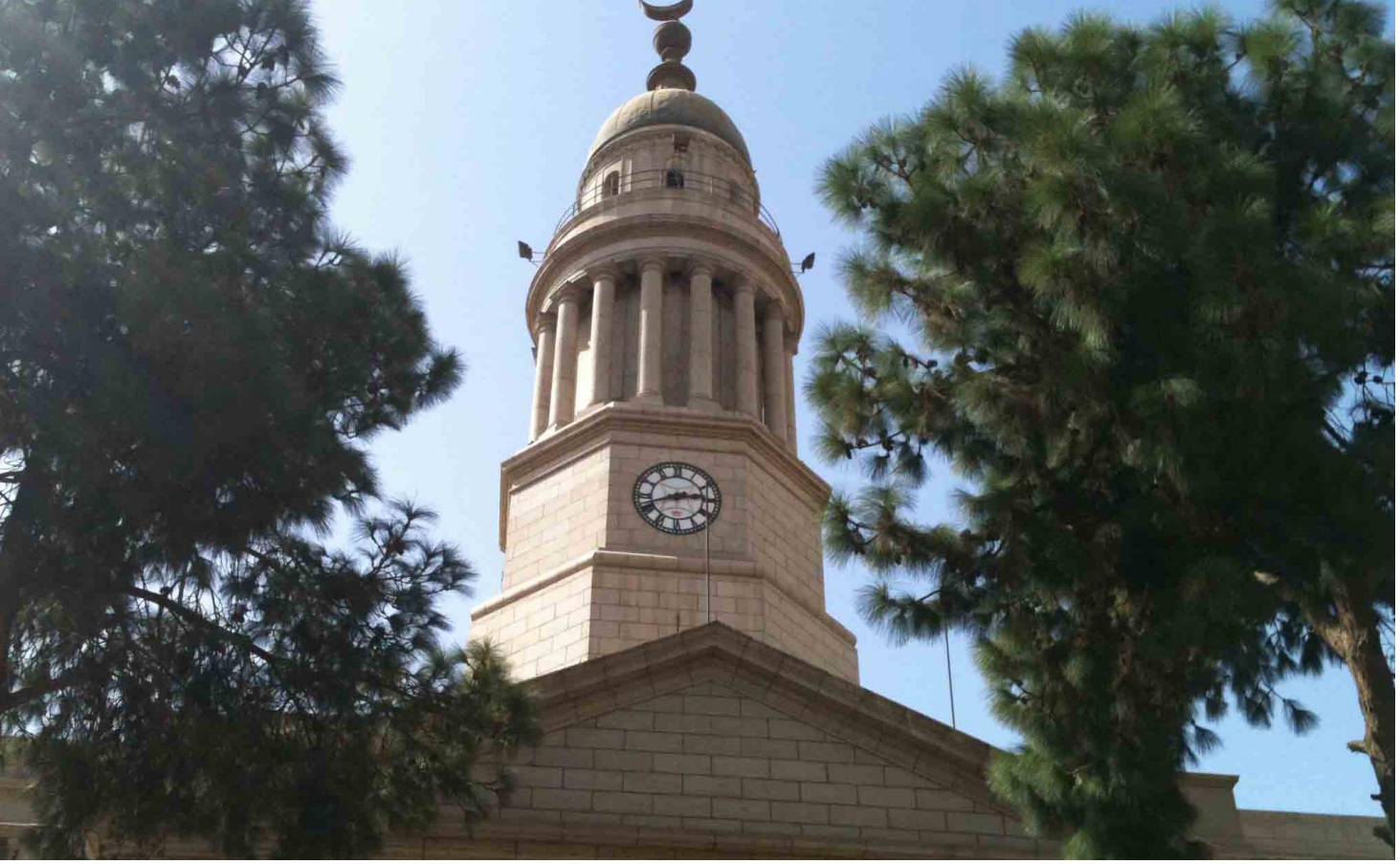

Tics and dyskinesias

Amr Hassan, MD,FEBN

Associate professor of Neurology

Cairo University

Any approach begins with . . .

