Breathing problems in Parkinson's disease: a common problem, rarely diagnosed

Parkinson's disease (PD) is the second most common neurodegenerative disorder after Alzheimer's disease. It is characterized by bradykinesia (slowness in movement) tremor, rigidity, and postural instability. Potential non-motor manifestations of PD include depression, anxiety, constipation, overactive bladder symptoms, dementia, and sleep disturbances.

Although James Parkinson, in 1817, described breathing abnormalities in his "Essay on the shaking palsy", there has been limited research on this important non-motor symptom.

People living with Parkinson's may present with a wide variety of respiratory symptoms, ranging from shortness of breath at rest (SOB) to acute stridor. Shortness of breath can be very distressing for patients and clinicians alike. Multiple investigations may be undertaken, looking for infection, blood clots and heart problems. Although these potential causes of breathing abnormalities need to be excluded, clinicians must remember that PD itself and its medications can cause SOB; and that normal investigations should not automatically lead to a diagnosis of anxiety, depression or lead to inappropriate treatment plans.

Several different patterns of breathing abnormality may be found in PD:

> Upper airway obstruction (UAO) has been reported in a third of people with PD. The most common manifestation of UAO is soft speech, which itself may affect up to 70% of people with PD. Two types of UAO have been described: (i) "respiratory flutter" whereby the vocal cords oscillate at a frequency similar to that seen in the peripheral tremor observed in PD and may result in a 'shaky' voice or in noisy breathing; (ii) less common is a delay in expiration (so it feels harder to breathe out), which can at times lead to complete airway obstruction.
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> Restrictive breathing abnormalities have been reported in 28% to 94% of people living with Parkinson's (the wide variation reflecting population selection bias). The underlying mechanisms of this pattern of breathing abnormality in PD are not fully understood but are likely to include a combination of factors: increased chest wall rigidity; a reduction in lung volume secondary to stooped posture; and lung changes secondary to ergot-derived dopamine agonist drugs (no longer in common usage).
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> Normally, our breathing is driven by high carbon dioxide (CO_2) and low oxygen (O_2) levels in our blood. This is regulated by the brainstem and carotid bodies respectively. However in PD, the perception of breathing can be heightened or lowered making the individual feel more SOB than they appear. This can result in them being misdiagnosed as being depressed or anxious. A possible explanation for this altered perception of breathing may

be due to the loss of dopaminergic input to the brainstem and carotid bodies leading to altered regulation of CO_2 and/or O_2.

Parkinson's medications can also be the cause of SOB. Ergot-derived dopamine agonists, rarely used in recent years, can result in pleural effusions and/or fibrosis leading to SOB. Levodopa induced diaphragmatic dyskinesias may affect breathing and SOB may also be a symptom of the levodopa wearing-off phenomenon.

At present, the exact mechanism(s) of breathing problems in PD is not clear. The prevalence of obstructive and restrictive respiratory problems in PD vary widely between studies and studies disagree on whether levodopa helps breathing problems; some even report levodopa makes breathing worse. More work is needed to look at the prevalence, underlying pathophysiology and management of breathing difficulties in PD.

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Publication

Respiratory dysfunction in Parkinson's disease.
Torsney KM, Forsyth D
J R Coll Physicians Edinb. 2017 Mar