Parkinson's Disease in Intensive Care Unit

Lionel Velly, Delphine Boumaza and Nicolas Bruder

Key Points

Parkinson's disease is the second most frequent neurodegenerative disease. It is due to the degeneration of pigmented cells in the dopaminergic system of the substantia nigra. Parkinson's disease is one of the rare neurodegenerative disease for which there exists a treatment. Levodopa stays the cornerstone of the medication. An interruption in levodopa or administration of dopamine antagonists can be responsible for a neuroleptic malignant syndrome-like. Dysautonomia is frequent, especially orthostatic hypotension. Aspiration pneumonia is one of the most frequent causes of death in these patients. Drugs interactions with Parkinson's medications are numerous and need to be known by physicians. Severe akinesia can benefit from a treatment with apomorphine. Opioids agents should be used with caution for patients with Parkinson's disease due to the risk of muscular rigidity and its complications.

Introduction

Idiopathic Parkinson's disease is after Alzheimer's disease the second most frequent neurodegenerative disease. Its prevalence in Europe is estimated at 1.6 % of the population over 65 years old in Europe. Frequency reaches 3.5 % of the population over 80 years [1]. This "shaking palsy" has been first described by James Parkinson in 1817. The cardinal symptoms are bradykinesia, extrapyramidal rigidity, rest tremor mainly asymmetrical and postural and gait disorders. Parkinson's disease is associated with the progressive emergence of a handicap, an impaired quality of life and an increased mortality.

L. Velly (✉) · D. Boumaza · N. Bruder
Department of Anesthesiology, CHU Timone Adultes, Aix Marseille University,
264 rue st Pierre, Marseille 13005, France
e-mail: lionel.velly@ap-hm.fr

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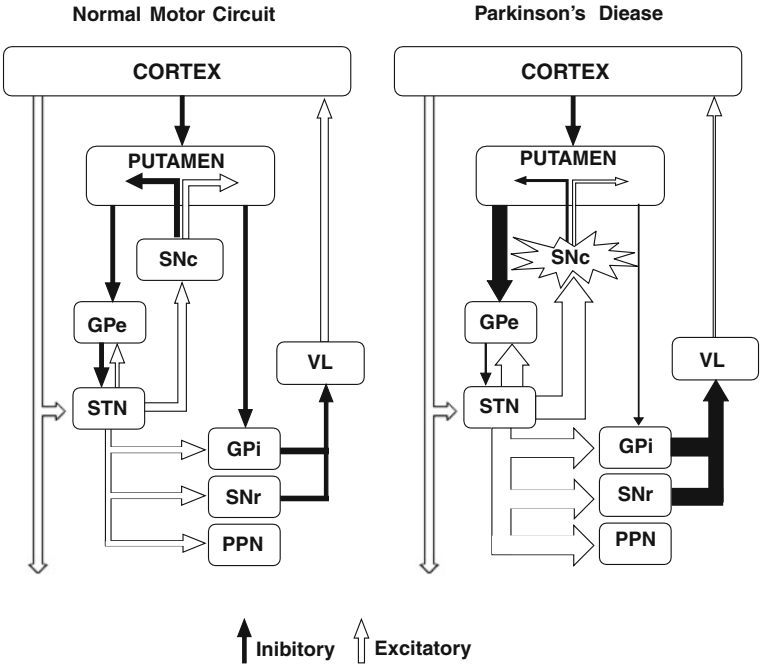

Fig. 1 Schematic representation of the classic model of the basal ganglia, illustrating the direct and indirect pathways connecting the striatum and the globus pallidus, and the modulatory effects of dopaminergic neurons on each of these systems. Excitatory fibres are shown in *black* and inhibitory fibres in *white*. The model predicts that neuronal firing in the STN and GPi are increased in the parkinsonian state, leading to excessive inhibition of brainstem and thalamocortical neurons with the development of parkinsonian motor features. In contrast, the model proposes that dyskinesia is related to decreased firing in the STN and GPi, with reduced inhibition of thalamic and cortical motor regions. *SNc* substantia nigra pars compacta; *GPe* external globus pallidus; *STN* subthalamic nucleus; *VL* ventralis lateralis; *Gpi* internal globus pallidus; *SNr* substantia nigra pars reticularis; *PPN* pedunculopontine nucleus; *DA* dopamine. Adapted from Obeso et al.