A good history

A good physical exam

Keen sense of observation

A systematic differential diagnosis

Clinical assessment of AIM

- **D**escribe the movement
- **D**ifferentiate from other MD
- **D**istribution
- **D**ecreased by , Increased by
- **D**iurnal variation
- **D**uration
- **D**istinguished phenomenon

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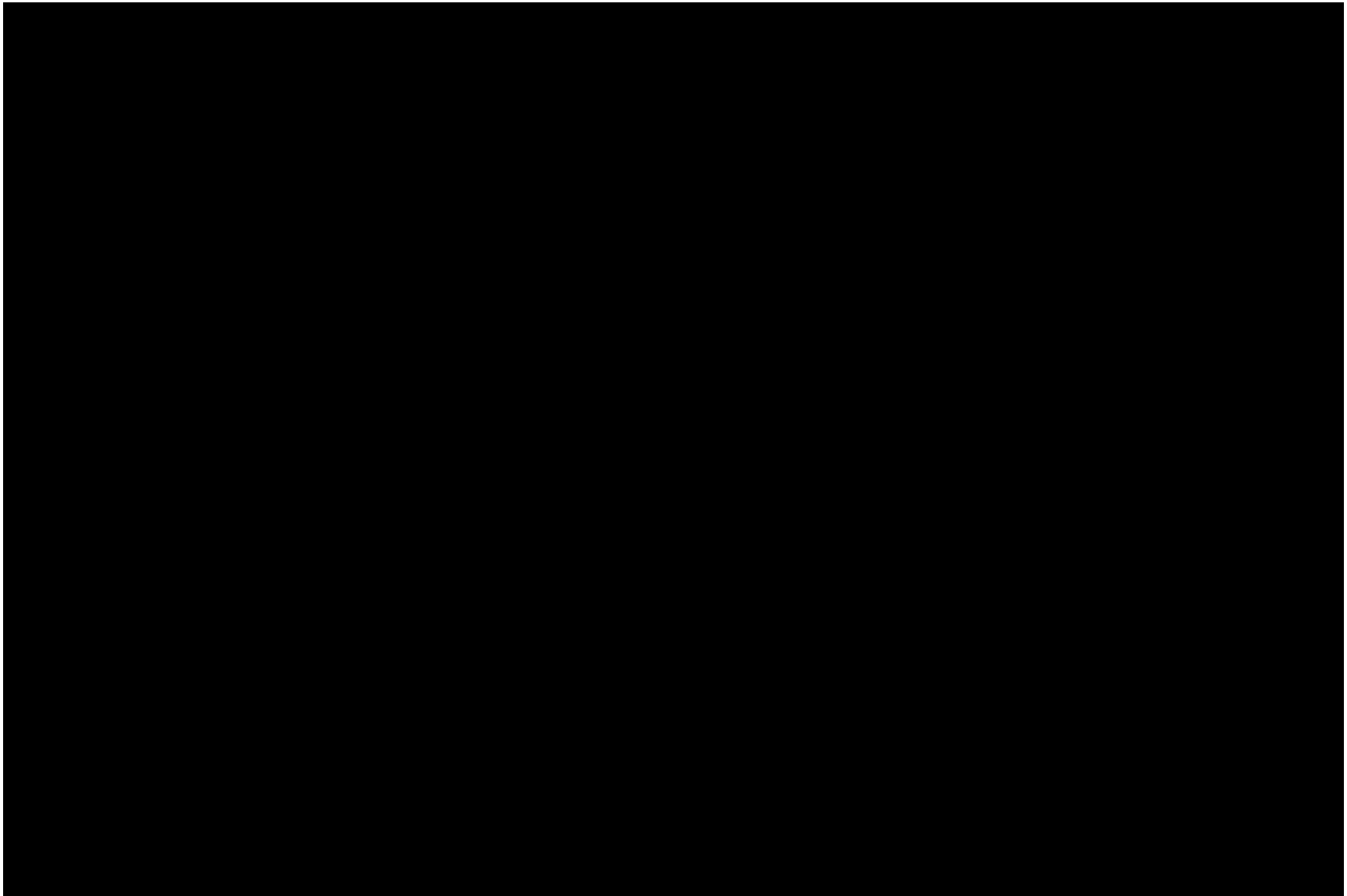
Describe the movement

