Drug Induced Movement Disorders

Image 1–4: Formafantasma: Seeing the Wood for the Trees, Kunsthaus Hamburg, Installation view, 2022, photos: Hayo Heye

1. Overview/Background
2. Tardive Syndromes
3. Drug Induced Parkinsonism
4. Akathisia
5. Drug induced Tremor

1. Overview

Spectrum of Drug-Induced Movement Disorders

- Parkinsonism
- Levodopa induced dyskinesias
- Tremor (parkinsonian; enhanced physiologic; cerebellar)
- Chorea
- Myoclonus
- Tics
- Tardive syndromes
  - Tardive dyskinesia
  - Tardive dystonia
  - Tardive stereotypies
  - Tardive Tourettism
  - Tardive akathisia
  - Tardive respiratory dyskinesias
  - Tardive pain syndrome (usually admixed with tardive dyskinesia)
  - Tardive oculogyric crises

Ann Med Psychol (Paris). 1952 Jun;110(2 1):112-7.

[Therapeutic use in psychiatry of phenothiazine of central elective action (4560 RP)].

[Article in Undetermined Language]

DELAY J, DENIKER P, HARL JM.

PMID: 12986408

[Indexed for MEDLINE]

Chlorpromazine, synthesised in December 1951

First tried clinically a few months later

Jean Delay & Pierre Deniker

for prompt control of

senile agitation

THORAZINE*

(chlorpromazine, S.K.F.)

'Thorazine' can control the agitated, belligerent senile and help the patients to live a composed and useful life.

To control agitation—a symptom that cuts across diagnostic categories

Thorazine®, a fundamental drug in psychiatry—Because of its sedative effect, 'Thorazine' is especially useful in controlling hyperactivity, irritability and hostility. And because 'Thorazine' calms without clouding consciousness, the patient on 'Thorazine' usually becomes more sociable and more receptive to psychotherapy.

® brand of chlorpromazine

leaders in psychopharmaceutical research SMITH KLINE & FRENCH

1957

First report of irreversible orofacial stereotypic involuntary movements: ‘paroxysmal dyskinesia’

Tardive syndromes and antipsychotics

Tardive: from French, Tardif, meaning late.

Complex Problem:

Months OR years of treatment with antipsychotics.

Condition is caused by antipsychotics,
BUT can also be suppressed by antipsychotics.

*It is not only dopamine blockers..

Lithium

Antidepressants

Anticholinergics

Anticonvulsants

Amiodarone

Contraceptives

Anabolic steroids

REVIEW ARTICLE

International Journal of Geriatric Psychiatry

Comparing the risk of tardive dyskinesia in older adults with first-generation and second-generation antipsychotics: a systematic review and meta-analysis

Angela O'Brien[1,†]

Waitemata District Health Board, Auckland, New Zealand
Correspondence to: Dr. A. O'Brien, E-mail: angela.obrien@waitematadhb.govt.nz

Conclusions: The risk of probable TD is more than three times lower in older adults receiving SGAs in comparison with FGAs after 1 year of treatment (23% vs 7%). The risk of persistent TD at 1 year with SGAs is particularly low. Evidence is lacking in regard to the longer-term risk of TD with SGAs, although the rates associated with the prolonged use of FGAs are high. Caution is therefore still required, particularly with the protracted use of both FGAs and SGAs.

Approach to drug induced abnormal movements

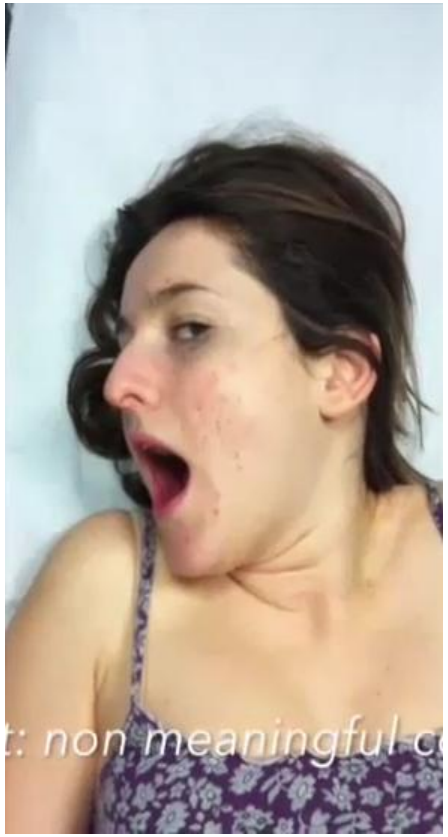
Time Course: Acute/Subacute/Chronic

<u>Each</u> has different presentations

ACUTE

Acute Drug Induced Dystonia

Affects virtually any part of the body, though the cranial and neck regions are most commonly involved



- Torticollis
- Retrocollis
- Jaw
- Swollen Tongue Difficulty swallowing→laryngospasm
- Occasionally Pisa syndrome

Acute Dystonic Reaction: oculogyric crisis

These are severe (potentially life threatening) problems, and urgent treatment is required.

Typically, an indication for intravenous anticholinergic agent.

Fortnightly review

Acute dystonia induced by drug treatment

Peter N van Harten, Hans W Hoek, Rene S Kahn

BMJ 1999;319:623–6

Drug induced dystonia can be prevented either by adding, during the first four to seven days of treatment, anticholinergic drugs to treatment with antipsychotic drugs or by starting treatment with atypical antipsychotics

Table 1. Medicines with clinically significant anticholinergic effects that are commonly used in older people with dementia[6–8]

	Antipsychotics	Antidepressants	Medicines for urinary incontinence	Antihistamines
Strong anticholinergic effects – avoid using in people with dementia	Chlorpromazine Olanzapine Pericyazine	Tricyclic antidepressants (eg. amitriptyline, doxepin, imipramine)	Darifenacin** Oxybutynin Propantheline Solifenacin** Tolterodine**	Brompheniramine*** Chlorpheniramine*** Cyproheptadine Diphenhydramine*** Promethazine***
Moderate anticholinergic effects – use with caution in people with dementia	Haloperidol Prochlorperazine Quetiapine Risperidone Ziprasidone	Desvenlafaxine Duloxetine* Fluoxetine Mirtazapine Paroxetine Reboxetine* Venlafaxine		

CNS Drugs (2024) 38:239–254
https://doi.org/10.1007/s40263-024-01078-z

CURRENT OPINION

An Evidence-Based Update on Anticholinergic Use for Drug-Induced Movement Disorders

Nora Vanegas-Arroyave[1] · Stanley N. Caroff[2,3] · Leslie Citrome[4] · Jovita Crasta[5] · Roger S. McIntyre[6,7] · Jonathan M. Meyer[8] · Amita Patel[10,9] · J. Michael Smith[11] · Khody Farahmand[12] · Rachel Manahan[12] · Leslie Lundt[12] · Samantha A. Cicero[12]

Accepted: 27 February 2024 / Published online: 19 March 2024

- DIMDs are neurobiologically and clinically distinct, with diferent treatment paradigms and varying levels of evidence for anticholinergic use.
- Anticholinergic use **not** supported for preventing DIMDs except in individuals at high risk for acute dystonia.

SUBACUTE

SUBACUTE

Parkinsonism due to dopamine blockade

Parkinsonism tends to have an insidious
onset and often starts after several weeks of antipsychotic treatment

SUBACUTE

Parkinsonism due to dopamine blockade

SUBACUTE

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- Anticholinergics can be effective for DIP and dystonia

SUBACUTE

Parkinsonism due to dopamine blockade

Although anticholinergics are very effective for the treatment of acute dystonic reactions in patients treated with neuroleptics, there is neither evidence nor rational support for the idea that these compounds could alleviate DIP [124].

Review

EXPERT OPINION

Drug-induced parkinsonism

José López-Sendón, Maria A Mena & Justo G de Yébenes[†]
[†]*Hospital Ramón y Cajal, Servicio de Neurología, CIBERNED, Madrid, Spain*

2. Tardive Syndromes

CHRONIC..”TARDIVE DYSKINESIA”

Tardive dyskinesia

- **Stereotyped choreoathetoid movements predominantly involving the lips, tongue and perioral region**
- **Movements predominate in the lower face**
- **Movements of the tongue in a writhing motion inside the mouth**
- **Rapid tongue protrusion and pushing the tongue against the inside of the cheek**
- **Chewing, grimacing, lip smacking and puckering**
- **May be accompanied by low amplitude choreiform movements of the distal extremities – fingers and toes**

Source: Waln, O., & Jankovic, J. (2013). An Update on Tardive Dyskinesia: From Phenomenology to Treatment. Tremor and Other Hyperkinetic Movements, 3, tre–03–161–4138–1.

International Parkinson and Movement Disorder Society

Epidemiology

Tardive dyskinesia is the most frequent TS (of 100 patents with TS approximately 70% will have Tardive dyskinesia., 30% tremor, 20% akathisia, and 15-20% dystonia (many people will have multiple symptoms.)

Although the tardive disorders are traditionally considered to arise late in the course of treatment

Shown for both OBLD and tardive dystonia that persistent cases may arise after **short** periods of exposure.

- **No minimum period of exposure that can be considered as entirely safe.**
- Can occur **years** after treatment started

Tardive Syndrome: wide range of phenomena

Dyskinesia
Dystonia
Akathisia
Myoclonus
Tics/Tourettism
Stereotypies
Tremor
Chorea
Tremor
Pain
Respiratory dyskinesias

Classic Tardive Dyskinesia
Mostly choreiform
OBL
Worsened by AntiCholinergics

These 2 conditions are not mutually exclusive

Tardive Dyskinesia and Dystonia

Who gets Tardive **dystonia**?

> 60% among those younger than 20 years

Almost zero after the age of 40 years.

Tardive dystonia can develop at any time between 4 days and 23 years after exposure to dopamine receptor blockers, and there is no safe period.

