Gastrointestinal Motility Problems in Patients with Parkinson’s Disease
Effects of Antiparkinsonian Treatment and Guidelines for Management

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Summary

Gastrointestinal (GI) motility disorders are frequent in patients with Parkinson’s disease, manifesting mainly as dysphagia, disorders of gastric emptying and constipation. The most likely causes of these disorders are cerebral degeneration and degeneration of the myenteric plexus. Although the effect of antiparkinsonian medication is largely overestimated, it certainly has an influence and should be adapted accordingly in patients with GI motility disorders. In particular, anticholinergic drugs should be avoided, and anamnesis, clinical examination and, if necessary, diagnostic tests performed.

Domperidone, a peripheral dopamine antagonist, is the drug of choice for motility disorders of the upper GI tract, although cisapride is an alternative. In the lower GI tract, conservative therapeutic options should be used in the first instance. The administration of cisapride leads to a marked temporary improvement in symptoms in lower GI disorders, while rare forms of anism (involuntary dystonic contraction of the anal sphincter) may be treated with botulinum toxin.

Disturbances of the gastrointestinal (GI) tract are considered to be the most frequent autonomic disorders in Parkinson’s disease (PD). They are probably caused by degenerative changes, evidenced by the presence of so-called Lewy bodies from the oesophagus to the rectum in the myenteric plexus.
plexus and the submucosal plexus. The entire GI tract may therefore be affected, resulting in dysphagia, delayed gastric emptying and constipation.

1. Hypersalivation

*His saliva was continually trickling out of his mouth, and he had neither the power of retaining it, nor of spitting it out freely.*

J. Parkinson

Hypersalivation is one of the classical symptoms of parkinsonism. Eadie and Tyrer described hypersalivation (defined as the clinical observation of saliva trickling from the corner of the mouth when slightly opened) in 74% of their patients with PD. This observation was confirmed by Oppenheim, who described hypersalivation as being a frequent and early symptom in parkinsonism. Oppenheim noted that hypersalivation did not appear to increase the frequency of swallowing, and assumed that the condition was a primary secretion disorder characterised by the hypersecretion of saliva.

However, more recent investigations have shown that hypersalivation results from dysphagia and not from the increased production of saliva. Bateson et al. demonstrated that salivation at rest and after stimulation with a sialogogue is reduced in parkinsonian patients compared with young, healthy individuals. They also showed that there was no difference in the production of saliva between parkinsonian patients with or without hypersalivation. Patients with PD have a tendency to bend forwards, lowering their heads and leaving their mouths slightly open. The tendency of patients to drool is favoured by the reduced tone of the orbicularis oris muscle.

Bateson et al. examined 8 parkinsonian patients who complained of hypersalivation, 9 patients with PD and dry mouth, and 14 control individuals. They observed that the production of saliva was reduced in elderly patients, and that men had slightly higher levels of production compared with women. There was no difference between the 2 PD groups, both showing lower levels of salivation than the control individuals.

Anticholinergic drugs are recommended for therapy, since they reduce salivary flow within a few weeks. Withdrawal of anticholinergic agents may provoke the rebound excessive production of saliva. Anticholinergics merely reduce the production of saliva, and do not influence the underlying cause of the sialosis. On the contrary, anticholinergics can actually exacerbate swallowing difficulties.

In summary, hypersalivation is a common complaint in PD, and is caused by disordered swallowing rather than the increased production of saliva. The ideal course of action is therefore not to reduce salivary secretion with anticholinergic drugs, but to treat the dysphagia (see section 2).

2. Dysphagia

Parkinson himself described severe dysphagia, allowing the ingestion of only liquid food, in his patients. Schwab and England also noted various swallowing disorders in patients with PD, especially problems with spontaneous swallowing leading to hypersalivation. Dysphagia is found in 50 to 75% of all PD patients, and some authors have reported a prevalence of up to 95%. Eadie and Tyrer examined 72 PD patients and found a significantly increased presence of hiatus hernia, gastro-oesophageal reflux, segmental spasms of the oesophagus and delayed transit through the oesophagus, compared with control individuals. In many patients dysphagia remains asymptomatic, but even in those patients swallowing disorders can be demonstrated. Some overlap with achalasia has also been described. The whole process of swallowing is disturbed in PD patients, and the problem is aggravated by insufficient chewing because of weakness or stiffness of the chewing muscles.

During the process of swallowing, food is not adequately pushed towards the pharynx, and transportation through the oesophagus is delayed. This is most likely to be caused by reduced peristalsis and disordered coordination, together with inadequate relaxation of the oesophageal sphincter. According to Eadie and Tyrer, chewing dis-