- Rhythmic vs. arrhythmic
- Sustained vs. nonsustained
- Paroxysmal vs. Nonparoxysmal
- Slow vs. fast
- Amplitude
- At rest vs. Action Vs Task Specific
- Patterned vs. non-patterned
- Combination of varieties of movements
- Supressibility

Describe the movement



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Paroxysmal vs. Nonparoxysmal

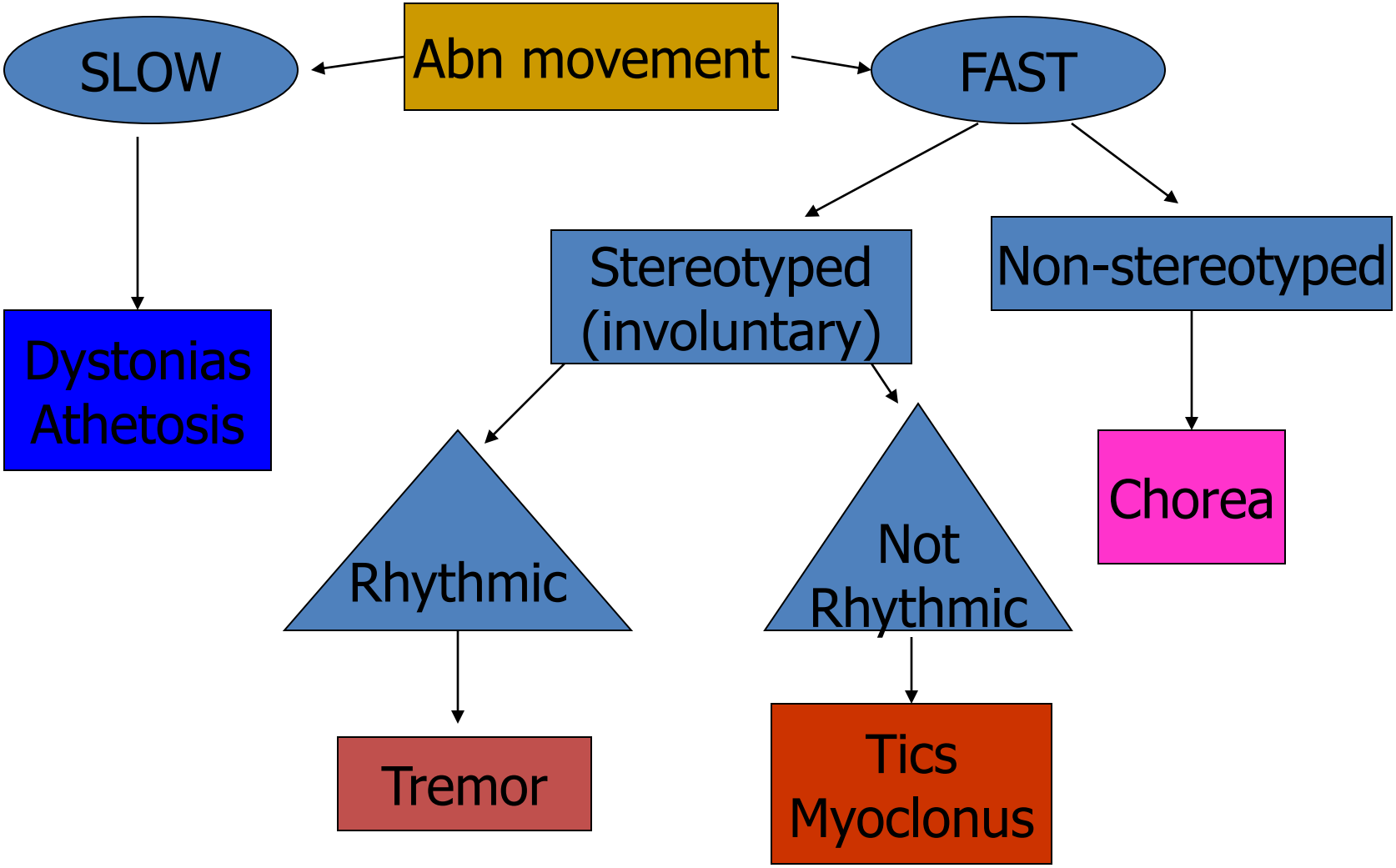
Paroxysmal

- Tics
- PKD
- PNKD
- Sterotypies
- Akathic movements
- Moving toes
- Myorhythmia