May spread and result in focal or generalized dystonia

.

The natural history of tardive dystonia

Brain (1998), 121, 2053–2066

Tardive Dyskinesia and **Dystonia**

Tardive dystonia

- 16F on risperidone for OCD and psychosis NOS
- Gradual onset involuntary movements affecting mainly the lower face, neck, trunk and upper limbs
- Retrocollis, truncal extension, hyperpronation of the arms
- Sensory tricks
  - Holding right side of neck
  - Curling onto right side
  - Crossing arms over chest or forehead

Dr. Tamara Pringsheim
University of Calgary (MDS Congress, Philadelphia, 2024)

Tardive dystonia, treated

- Treated with withdrawal of risperidone and started on anticholinergic (trihexyphenidyl)
- Maintained on trihexyphenidyl for two years, then gradually tapered off over one year

However, jaw dystonia is a chronic issue in patients of any age, and tracks OBLD (makes some sense).

Stephen Reich

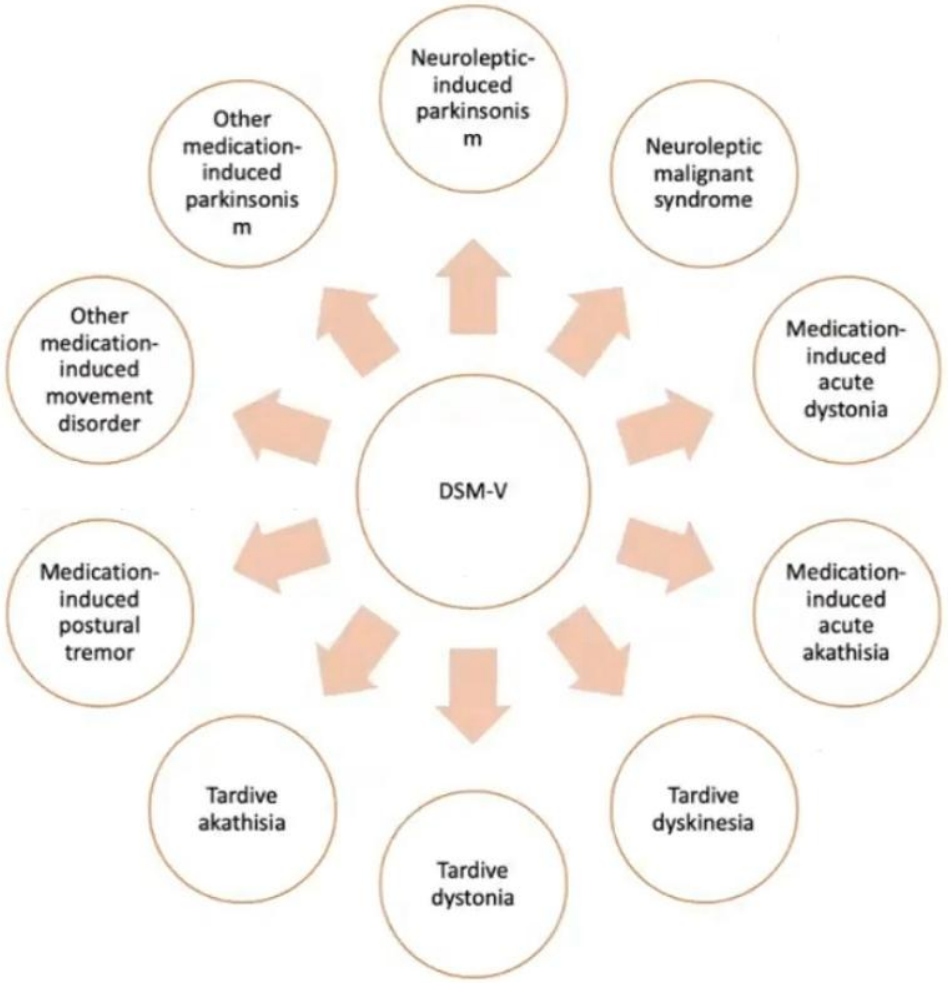
Tardive Oromandibular (jaw closing) dystonia

Tardive oromandibular (jaw opening) dystonia

Some additional entities

Withdrawal dyskinesias

Stereotypies



Withdrawal emergent dyskinesia

- Develops after either abruptly stopping or significantly reducing the dose of neuroleptic medications
- Predominantly affects children
- Usually manifests as generalized chorea
- Usually self limiting and resolves over weeks
- Re-start medication at lower dose and perform slower taper

Withdrawal emergent dyskinesia

- 9M treated with risperidone 3 mg for irritability related to autism since age 3 (6 years)
- Community pediatrician decided to taper and discontinue medication over a period of 3 weeks (1 mg per week)
- Whole body involuntary and continuous movements emerged during the final days of the tapering process

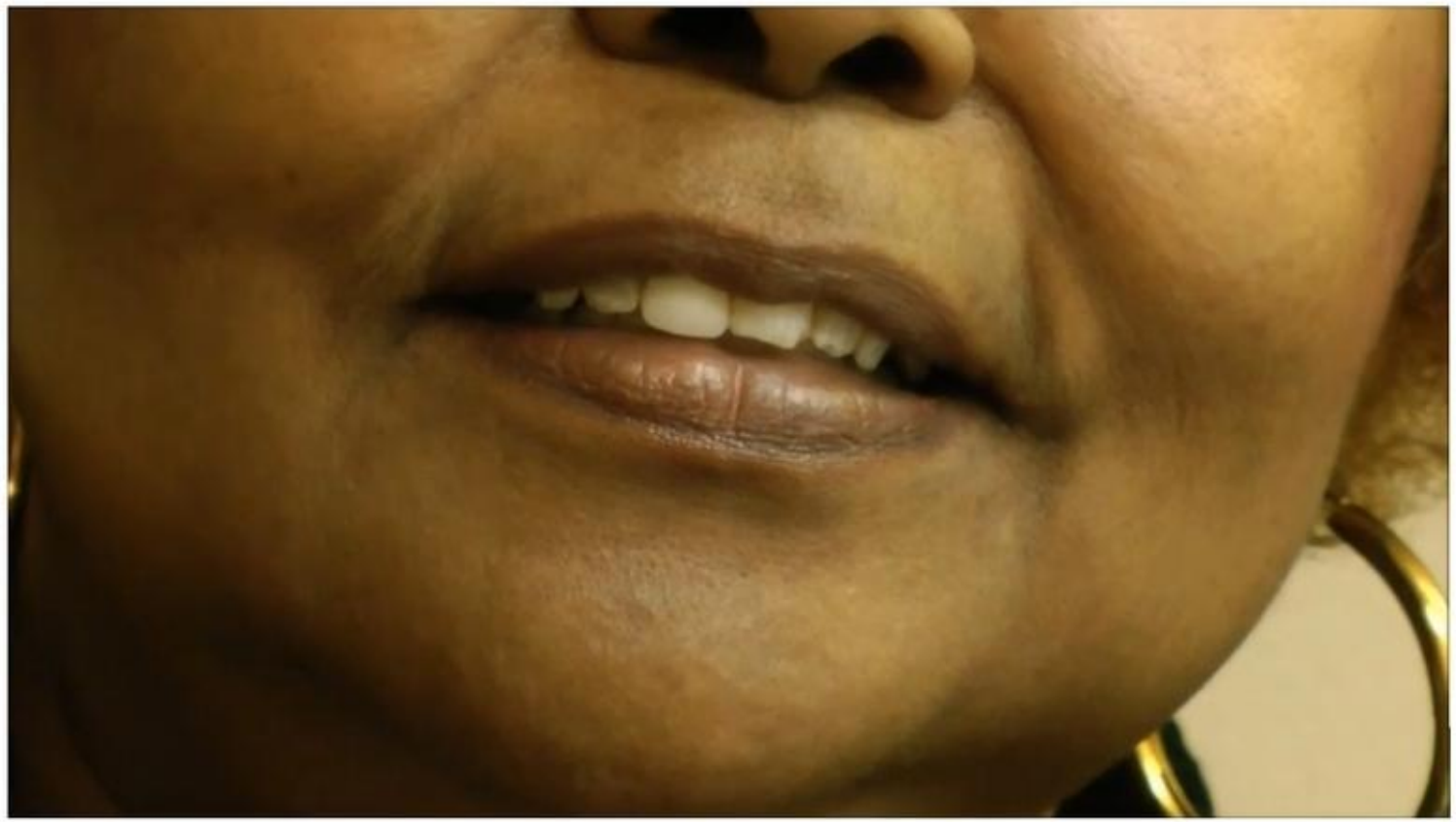
Dr. Tamara Pringsheim

University of Calgary (MDS Congress, Philadelphia, 2024)

Withdrawal emergent dyskinesia

- Restarted risperidone at 2.5 mg
- Tapered risperidone by 0.5 mg every three months
- Dyskinesia stopped immediately on resuming medication
- Did not re-emerge over course of tapering medication