Physiopathology

Physiopathology of Parkinson's disease is complex, even though considerable progress has been done after discovering numerous genes involved in rare forms of the disease [2]. The correlation between clinical symptoms and the degeneration of pigmented cells in the substantia nigra dopaminergic system was not recognized until 1983. Briefly, dopaminergic insufficiency in the basal ganglia is responsible for a hyperactivity of the cholinergic neurons of the substantia nigra explaining the rigidity and a hypoactivity of the pallidum explaining the akinesia and the tremor (Fig. 1). Many cerebral structures are affected during the course of the disease. Olfactory process and locus coeruleus are affected early, leading to the initial occurrence of olfactory troubles and sleeping disorders (night agitation and

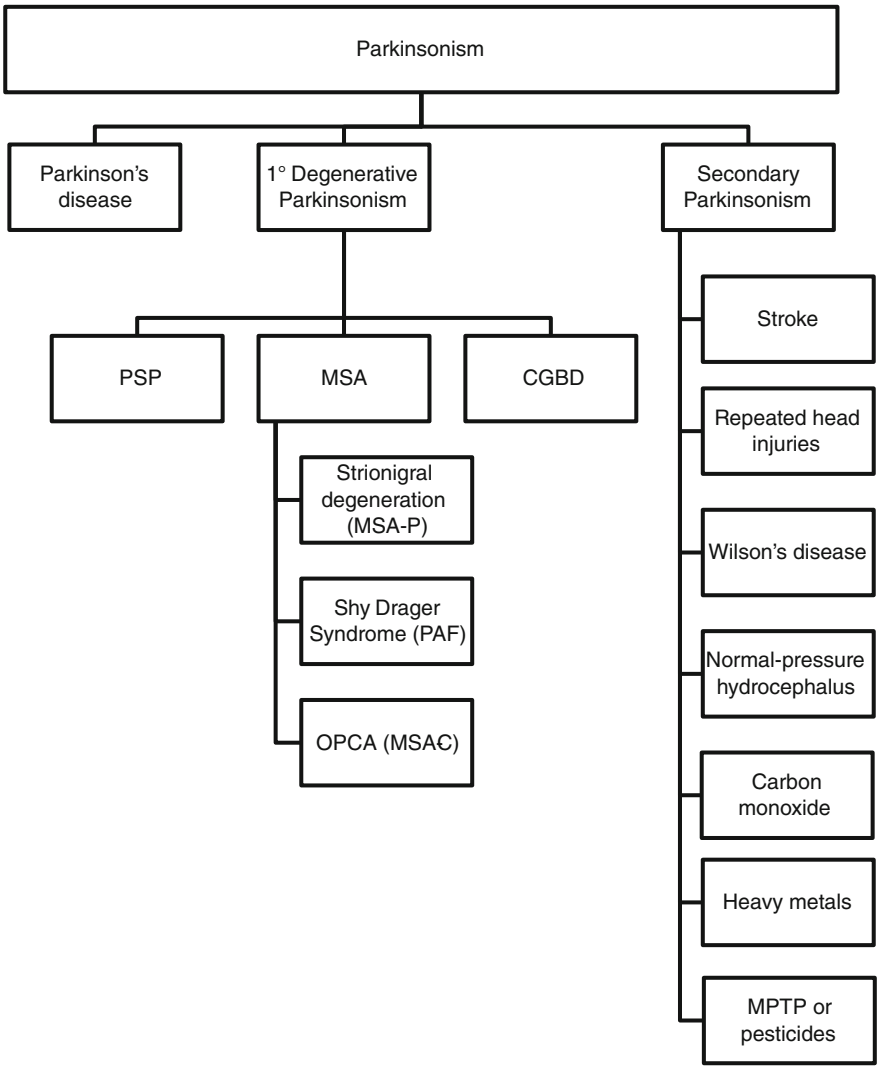

Fig. 2 Differential diagnosis of Parkinson's disease. *MSA-P* Multiple System Atrophy-Parkinsonian; *MSA-C* Multiple System Atrophy-Parkinsonian cerebellar; *PSP* Progressive Supranuclear Palsy; *CGBD* Cortical Basal Ganglionic Degeneration; *PAF* Pure Autonomic Failure; *OPCA* Olivopontocerebellar Atrophy

nightmares). Later on, structures involved in motor regulation such as the substantia nigra, located in the upper part of the brainstem, are involved and lead to motor disorders, characteristic of Parkinson's disease. Lastly, after many decades of evolution, cortical structures can be affected.

Parkinsonism, it is a generic name which includes clinical situations similar to Parkinson's disease (Fig. 2), which can have multiples causes such as arterial

sclerosis (strokes affecting the basal ganglia), repeated head injuries, enzymatic deficiency like Wilson's disease (differential diagnosis to evoke in young patients with familial history), normal-pressure hydrocephalus or following an intoxication with carbon monoxide, heavy metals, MPTP or pesticides,… [3]. It can also occur in the course of diffuse degenerative disease of the central nervous system, such as Lewy-bodies dementia (Parkinsonism appears then in old patients, rapidly associated with cognitive disorders, attention troubles, sleeping disorders and visual hallucinations), progressive supranuclear palsy or Steel Richardson's disease, corticobasal ganglionic degeneration or multiple-system