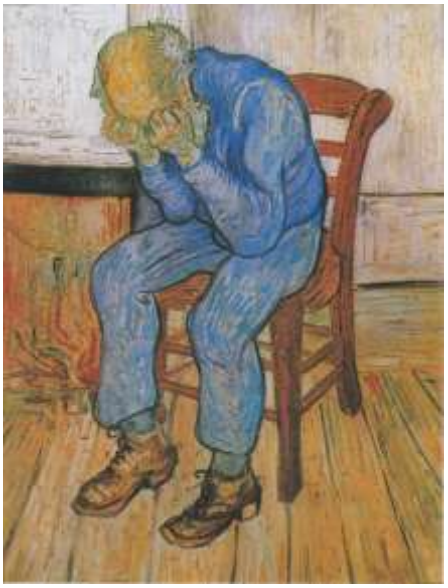
Perceiving Parkinson's

Cognitive Difficulties In Parkinson's (Day 63)

Thus far, the nonmotor symptoms of Parkinson's such as autonomic and enteric dysfunction, mood disorders, sleep disruption, and pain may manifest at any time over the course of the condition, even before the motor symptoms appear. It is not until the **later stages of Parkinson's** that cognitive difficulties show up - namely, dementia and psychosis.

Dementia

Eventually, **20-50%** of people with Parkinson's will develop **minimal cognitive impairment**, but only **10-30**% of people with Parkinson's will develop **dementia**. The difference between the two is that minimal cognitive impairment is a cognitive deficit that **does not** impair normal daily function, but dementia is a cognitive deficit that **does** impair normal daily function. Dementia must appear one year or more after the motor symptoms for it to be called **Parkinson's dementia**; if dementia appears any sooner than this, it may be **Lewy body dementia**.



Van Gogh's *Sorrowing Old Man (At Eternity's Gate)* could depict depression, dementia, or both.

People with Parkinson's dementia have **difficulty performing activities of normal daily living** due to impairments in one or more of interest, attention, concentration, planning, multi-tasking, short term memory, and visuospatial function. One of the earliest features of Parkinson's dementia is **apathy**, a state of disinterest or indifference to things that used to be of interest, such as hobbies. While apathy is often misinterpreted as depression, it is more closely related to dementia.

The **management** of Parkinson's dementia is as follows:

(1) First, **reversible sources of dementia must be excluded**. There are many "dementia mimics" including **depression**, **excessive daytime sleepiness**, **thyroid disease**, **severe anemia**, **liver or kidney disease**, **brain tumour**, **stroke**, and **medications that produce memory impairment** such as dopamine agonists (ropinirole, pramipexole), benzodiazepines (clonazepam, lorazepam), narcotics (codeine, oxycodone), antidepressants (amitriptyline, nortriptyline, trazodone, mirtazapine), antipsychotics (quetiapine, olanzapine), baclofen, and tramadol. Brains scans, blood tests, and even a lumbar puncture may be required to exclude a reversible source.

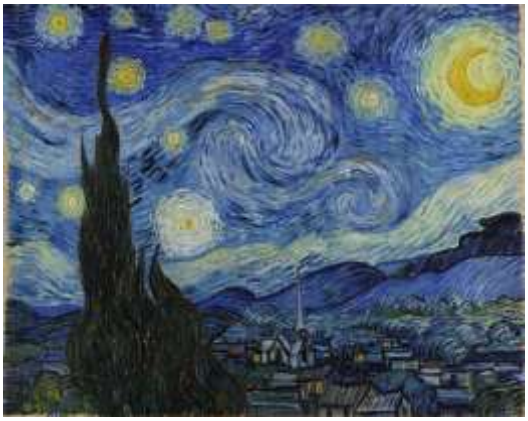
(2) If no reversible source is found, anti-dementia **medications** may be tried. Unfortunately, these medications have only a very modest benefit in Parkinson's dementia. **Donepezil** is weakly effective but some people cannot tolerate the side-effects of insomnia, nausea, and diarrhea. **Rivastigmine** also works but its side-effects include nausea and weight loss. Less common options include **galantamine**, which induces nausea and diarrhea, and **memantine**, which induces headache and confusion.

(3) Specific evidence for further options in Parkinson's dementia is lacking, but some Parkinson's specialists advocate **exercise**, **art therapy**, and **music therapy**.

(4) Eventually, many people who develop Parkinson's dementia require **a carer or care facility**.

Psychosis

Parkinson's dementia often coexists with **psychosis**, defined as a loss of contact with reality. The symptoms of psychosis are **hallucinations** (sensing things that are not there), **delusions** (believing things that are not true), **illusions** (misperceptions of things that are there), and **a false sense of presence** (a feeling that someone else is there, even though there is nobody present).



Van Gogh's *The Starry Night* depicts changes in light and colour that can appear in psychosis.

The most common symptoms of psychosis are **visual hallucinations**, usually of small children or animals; visual hallucinations affect **up to 30%** of people with Parkinson's. Hallucinations related to the other senses - hearing, smell, and touch - may also occur. Less frequently, there may be **paranoid or jealous delusions**, often related to spousal infidelity or abandonment by family; delusions affect

only **5-10%** of people with Parkinson's. If hallucinations or delusions progress, loss of insight follows as the person gradually loses touch with what is real, and what is not.

The **management** of psychosis in Parkinson's is as follows:

(1) First, **reversible sources of psychosis must be excluded**. The possibilities include **infections** and **toxins**, but the **dementia mimics** must be considered too, as they can also produce psychosis. If medications are suspected, the usual suspects such as benzodiazepines (clonazepam, lorazepam), narcotics (codeine, oxycodone), and anticholinergics (amitriptyline, nortriptyline) should be pulled off first, followed by dopaminergic oral medications, leaving levodopa until last.

(2) If no reversible source is found, antipsychotic **medications** may be tried. Quetiapine and clozapine have the best evidence for psychosis in Parkinson's. **Quetiapine** has weak evidence but a somewhat tolerable side-effect profile of sedation, postural hypotension, and a slightly increased risk of death compared to placebo. **Clozapine** has strong evidence, but its side-effect profile is tricky - in addition to sedation, postural hypotension, and an increased risk of death compared to placebo, clozapine has a <1% chance of inducing a potentially lethal condition called **agranulocytosis**, therefore blood tests must be done every week for six months, followed by fortnightly for six months, followed by monthly indefinitely. Thus, clozapine has risks and involves a degree of hassle, but it may be useful in select people. Some Parkinson's specialists believe that **donepezil** and **rivastigmine** may be helpful in mild to moderate psychosis.

The bottom line is this - if a person with Parkinson's is developing dementia or psychosis, it is vital to **exclude reversible sources** for the dementia or psychosis. If a reversible source is found, its removal can result in a drastic reduction or elimination of the cognitive difficulties. If no reversible source is found, management is admittedly difficult.

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References

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(2) Ahlskog. 2015. The New Parkinson's Disease Treatment Book. Oxford University Press.

(3) Sveinbjornsdottir. 2016. The clinical symptoms of Parkinson's disease. Journal of Neurochemistry 139(Suppl. 1), 318-324.