Withdrawal Emergent Tardive Dyskinesia

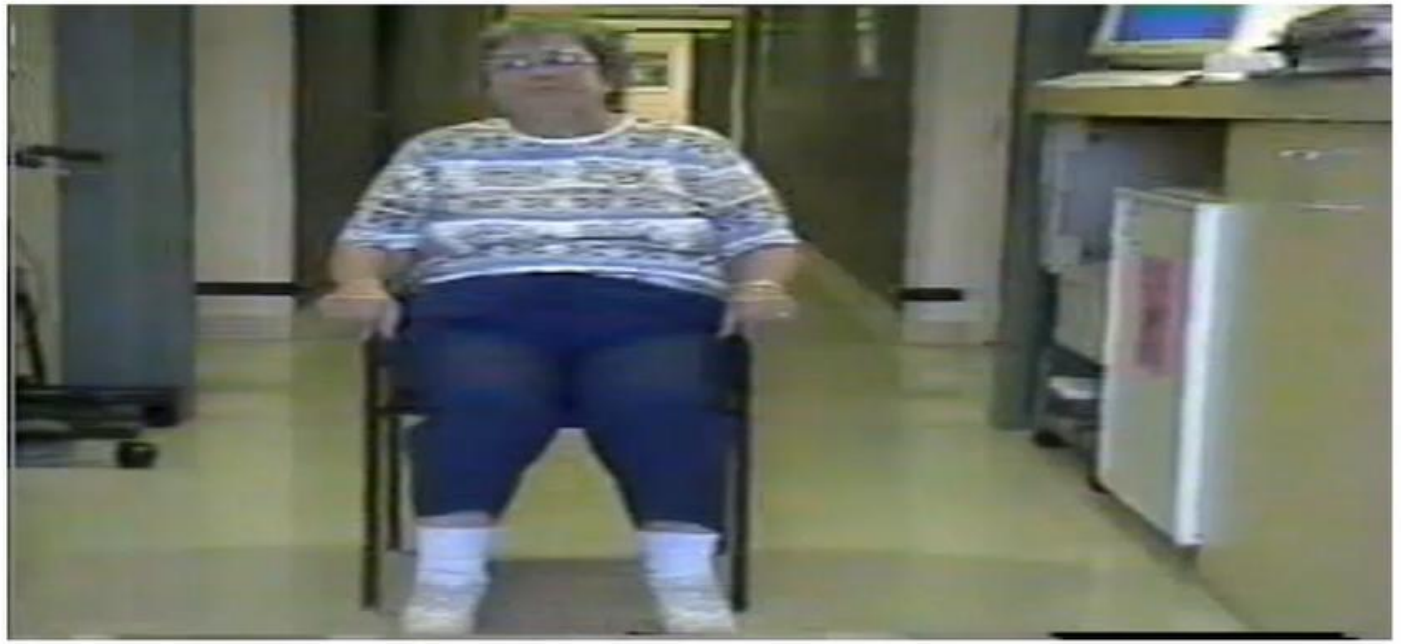


Withdrawal Emergent Tardive Dyskinesia

Tardive stereotypies

These are patterned, purposeless, repetitive and somewhat ritualistic movements that may appear as truncal rocking, pelvic thrusting, to-and-fro leg movements, hand-wringing or crossing/uncrossing of the legs. They may outwardly resemble akathisia but are not accompanied by inner restlessness.

Tardive stereotypy (continuous pelvic thrusting)
"Copulatory dyskinesia"



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Canadian Psychiatric Association
Association des psychiatres du Canada

Systematic Review

The Canadian Journal of Psychiatry / La Revue Canadienne de Psychiatrie
2019, Vol. 64(6) 388-399

DOI: 10.1177/0706743719828968
TheCJP.ca | LaRCP.ca
SAGE

Treatment Recommendations for Tardive Dyskinesia

Recommandations sur le traitement de la dyskinésie tardive

Lucia Ricciardi[1], Tamara Pringsheim[2], Thomas R.E. Barnes[3], Davide Martino[4], David Gardner[5], Gary Remington[6], Donald Addington[7], Francesca Morgante[1], Norman Poole[8], Alan Carson[9], and Mark Edwards[1]

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1. Taper and discontinue offending medication over weeks to months (if possible). Counsel regarding transient worsening of TD and that remission could take weeks to months.
2. Switch to clozapine or quetiapine
3. Discontinue anticholinergic medication
4. VMAT2 inhibitors – valbenazine, deutetrabenazine
5. Amantadine
6. Botulinum toxin injections
7. GPi DBS

The natural history of tardive dystonia
A long-term follow-up study of 107 cases

Vassilios Kiriakakis, Kailash P. Bhatia, Niall P. Quinn and C. David Marsden

University Department of Clinical Neurology, Institute of Neurology, Queen Square, London, UK

Correspondence to: Dr K. P. Bhatia, Department of Clinical Neurology, Institute of Neurology, Queen Square, London WC1N 3BG, UK

Remission: only 14% (follow up of 8 ½ years)

Discontinuation of neuroleptics ↑ chance of remission four times

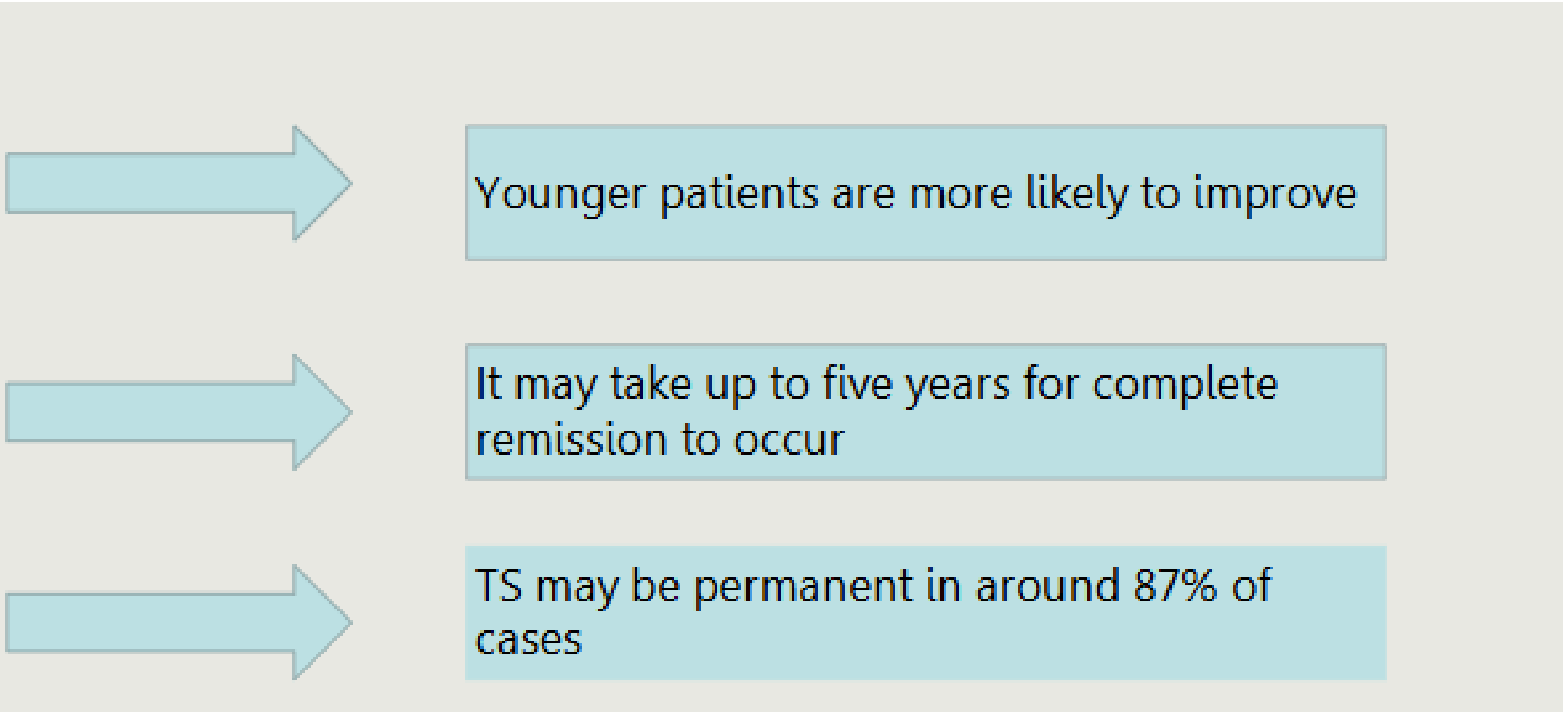
Likely irreversible in many

Table 9 *Remission rates of tardive dystonia reported in various studies*

Study	Patients (*n*)	Follow-up from onset (years)*	Follow-up from DRA withdrawal (years)*	Remitting patients (*n*)
Burke *et al.* (1982)	42 (9†)	3.1	1.5	5 (1‡)
Gimenez-Roldan *et al.* (1985)	9	4.7	n.s.	0
Kang *et al.* (1986)	67	~4.8	2.8	5
Gardos *et al.* (1987*a*)	10	~5.2	n.s.	0
Wojcik *et al.* (1991)	29	7.3	n.s.	0
Kiriakakis *et al.* (1997)	107	8.3	3.9	15

*Means. †Excluding 16 and 17 patients followed up later by Kang *et al.* (1986) and Kiriakakis *et al.* (1997), respectively. ‡Excluding one and three patients followed-up later by Kang *et al.* (1986) and Kiriakakis *et al.* (1997), respectively. Approximation (~) = not stated explicitly, but calculated by us; n.s. = not stated at all.