atrophy also known as Shy-Drager syndrome (extrapyramidal signs associated with pyramidal signs, dysautonomia and cerebellar ataxia). These atypical patients have classically a poor response to treatment with Levodopa (L-Dopa: 3,4-dihydroxy-l-phenylanine), in terms of hypertonia and tremor. Furthermore, they suffer from hallucinations and dementia from the early stage of the disease. When a Parkinsonism or a Parkinson's disease is suspected in ICU, the advice of a neurologist is precious. The neurologist can help the diagnosis of an exacerbation of Parkinson's disease and help the prescription of L-Dopa. Parkinsonism can also be induced by administration of classical neuroleptics (butyrophenones, phenothiazines), atypical neuroleptics (amisulpride, risperdone, clozapine, quetiapine,…), but also hidden neuroleptics (metoclopramide) and the use of certain herbs such as kava. In this case, Parkinsonism is reversible with withdrawal of the treatment. When neuroleptics must be maintained, physicians should use preferably atypical neuroleptics such as clozapine or quetiapine, which are less suppliers of Parkinsonism.

Main Elements in the Treatment of Parkinson's Disease

Parkinson's disease is one of the rare neurodegenerative disease for which it exists a treatment. The aim is to reach a balance in striatum activity by increasing the cerebral dopaminergic capacities and/or decreasing the cholinergic activity. There are a lot of research leads regarding Parkinson's disease therapy but L-Dopa stays the cornerstone of the treatment [4].

Medical Treatment

L-Dopa and Adjuvant Therapy

L-Dopa stays since 30 years the most effective anti-Parkinson therapy. L-Dopa is the precursor of dopamine; it crosses the blood–brain barrier and helps to restore balance of the neurotransmitters between dopamine and acetylcholine (Table 1). L-Dopa is always combined with peripheral inhibitor of dopa-decarboxylase, benzerazide (co-beneldopa; Madopar®, Modopar®) or carbidopa (co-careldopa;

Sinemet® Caramet®), which inhibits the peripheral destruction of L-Dopa without crossing the blood–brain barrier. This combination decreases the adverse effects (e.g. nausea, hypotension) and increases cerebral bioavailability of dopamine. The aim of these inhibitors is to avoid the transformation of L-Dopa in dopamine in the digestive tract.

