomography (CT) of the head, reveal any consistent findings. Subtle abnormalities are sometimes found in the region of the substantia nigra in MRI of the brain in late stage PD (Stern et al. 1989) but they are not sufficiently common or definitive to be of great practical benefit in everyday practice. Similarly, neurophysiologic tests such as electroencephalography, evoked potentials, and blink reflex studies, while revealing subtle abnormalities, are seldom useful in establishing a diagnosis of PD. Neuroimaging is occasionally useful in evaluation of patients with other forms of degenerative parkinsonism. This is especially true of advanced cases. Therefore, the primary rationale behind obtaining a neuroimaging study in a patient with parkinsonism is not to confirm a diagnosis of PD, but to determine

if a multisystem degenerative or vascular form of parkinsonism is present. In a survey of 49 movement disorder specialists, Anouti and Koller (1996) found that CT or MRI scans were ordered between 76% to 100% of the time to evaluate patients with suspected non-PD akinetic-rigid syndromes. Surprisingly, in the same survey, these studies were ordered 50% to 75% of the time in patients with a clinical diagnosis of PD.

In the late stages of PSP, MRI and CT may reveal atrophy of the dorsal mid brain with associated dilation of the cerebral aqueduct (Schonfeld et al. 1987, Yagishita and Oda 1996). A higher instance of multiple cerebral infarcts has also been noted in the scans of patients with PSP, raising the possibility that ischaemia may play an aetiological role in this condition (Dubinsky and Jankovic 1987).

A variety of abnormalities can be uncovered by neuroimaging in multiple system atrophy, again largely in the advanced stages of illness. In the striatonigral form of MSA signal abnormalities in the striatum can be seen, which helps distinguish the syndrome from PD. Most typical is putaminal hypodensity on T2 weighted MRI scans (Olanow 1992). In addition, a narrow band of hyperintensity can be seen in the lateral putamen, a finding not seen in PD (Konagaya et al. 1994). In the olivopontocerebellar type of MSA brain CT or MRI may reveal cerebellar atrophy and enlargement of the fourth ventricle (Mark and Sage 1993). In cases of suspected vascular parkinsonism diagnosis can be strongly supported by CT or MRI findings of discrete infarctions, lacunes or diffuse deep white matter signal abnormalities. The latter two abnormalities are much better demonstrated by MRI than CT. The appearance of these imaging abnormalities in elderly patients with clinically typical PD, however, cannot be given too much weight since cerebral vascular disease and PD are both common in this age group and may appear in the same patient independent of one another.

In normal pressure hydrocephalus a definitive diagnosis cannot be made without neuroimaging. Therefore, in patients in whom this diagnosis is being considered, and who are candidates for surgical intervention, CT or MRI must be performed to look for evidence of enlarged ventricles in the absence of significant brain atrophy. The diagnosis of several other less common causes of parkinsonism in elderly subjects is occasionally aided by neuroimaging. In CBD, CT or MRI can reveal frontoparietal atrophy, which is sometimes asymmetric (Riley et al. 1990). In Hallervorden–Spatz syndrome pallidal hypointensity on T2 weighted MRI scans is greater than that seen in normal individuals (Rodnitzky 1993). In advanced cases the ‘eye of the tiger’ sign is seen in the globus pallidus, consisting of a central increased signal surrounded by a zone of hypointensity (Sethi et al. 1988).

Positron emission tomography (PET) can provide a powerful research tool to define selective patterns of disruption of regional cerebral metabolism that helps distinguish between PD and other forms of neurodegenerative disease causing

parkinsonism (Brooks 1993). PET can also detect early sub-clinical PD in at-risk subjects.

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