Treatment: Drug withdrawal



Bilateral Deep Brain Stimulation of the Globus Pallidus Internus in Tardive Dystonia

Wataru Sako, MD,[1] Satoshi Goto, MD, PhD,[1*]
Hideki Shimazu, MD, PhD,[1] Nagako Murase, MD, PhD,[1]
Kazuhito Matsuzaki, MD, PhD,[2] Tetsuya Tamura, MD,[2]
Hideo Mure, MD,[2] Yusuke Tomogane, MD, PhD,[3]
Norio Arita, MD, PhD,[3] Hiroo Yoshikawa, MD, PhD,[4]
Shinji Nagahiro, MD, PhD,[2] and Ryuji Kaji MD, PhD[1]

Movement Disorders, Vol. 23, No. 13, 2008

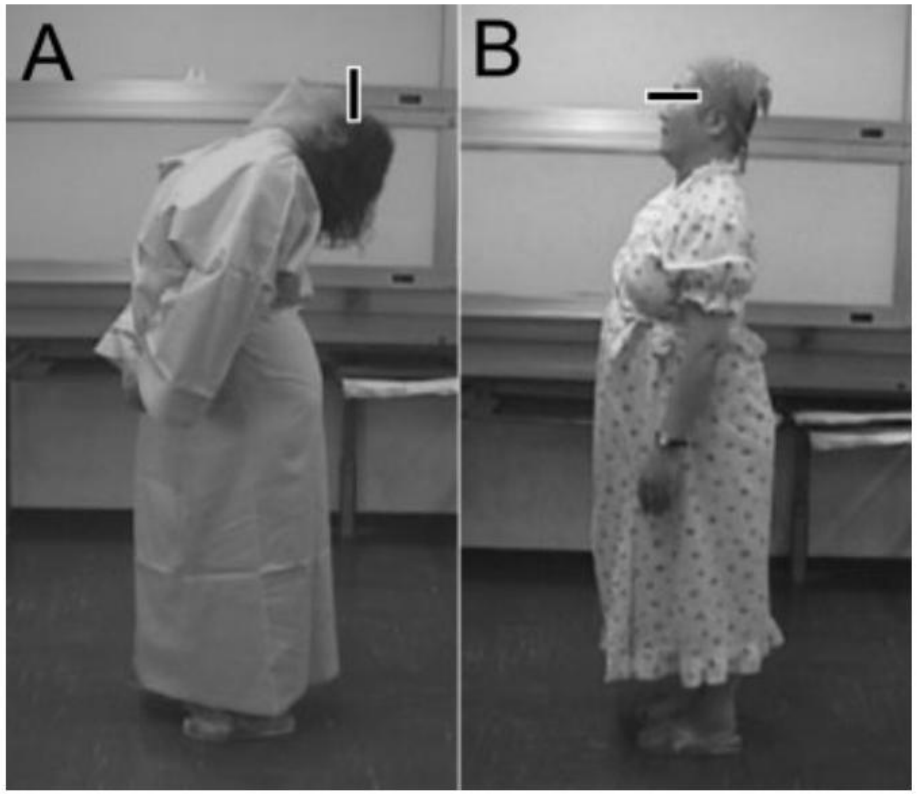


FIG. 1. Surgical result in a patient with tardive dystonia who underwent bilateral pallidal stimulation. Preoperatively (**A**) she manifested severe posterior truncal bending and retrocollis. At 21 days after electrode implantation with continuous pallidal stimulation (**B**), the dystonic symptoms were markedly alleviated.

Unilateral Deep Brain Stimulation of the Internal Globus Pallidus Alleviates Tardive Dyskinesia

Christoph Schrader, MD,[1*] Thomas Peschel, MD,[1]
Michael Petermeyer, MD,[2] Reinhard Dengler, MD,[1]
and Dieter Hellwig, MD[2]

[1]*Department of Neurology, Medizinische Hochschule Hannover, Hannover, Germany*
[2]*Department of Neurosurgery, Philipps-Universität, Marburg, Germany*

VIDEO SUPPLEMENT

3. Drug Induced Parkinsonism

Drug-induced parkinsonism

- 75M with Tourette syndrome and treatment resistant depression
- On risperidone 2 mg BID for decades
- Aripiprazole 20 mg added for treatment resistant depression

Potential offending drugs causing parkinsonism

- DRBDs
- Dopamine synthesis blockers (Methyldopa)
- Calcium channel antagonists
- Dopamine storage and transport inhibitors
- Antiepileptics (valproate)
- Lithium
- Chemotherapeutics (cytosine arabinoside, cyclophosphamide, vincristine, Adriamycin, doxorubicin, pacliataxel, etoposide)

Which of the following is considered the best treatment for patients with drug-induced parkinsonism?

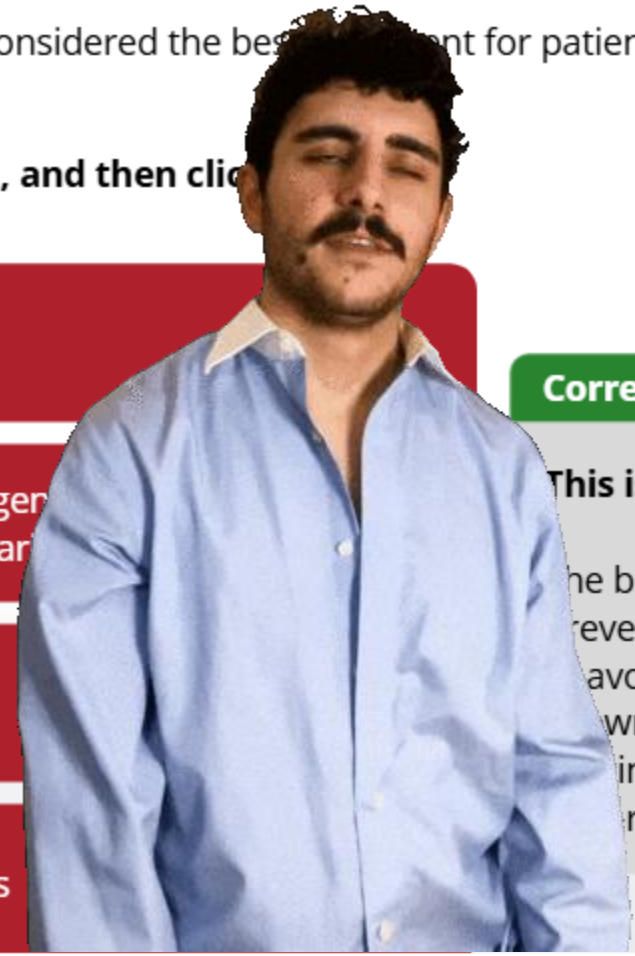
Select the correct option, and then click SUBMIT.

- Amantadine
- Avoidance of agents known to cause drug-induced parkinsonism
- Levodopa
- Anticholinergics

Which of the following is considered the bes nt for patients with drug-induced parkinsonism?

Select the correct option, and then clic

- Amantadine
- Avoidance of ager drug-induced par
- Levodopa
- Anticholinergics



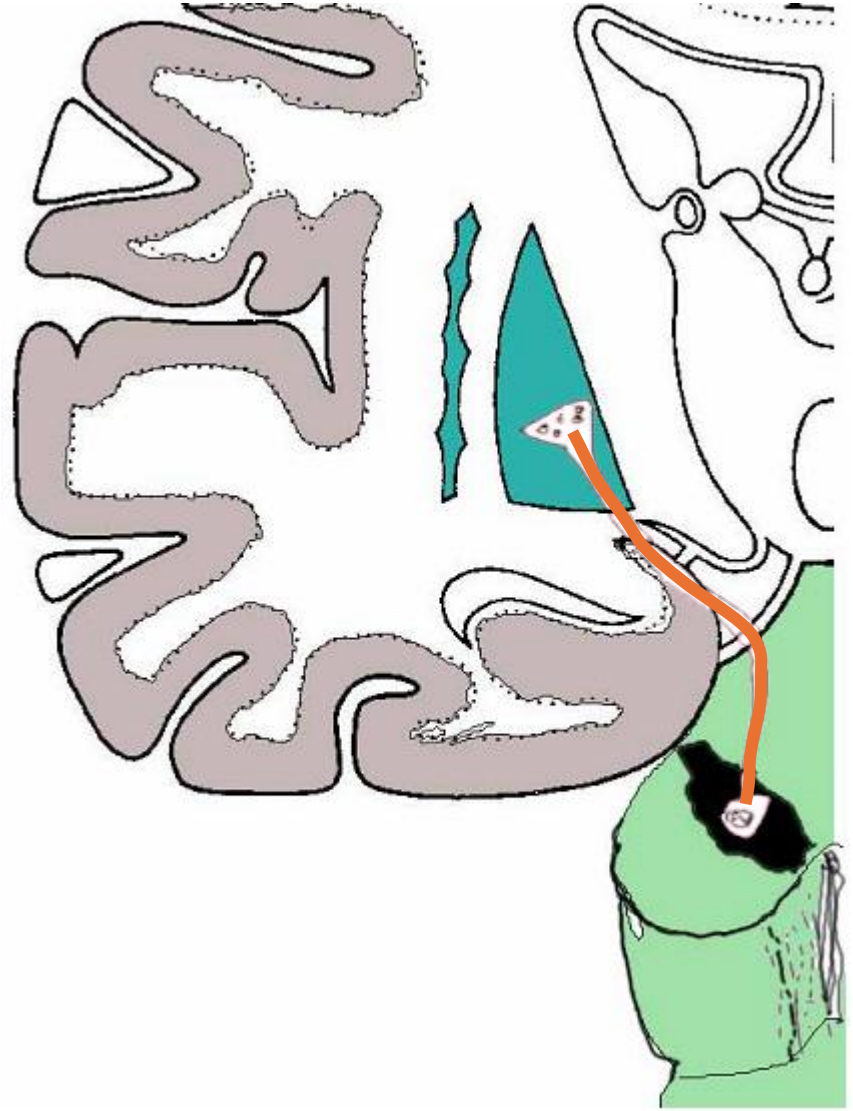
Correct

This is correct!

he best treatment is revention. It is most advisable avoid the use of medications wn to cause drug-induced insonism, especially in risk populations.