Efficacy of L-Dopa is superior to dopamine agonists. By contrast, it is now well established that L-Dopa has to be introduced later on in the course of the disease, especially in young patients, in order to delay the occurrence of dyskinesia that could be impressive and very disabling. These dyskinesias are writhing movements of various voluntary movements (opening and closing eyes, grimace, tongue movement, head rotation, shoulder lifting, winding of an arm or a leg, etc.). Mechanisms of occurrence of dyskinesia and motor fluctuations are complex and imperfectly understood but they are related to pulsatile administration of L-Dopa, severity of dopaminergic loss, absence of dopamine storage ability and changes of the glutaminergic system. In advanced PD, in order to maintain stable plasma levels, a suspension of micronized levodopa (20 mg/ml) and carbidopa (5 mg/ml) in a methylcellulose gel (Duodopa®) can be infused using a pump via an intestinal catheter placed inside a percutaneous endoscopic gastrostomy tube.

Tolerance of L-Dopa is far better than other Parkinson's medications. There are almost no contraindications, except for myocardial infarction in the acute phase, due to an increased risk of acute dysrhythmias. Confusion and hallucination can occur under treatment with L-Dopa, but rarely compare to these same adverse effects with anticholinergic drugs or dopamine agonists. Administration of L-Dopa with other enzymatic inhibitors allows improving tolerance. Those are the inhibitor of monoamine oxidase B (MAO-B) and inhibitor of catechol-O-methyltransferase (COMT). Selegiline is a relatively selective inhibitor of MAO-B with few effects on MAO-A. COMT is the other main destruction path of L-Dopa in periphery and less importantly in the synapse. COMT inhibition allows delivering a larger amount of L-Dopa, 30 % on average. Entacapone (COMTan® or co-careldopa plus entacapone: Stalevo®) and tolcapone (Tasmar®) are the COMT inhibitors currently commercialized in Europe. They act only in association with L-Dopa. Owing to hepatic side effects, tolcapone should only be used in case of non-response to entacapone.

Dopamine Agonists

The lessening of response and the motor side effects of dopatherapy in the course of the disease has led to the research of other therapeutics to improve dopaminergic transmission by stimulating directly post synaptic dopaminergic receptors in the striatum. Many dopamine agonists have then been discovered either derivate from ergot (bromocriptine, lisuride, pergolide) or synthetic drugs (piribedil, ropinirole, rotigotine). Except from apomorphine, all dopamine agonists have a lower power than L-Dopa, but their half-life and their duration of action are significantly longer and can reach hours. It allows a long-lasting and more stable

Table 1 Main anti-Parkinson Therapy

Levodopa + peripheral inhibitor of dopa-decarboxylase	Inhibitor of COMT	Inhibitor of MAO-B	Dopamine agonists	Anticholinergic agents	Antiglutamatergic agent
L-Dopa + benzerazide	Entacapone	Selegiline	*Derived from ergot*	Trihexyphenidyl	Amantadine
L-Dopa + carbidopa	Tolcapone		Bromocriptine	Tropatepine	
L-Dopa + carbidopa + entacapone			Lisuride	Biperiden	
			Pergolide		
			Synthetic drugs		
			Piribedil		
			Ropinirole		
			Rotigotine		