Treatment of Drug Induced Parkinsonism

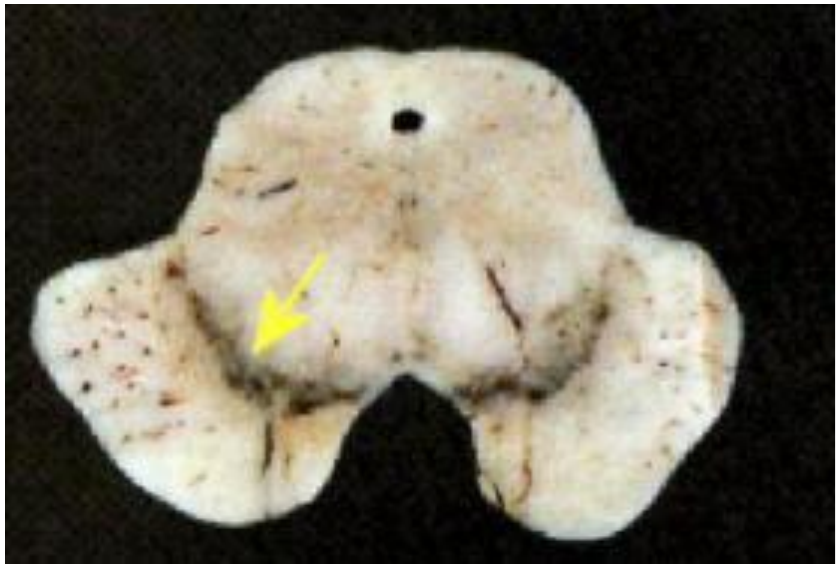
Striatum &
Basal Ganglia

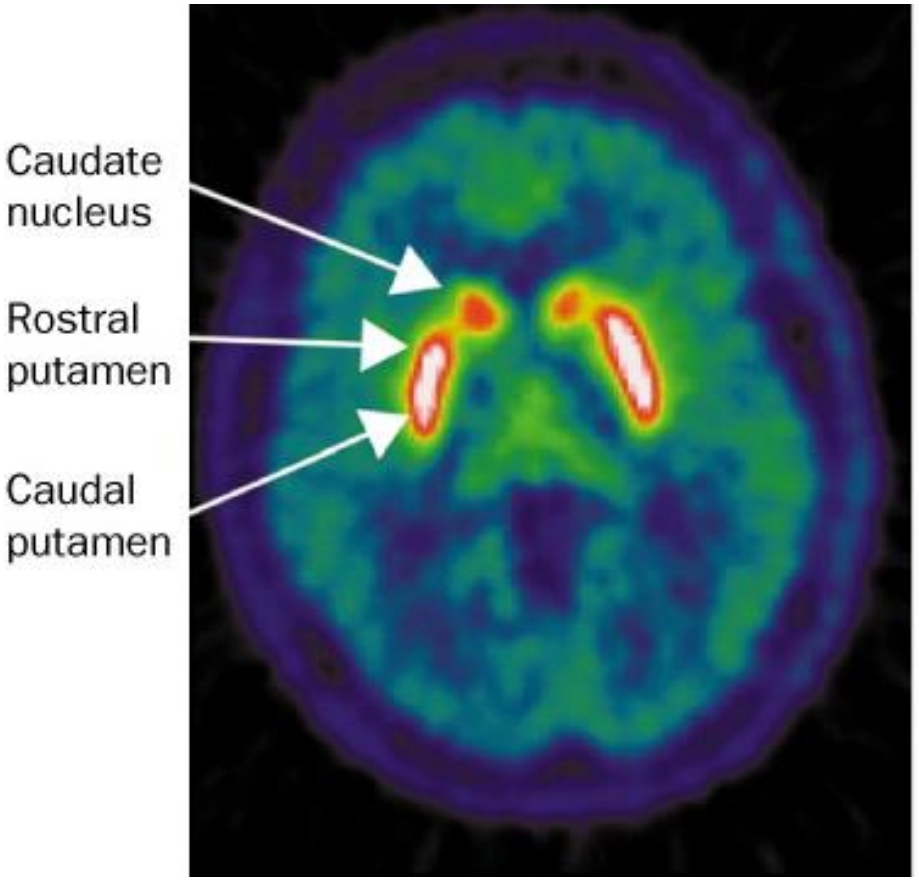


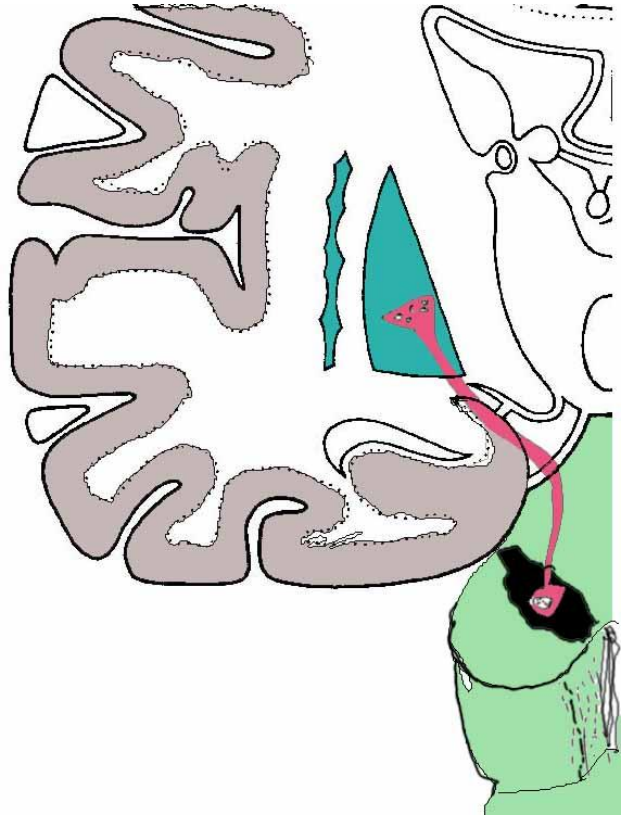
Dopaminergic
Neuron

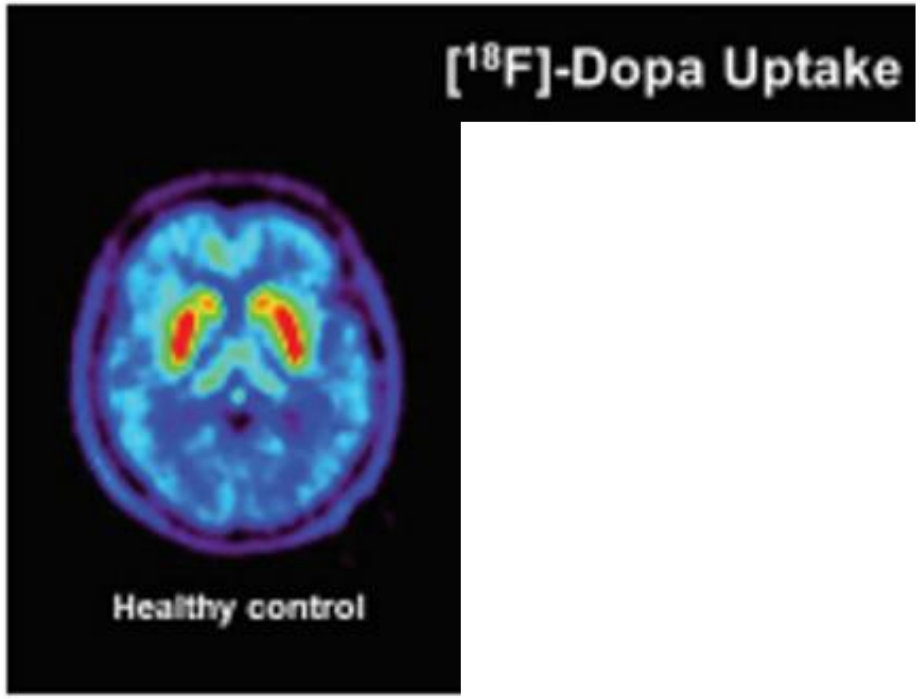
Midbrain, with
Substantia nigra

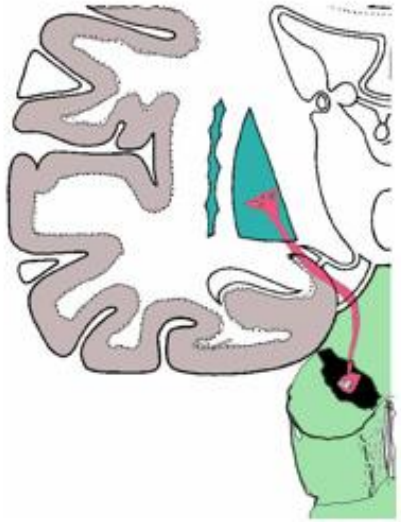
Substantia Nigra, where dopaminergic neurons have their cell bodies



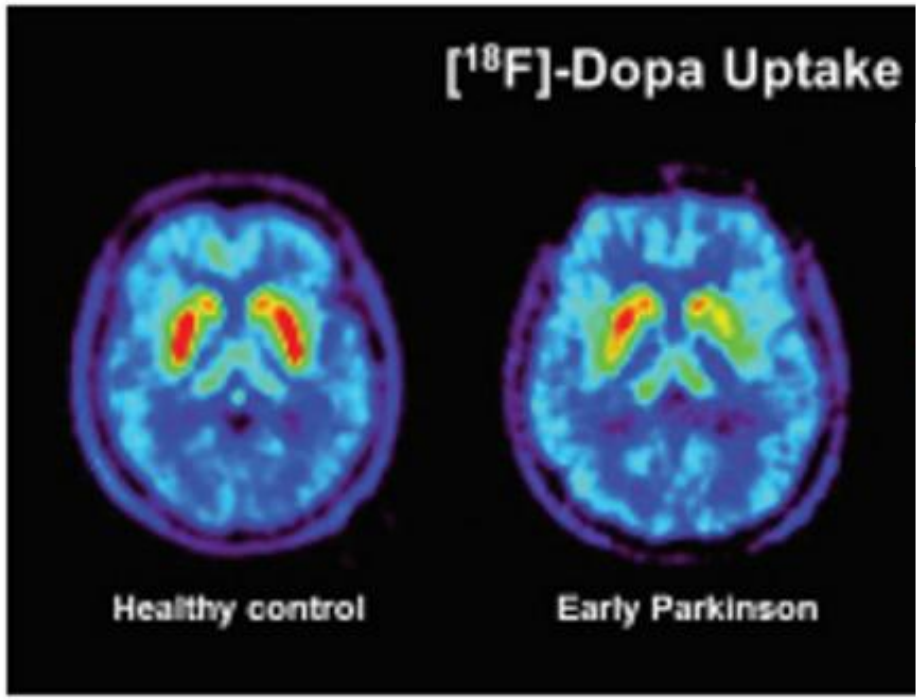

Caudate nucleus
Rostral putamen
Caudal putamen

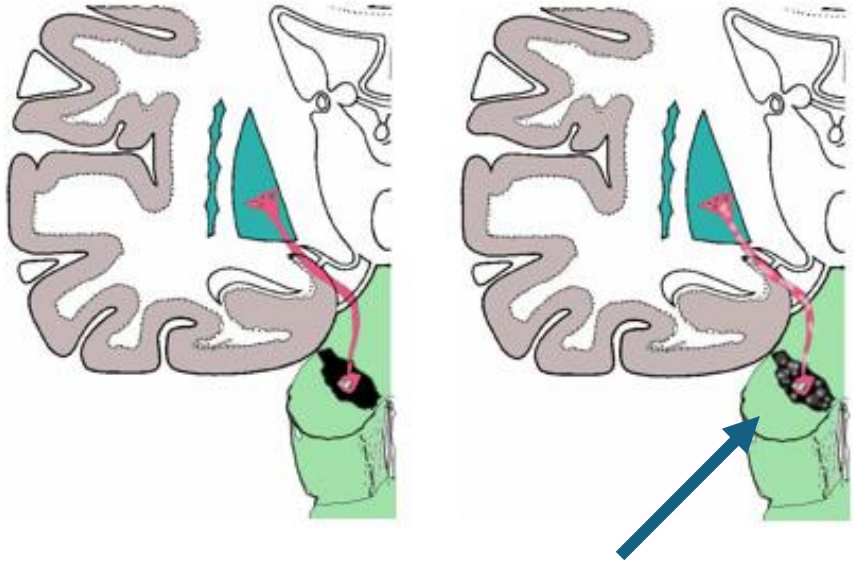






Booth TC . AJNR Am J Neuroradiol. 2015 Feb;36(2):229-35.





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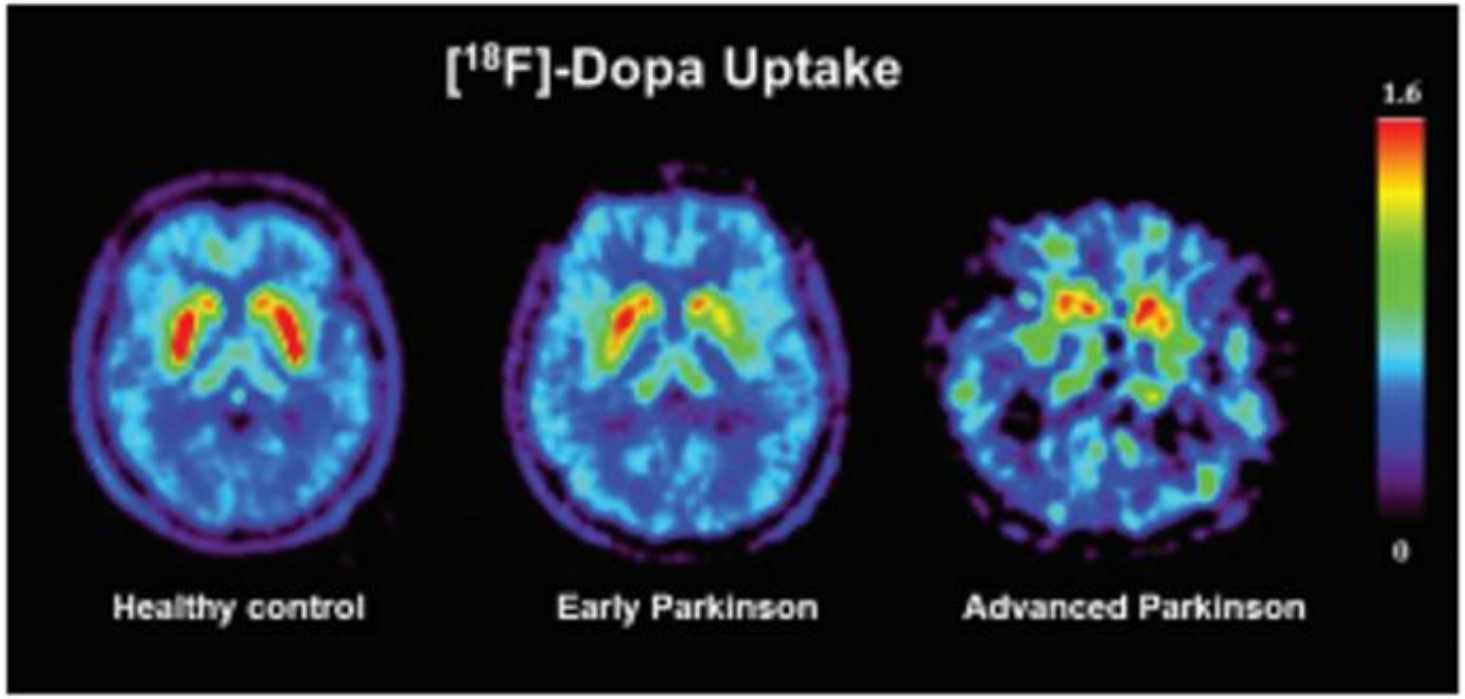
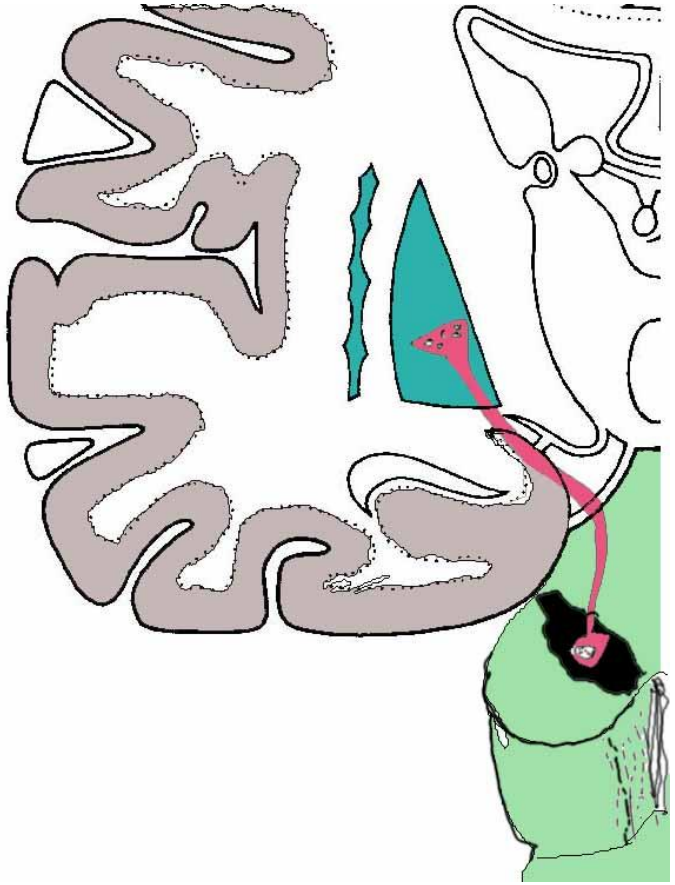
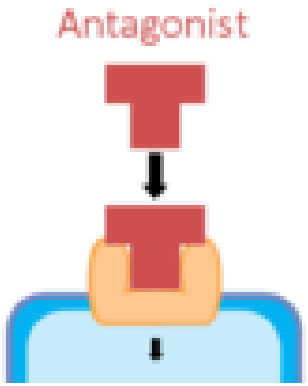
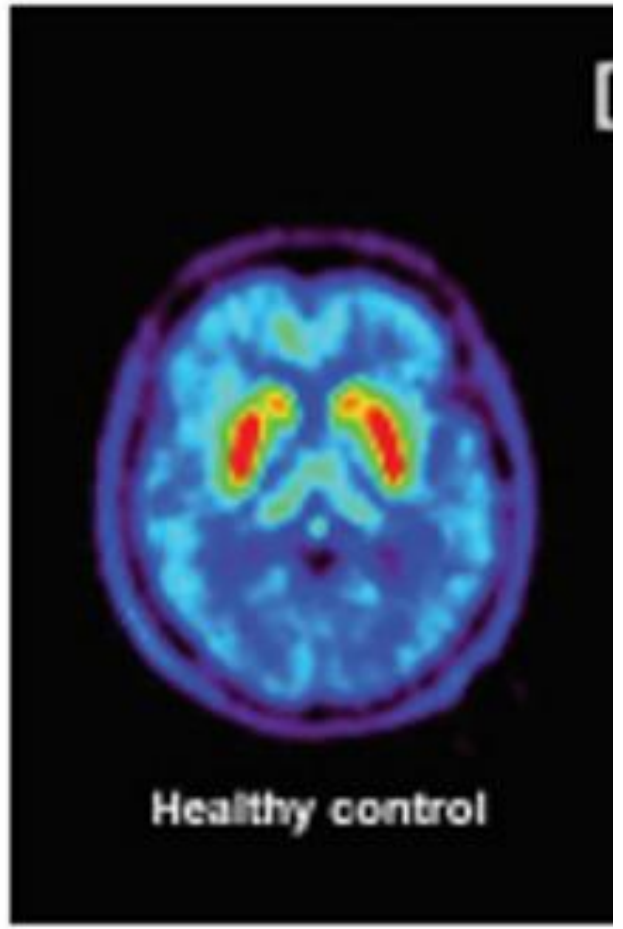


Booth TC . AJNR Am J Neuroradiol. 2015 Feb;36(2):229-35.