L-Dopa 3,4-dihydroxy-l-phenylanine; *MAO-B* L-monoamine oxidase B; *COMT* catechol-O-methyltransferase

stimulation of the postsynaptic dopaminergic receptors. Their efficiency as a monotherapy in the early stage of the disease has clearly been demonstrated in various studies. These studies have revealed a diminished incidence of motor complications, particularly the dyskinesia under dopamine agonists compared with L-Dopa alone. But as the disease evolves, association of dopamine agonists and L-Dopa seems superior to L-Dopa alone. When motor fluctuations and dyskinesia occur, the main interest of dopamine agonists is to reduce severity and duration of the "off period". All Dopamine agonists have more or less the same side effects such as nausea, abdominal pain, orthostatic hypotension, dizziness or somnolence. Lower limbs swelling occur with agonists, but more frequently with ergot-derivative drugs. Pulmonary and retroperitoneal fibrosis, although rare, have also been described with ergot-derivative drugs and seems related to a class-effect. Recently, cardiac valvular side effects have been reported with long-term pergolide treatment, probably also due to a class-effect [5]. This problem is thought to be due to pergolide's action at the 5-HT_{2B} serotonin receptors of cardiac myocytes, causing proliferative valve disease by the same mechanism as ergotamine. Switching treatment among agonists involves an estimation of dose equivalence and is done by replacing the agonist with the other one day from another (Table 2). A new transdermal dopamine agonist (rotigotine) is available. However, it seems less efficient than ropinirole and its indication remains to evaluate.

Other Medical Treatments

Amantadine initially prescribed as an antiviral agent has shown interesting anti-Parkinson effects and particularly a significant anti-dyskinesia effect.

Anticholinergic agents (trihexyphenidyl, tropatepine, biperiden) have been historically the first treatments available in Parkinson's disease. Numerous anticholinergic drugs exist and are mainly efficient on tremor, but out over many abdominal, ocular, sphincterian or neuropsychological side effects. Their use is therefore limited and restricted to young patients.

Surgical Treatment

This type of procedure is limited to patients with advanced Parkinson's disease, with frequent blockages, tremor, bradykinesia, rigidity and walking disorders or to patients with severe side-effects of medical treatment. Surgical ablation procedures such as thalamotomy (indicated in severe tremor) or pallidotomy (indicated in disabling rigidity) have been proved efficient but are irreversible. They are nowadays less and less performed. Subcortical stimulation using implanted electrodes has progressively replaced these ablation procedures [6]. Cerebral stimulation has been described for the first time in 1987 in the treatment of Parkinson's disease. Its aim is to trigger a functional inhibition by high frequency stimulation. At first, the

Table 2 Dopamine agonists

Drugs	Trade names	Ergot derivated drugs	Half life (hours)	Dose equivalent to 100 mg of L-Dopa (mg)	Dosing range (mg/day)
Apomorphine (30 mg/30 ml)	*Apokyn, lxense, Spontane, Uprima, Apokinon*	No	0.3–0.5	–	1–10 mg/injection 1–8 injections/day
Ropinirole (0.25; 0.5; 1; 2 and 5 mg)	*Requip, Ropark, Adartrel*	No	3–6	5–6	6–24
Piribedil (20 and 50 mg)	*Pronoran, Trivastal Retard, Trastal, Trivastan*	No	21	50–60	50–250
Pramipexole (0.18 and 0.7 mg)	*Mirapex, Mirapexin, Sifrol*	No	8–12	0.7	1.5–4.5
Bromocriptine (2.5; 5 and 10 mg)	*Parlodel, Cycloset, Bromo-Kin*	Yes	3–8	10	5–40
Pergolide (0.25; 0.25 and 1 mg)	*Permax, Celance*	Yes	16–21	1	0.5–5
Lisuride (0.2 and 0.5 mg)	*Dopergin, Proclacam, Revanil, Arolac*	Yes	1–7	0.6	0.5–5

intermediary ventral nucleus was the target of the stimulation. Now, the stimulation aims the subthalamic nucleus. Cerebral stimulation reduces efficiently for 2–5 years the motor fluctuations and dyskinesia in idiopathic Parkinson's disease. In most centers, after expert assessment by a neurologist seasoned in pharmacological treatment of Parkinson's disease, a pluridisciplinary team selects patients eligible for surgery. The ideal patient in this indication is the one who is severely impaired during the "off period" and in the meantime totally independent during "on period" without significant cognitive disorders. The most accurate prognostic test should be the remaining of a response to L-Dopa therapy. Principal complications related to the surgical technique are seizures (10 %), intra-cerebral hemorrhage (8 %; its incidence increases with per-operative high blood pressure) and temporary post-operative confusion (10 %). This type of procedures generally requires a post-operative care in ICU.