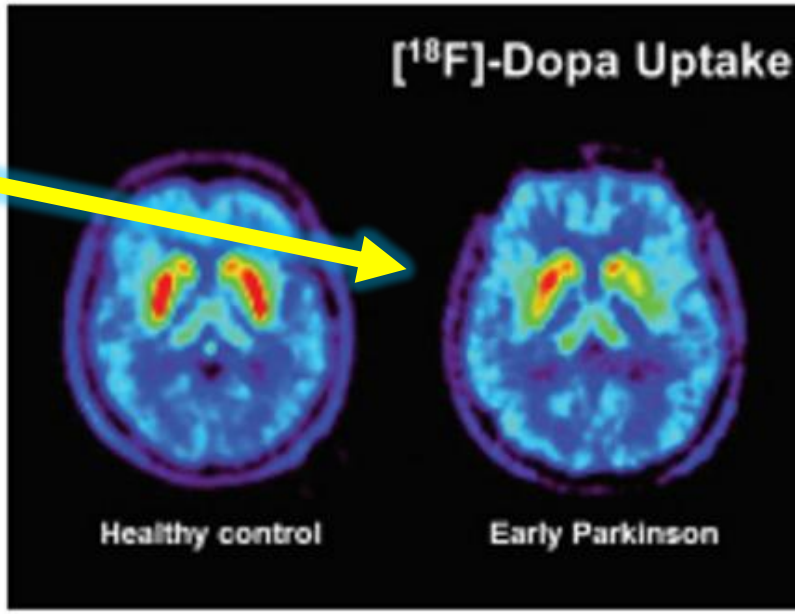
What about DiP?

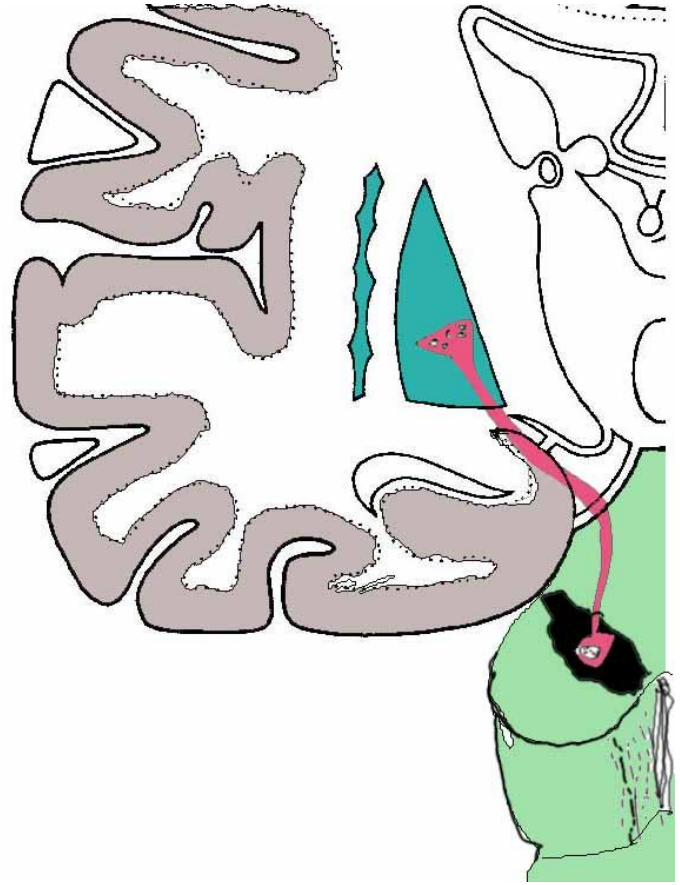


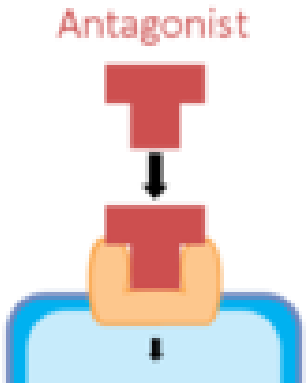


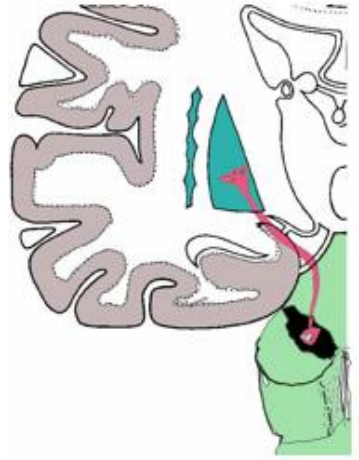


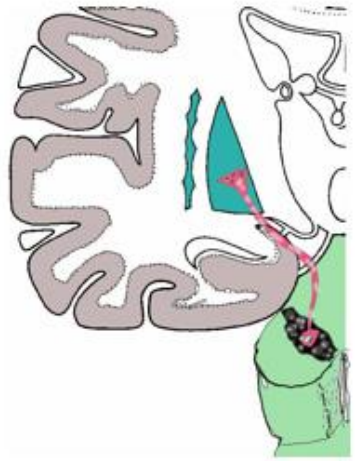
In fact, MANY cases look like this











VIEWPOINT

Parkinsonism Following Neuroleptic Exposure: A Double-Hit Hypothesis?

Roberto Erro, MD,[1,2*] Kailash P. Bhatia, MD, FRCP,[1] and Michele Tinazzi, MD, PhD[2]

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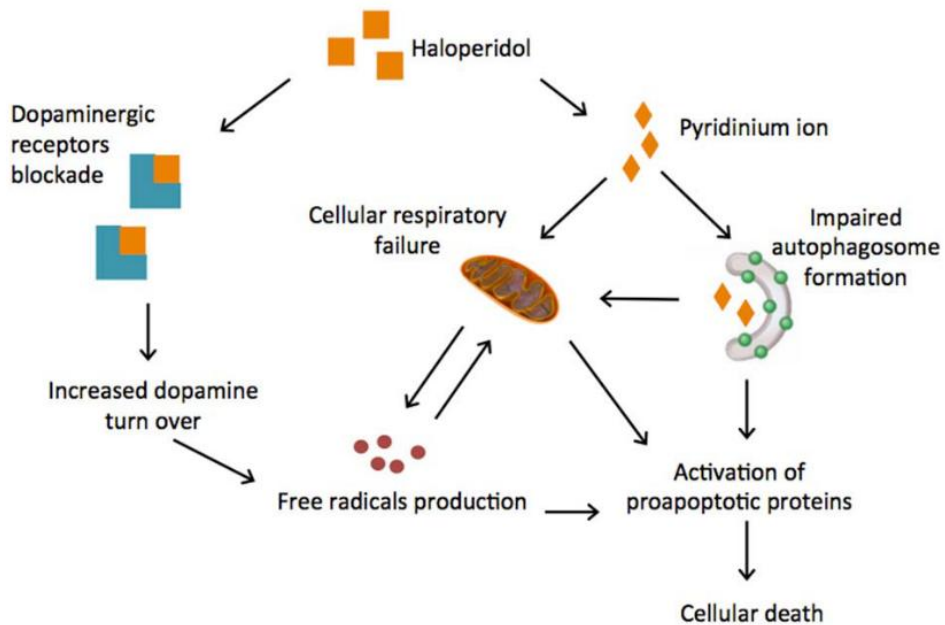


4. Akathisia

4. Akathisia

Acute Akathisia

- Akathisia= state of excessive restlessness with a need to move
- Untreated, can progress to chronic akathisia

Tardive Akathisia

- Inconsistently used term
- Delayed onset of akathisia after starting medication or increasing dose, typically after 3 months

Akathisia: commonest movement disorder due to Dopamine Receptor Blockade

Akathisia has a subjective and objective element.

Subjectively patients complain of unease, distress, dysphoria and inner restlessness, anxiety, restlessness in the legs that precede and later accompany the compulsion to move about. Feelings of fear and rage may also be reported.

Hum. Psychopharmacol Clin Exp 2008; 23: 15–26.

Akathisia: commonest movement disorder due to DRB

Objectively : global restlessness and increased, semi-purposeful motor activity, including the incapacity to stay still:

Observe repetitive movements of the legs and feet, often manifest as

Pacing

Marching in place

Rocking from foot to foot

If the patient is sitting they may stand and sit repeatedly or frequently shift their body position in the chair.

Moaning to relieve discomfort

Burkhard P

Acute and subacute drug-induced movement disorders

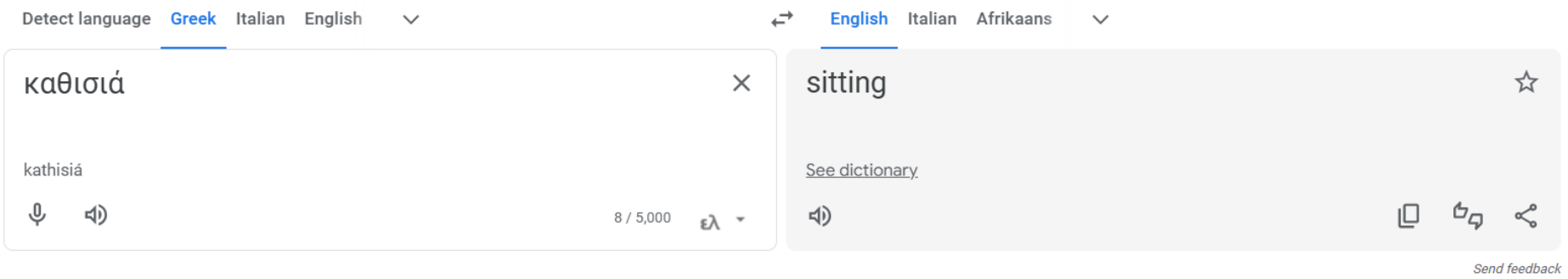
Akathisia

Dose dependent, and disappears if the dose is reduced or the drug is stopped.

If treatment is continued it commonly subsides after two to three months

Subjective discomfort appears to decline with time

Can occur in psychiatrically normal individuals when treated with neuroleptic drugs.



Ladislav Haškovec, Considered akathisia to be related to “hysteria” or “neurasthenia.”

Post encephalitic Parkinson’s

In 1947, the pharmaceutical company, Rhône-Poulenc, produced promethazine, a first generation antihistamine

…that year, drug-induced akathisia described in a patient with Parkinson’s, who developed restlessness

Akathisia

Movement Disorders
Official Journal of the International Parkinson and Movement Disorder Society

Article | Full Access

†Tardive akathisia: An analysis of clinical features and response to open therapeutic trials

Dr. Robert E. Burke, Un Jung Kang, Joseph Jankovic, Lucinda G. Miller, Stanley Fahn

First published: 1989 | https://doi.org/10.1002/mds.870040208 | Citations: 108

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Potential offending drugs causing akathisia

- Dopaminergic receptor blocking drugs (DRBDs)
- Dopamine storage and transport inhibitors
- Dopaminergic drugs
- Antidepressants

From Pandey et al, 2023, Drug-induced movement disorders

Table 3. Medications associated with akathisia

Drug class	Examples
Selective serotonin reuptake inhibitors	Fluoxetine,[21] paroxetine,[41] sertraline[10]
First-generation (typical) antipsychotics	Haloperidol,[3] perphenazine[28]
Antiemetics	Metoclopramide,[1] prochlorperazine[1]
Second-generation (atypical) antipsychotics	Risperidone[19]
Tricyclic antidepressants	Clomipramine[7]
Selective norepinephrine reuptake inhibitors	Venlafaxine[23]

Selective serotonin reuptake inhibitor–induced akathisia

Lindsey P. Koliscak and Eugene H. Makela

Akathisia in idiopathic Parkinson´s disease

(Lang and Johnson, Neurology 1987)

- 2/3 periodically experienced the need to move and inability to remain still
- 1/4 could not explain the inability to remain still: true akathisia

Impossible to drive long distances, sit through a movie, attend social gatherings

- Some greatly improved with levodopa.

RLS…sensory discomfort

An urge to move the legs, usually accompanied or caused by uncomfortable sensations in the legs.

> with rest or inactivity

Relieved by movement

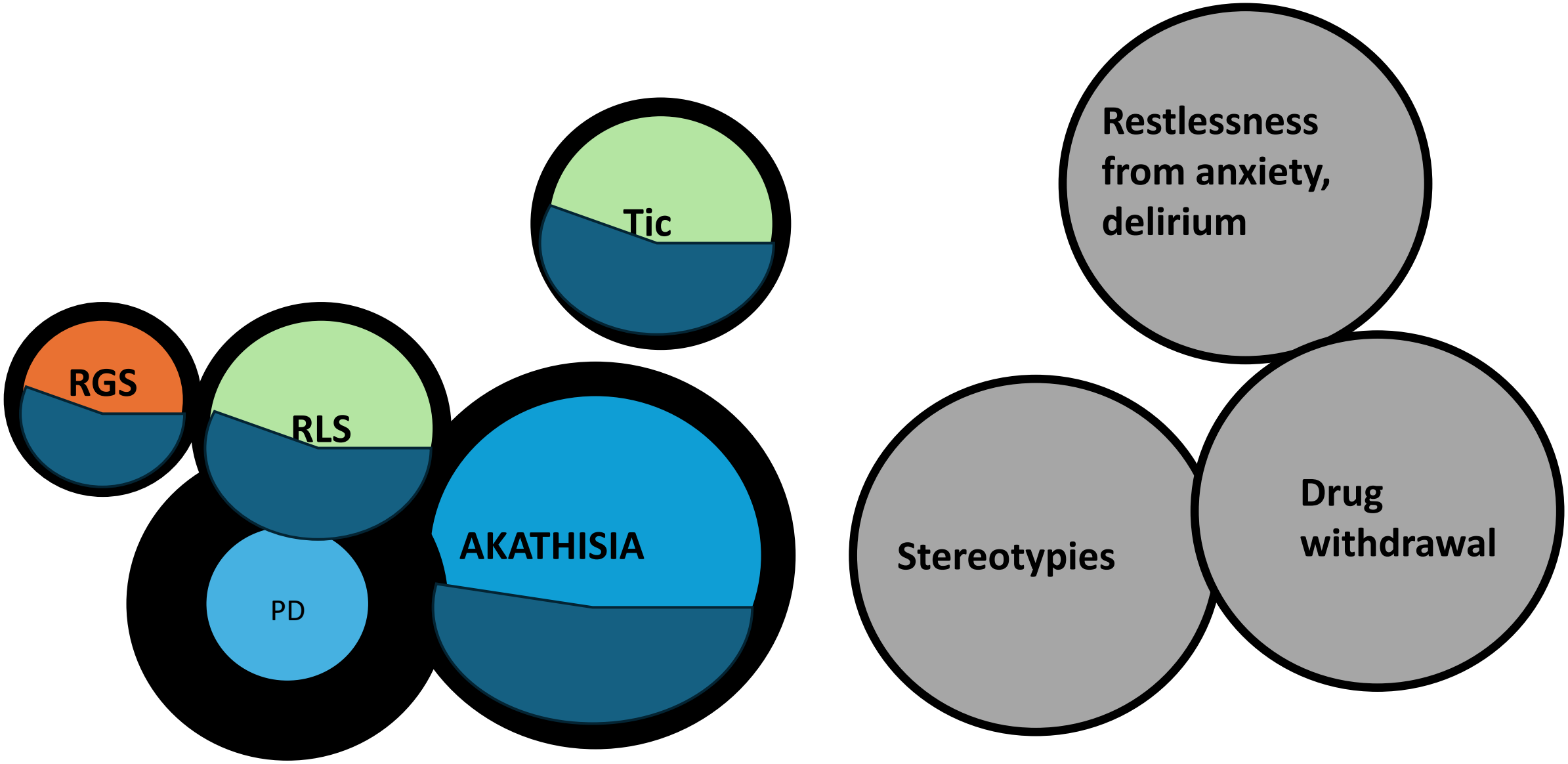
> evening or at night


Innsbruck Medical University
Dep. of Neurology

Opioid withdrawal

35- 50% reported transient symptoms of RLS during detoxification

Gupta R, Ali R, Ray R. Willis-Ekbom disease/restless legs syndrome in patients with opioid withdrawal. Sleep Med. 2018;45:39– 43. doi:10.1016/j.sleep.2017.09.028 16. McCarter SJ, Labott JR, Mazumder MK, et al. Emergence of restless legs syndrome during opioid discontinuation. J Clin Sleep Med. 2023;19(4):741–748. doi:10.5664/jcsm.10436


Tic
RGS
RLS
AKATHISIA
PD
Restlessness from anxiety, delirium
Stereotypies
Drug withdrawal

SLEEP, 2024, 47, 1–6
https://doi.org/10.1093/sleep/zsad273
Advance access publication 21 October 2023
Perspective

Perspective

Restless legs syndrome, neuroleptic-induced akathisia, and opioid-withdrawal restlessness: shared neuronal mechanisms?

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Treatment: acute akathisia

Reduce/discontinue antipsychotic

Lower potency agent

Clozapine/Quetiapine

Symptomatic:

Benzodiazepine

Calcium channel blocker: gabapentin (1200-3600 mg/day); pregabalin (300-600 mg/day);

Opioid

The Assessment and Treatment of Antipsychotic-Induced Akathisia

The Canadian Journal of Psychiatry / La Revue Canadienne de Psychiatrie
1-11

DOI: 10.1177/0706743718760288
TheCJP.ca | LaRCP.ca
SAGE

Tamara Pringsheim, MD[1], David Gardner, PharmD[2], Donald Addington, MD[3], Davide Martino, MD, PhD[4], Francesca Morgante, MD, PhD[5,6], Lucia Ricciardi, MD, PhD[7], Norman Poole, MD[8], Gary Remington, MD[9], Mark Edwards, MD[10], Alan Carson, MD[11], and Thomas R. E. Barnes, MD[12]

1. Prevention – avoid rapid escalation of antipsychotic dose
2. Avoid antipsychotic polypharmacy
3. Consider antipsychotic dose reduction
4. Switch to clozapine, olanzapine or quetiapine
5. Propranolol
6. Anticholinergic (if co-occurring drug-induced parkinsonism present)
7. Mirtazapine
8. Clonazepam
9. Clonidine

Treatment: ~~acute~~ chronic akathisia

Reduce/discontinue antipsychotic

Lower potency agent

Clozapine/Quetiapine

Symptomatic:

Benzodiazepine

Calcium channel blocker: gabapentin (1200-3600 mg/day); pregabalin (300-600 mg/day);

Opioid

Propranolol……..+ combinations/rotations

5. Drug induced tremor

5. Drug induced tremor

- Worth emphasizing that most tremor is postural/action.

- Rest tremor: virtually diagnostic of either PD, or DiP

Drug induced tremor= enhanced physiological tremor

Potential offending drugs causing tremor

- DRBDs (e.g., antipsychotics, metoclopramide)
- Antidepressants (e.g., SSRIs, TCA, mirtazapine)
- Lithium
- Anticonvulsant drugs (e.g., valproic acid)
- Beta-adrenergic agonists
- Theophylline
- Amphetamines
- Thyroxine
- Antihyperglycemic drugs
- Caffeine
- Corticosteroids
- Calcium antagonists (e.g., flunarizine, cinnarizine)
- Amiodarone
- Chemotherapeutics (e.g., cytarabine, thalidomide)
- Immunosuppressants (e.g., cyclosporine)

From Pandey et al, 2023, Drug-induced movement disorders

Treatment

Beta-blocker

Acetazolamide

Tetrabenazine (for TD tremor)

DBS


LOONEY TUNES