Management of Parkinson's Disease Patients in ICU

Fortunately, Parkinson's disease patients are often admitted to ICU with a pre-established diagnosis of the disease. ICU is not an appropriate place to diagnose *de novo* Parkinson's disease or distinguish the different etiologies of Parkinsonism. Many factors can interfere with neurological assessment (sedation, mechanical ventilation) and make the clinical examination incomplete. The main error that should not be done in the management of these patients is to stop, even for a few hours the dopaminergic therapy. An interruption of this treatment can have dramatic consequences. Some specificities have also to be known in Parkinson's disease patients. Besides the fact that it occurs in an aging population, the major risks are related to the respiratory impairment, favored by swallowing disorders and dysautonomia. It is also important to precise by interviewing family the level of handicap of the patient in daily-living activities.

Specificities Due to the Medical History

Respiratory Failure

The occurrence of respiratory impairment in the course of the Parkinson's disease has been observed since the first description of the disease in 1817 and pulmonary complications, especially aspiration pneumonia are the most frequent cause of death in those patients [7]. These alterations of the respiratory function are more frequent when a dysautonomia is associated to the Parkinsonism. Dysphagia appears from the early stage of the disease [2] and is due to asynchrony between swallowing and respiration and also to an important diminution of efficiency of the cough reflex [8]. Finally a hyper-sialorrhea is frequently present in those patients,

as well as an extended opening on the high esophagus sphincter and a slowing of the gastro-intestinal transit time favoring regurgitations. In any case, these troubles should be systematically investigated, particularly by screening severity criteria: cough during meals or during swallowing, recurrent pneumonias, time for the meal over 1 h, loss of weight. Warning signs should also be collected: tremor of the tongue, blockage of the alimentary bolus in the aerodigestive tract, hypersalivation and salivary incontinence, masseter hypertonia, fractioned swallowing, oral and nasal refluxes, dysphagia, and heartburn. When the reason for admission in ICU is related to pneumonia, an ENT endoscopic screening should be performed. When obvious food aspiration is observed, the indication of tracheotomy should be discussed with neurologist and ENT specialists.

During the course of Parkinson's disease, lung function tests are frequently altered [9]. An obstructive syndrome, responsible for a post-operative respiratory weakness is present in one-third of the patients. The obstructive syndrome can also be associated to a restrictive syndrome. This can be easily understood by the coexistence of rigidity and akinesia of respiratory muscles associated with laryngeal hypertonia, responsible for an inspiratory or expiratory obstruction of the upper airway. This rigidity of the chest wall is particularly important in ICU because it can compromise mechanical ventilation and the withdrawal from ventilator support. After extubation thoracic rigidity leads to hypoventilation, an inefficient cough, atelectasis and an increased risk of aspiration pneumonia. It is then advisable to limit as much as possible the "off periods". Similarly, after extubation, laryngeal akinesia can lead to laryngospasm and acute respiratory failure. L-Dopa or dopamine agonists intake have also a remarkable effect on airway obstruction. These patients have also an increased frequency of sleep apnea. Seldom, severe central apneas, dysrhythmic respiration or a Cheyne-Stokes pattern and central hypoventilation were described during severe Parkinson's syndrome with dysautonomia. Prolonged treatment with ergot-derivated drugs (bromocriptine, lisuride, pergolide) has been associated with pulmonary fibrosis [10].

Autonomic Disorders

They are frequent in Parkinson's disease and are related to a neurodegenerative process which causes a dysfunction of the *locus coeruleus* norepinephrine system and of the dorsal nucleus of the vagus nerve [11]. Dysautonomia is responsible for gastro-intestinal troubles (dyspepsia, hyper-sialorrhea, slowing of the bowel movement with constipation), urinary retention, tachycardia and cardiac arrhythmias. Dysautonomia is associated in more than 70–80 % of case with circulatory disorders. The most frequent trouble is orthostatic hypotension, present in more than 70 % of Parkinson's disease patients, which can be worsened by dopamine agonists. In addition to dysautonomia, the patients hold concurrently several risk factors for hypotension: old age, undernutrition, dehydration, and particular sensitivity to anesthetic agents. Intravenous administration of dopamine as a

vasopressor drug is not recommended in order to control Parkinsonism in ICU. Intravenous dopamine cannot reach the brain and can lead to many cardiovascular side effects such a tachycardia and arrhythmias. Anesthetics agents used for sedation should be titrated according to the clinical effectiveness and not according to defined dosages. These patients can also have thermoregulation troubles with increased sensitivity to hypothermia.

Practical Approach Concerning Anti-Parkinson Therapy in ICU

The principal aim during the stay in ICU is not to stop the dopaminergic treatment, particularly L-Dopa. The half-life of L-Dopa is very short (<3 h) and its interruption, even for a short time period can have important repercussion on muscular rigidity. It is frequent that an interruption of L-Dopa leads to akinesia, swallowing disorders, leading respiratory complications or worsening of a pre-existing respiratory condition. Moreover, an interruption of L-Dopa can also be responsible for an equivalent of malignant neuroleptic syndrome. It combines fever, rigidity, altered consciousness, rhabdomyolysis, sometimes complicated with acute renal failure and coagulation disorders [12]. Treatment with L-Dopa will be maintained at the same dosage than previously, taking care to ensure that the patient did not take more drugs in self-medication than prescribed. Because L-Dopa is absorbed in the jejunum, the difficulty is not to maintain dopa-therapy but rather to administrate it effectively. It is not always easy in those hemodynamically unstable patients to reach a therapeutic concentration range. Yet, at an advanced stage of the disease, motor fluctuations are very sensible to minimal changes in the rhythm of administration or the dose of L-Dopa, even at the protein contain of the food intake. A large intake in protein should decrease the absorption of Sinemet. Protein in the meal is broken down in the intestine into amino acids that must travel across the intestinal wall to get into the blood. Levodopa also must transit the intestine using exactly the same carrier system as the amino acids. In ICU, an administration through a gastric tube, sometimes through a post-pyloric tube in case of gastroparesia, is often the only option. Many galenical formulations exist: standard, sustained-release or dispersible for drinkable solution (rapid action). The standard form of Sinemet®, unlike the sustained form and certain capsule, can be used without significant modification of its pharmacokinetic pattern by muddling and administration through gastric tube. The dispersible form of levodopa can be very useful since it has a much faster and more constant onset of action than the standard preparation. However it has to be given every 2–4 h due to its short half-life. In countries where an intravenous form of L-dopa is available, intravenous administration is convenient and effective for perioperative management. In any case, neurologist advice is essential to adjust the treatment. Administration of vitamin B6 (pyridoxine) should be avoided. Vitamin B6 is a coenzyme of

peripheral L-Dopa decarboxylase, favoring the inactivation of L-Dopa and decreasing its efficacy. When prolonged fasting is necessary (e.g. gastric surgery), administration of Parkinson's medications becomes a real challenge. Subcutaneous injection of apomorphine or transdermal administration of rotigotine (Neupro®) may be an option. The dose of apomorphine to treat akinesia is 2 mg intravenously or subcutaneously every 10–15 min until regression of the symptoms. An important side effect is nausea and vomiting, even more when the patient has never been treated with apomorphine. The approach is less consensual for other treatments. It is probably advisable to stop progressively anticholinergic agents because they increase the risk of delirium. The pursuit of ergot-derivative dopamine agonists should be cautious in case of prescription of catecholamine and strictly proscribed when a prescription of macrolides (erythromycin, josamycine) is needed. There is a potential risk of "*ergotism*", responsible for necrosis of extremities by severe vasoconstriction. Substitution by a synthetic dopamine agonist may be considered after neurologist advice. Monoamine oxydase (MAO) inhibitors type A have been associated with major hemodynamic changes, especially in association with sympathomimetic agents. MAO inhibitors used in the treatment of Parkinson's disease (selegiline) have a selective action on cerebral MAO-B with low affinity on peripheral MAO-A. This explains the possibility to use indirect sympathomimetic agents such as ephedrine, with selegiline. A severe interaction persists though between selegiline and pethidine. The co-prescription of selegiline and opioids is restricted. Usually, selegiline may be interrupted with few adverse effects.

Medication Worsening Parkinsonism

Parkinson's disease patients and neurodegenerative Parkinsonism are particularly susceptible to anti-dopamine agents such as neuroleptics (phenothiazine, butyrophenone) or certain anti-emetics (metoclopramide, promethiazine). Very low doses of these drugs can give rise to severe hypertonia, akinesia and a neuroleptic-like malignant syndrome. If administration of an anti-psychotic agent is necessary, newer agents should be preferred (clozapine or quetiapine) with systematic and repeated screening for worsening of a Parkinson's symptoms or fever. Concerning anti-emetics, it is preferable to use setrons.

Practical Approach Concerning Anaesthetic Agents

Some specificities have to be known by physicians in sedation agent management [13, 14]. Concerning hypnotic agents, little evidence is currently available in literature about Benzodiazepines. It seems that just as the elderly patients, Parkinson's disease patients have an increased susceptibility to this class of drugs.

No complication such as hypertonia or rigidity has been reported with benzodiazepines. Concerning Thiopental, several cases have been reported with worsening of the Parkinson's syndrome following its use. The evidence of a causal link has not been established but its administration should be cautious. In animals, thiopental diminished the dopaminergic release in the striatum. Propofol can be used but with reduced dosage due to the higher susceptibility of Parkinson's disease patients [15]. However dyskinesia with propofol have been described, particularly after interruption of L-Dopa therapy. In patients receiving L-Dopa, co-administration of ketamine can be responsible for an exacerbated sympathetic response. Low doses (0.1–0.5 mg kg^{-1}) treatment has been reported without significant adverse effects. Opioid use should also be cautious in Parkinson's disease patients. There are several cases of muscular rigidity reported in literature following the use of fentanyl. Opioid agents in general induct a muscular rigidity by presynaptic inhibition of dopamine release. Cases of acute dystonia have also been described with alfentanil. Titration of narcotics is difficult because the classical signs of pain (tachycardia, hypertension) can be masked by dysautonomia. Moreover, behavioral pain scales can also be misinterpreted in cases of akinesia. Use of morphine in Parkinson's disease allows, at low doses, a diminution of dyskinesia while high doses can lead to akinesia. There are no reported cases of non-depolarizing neuromuscular blocking drugs worsening the symptoms of Parkinson disease.

Other Preventive Measures

Parkinson's disease patients are more at risk of post-operative complications that are often, diagnosed late in those patients. The diminution of spontaneous movements of the body and limbs due to rigidity and akinesia exposes these patients to an increased risk of deep vein thrombosis, pulmonary embolism, bedsores and peripheral neuropathy by compression. As for the other cases of immobility, anticipation and prescription of prophylactic measures are essential. It is recommended to resort to early mobilization out of bed, intermittent pneumatic compression of the lower limbs and a prescription of a preventive dose of heparin (low molecular weight or unfractionated heparin).

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