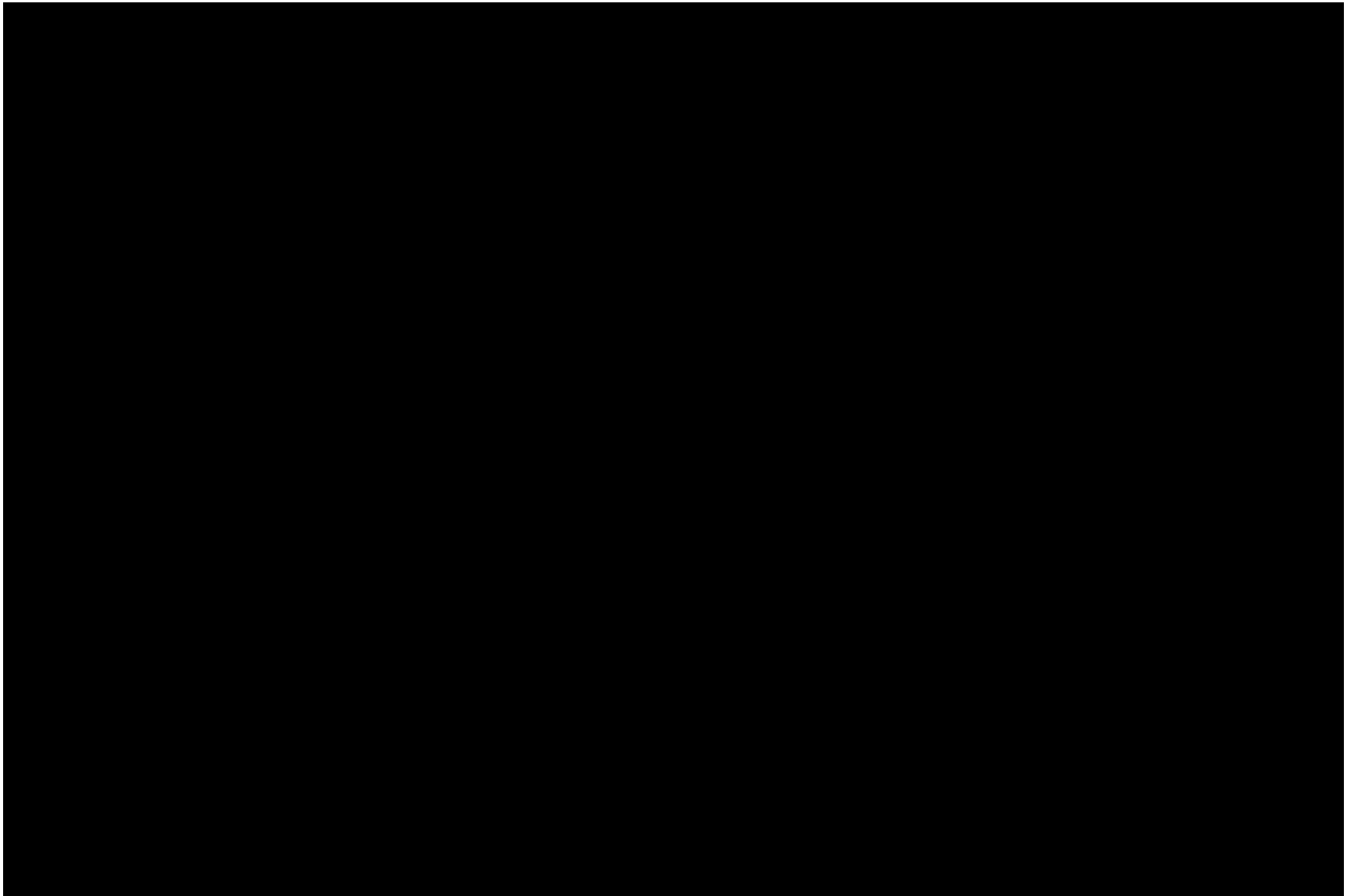
Continous

- Abdominal dyskinesias
- Athetosis
- Tremors
- Dystonic postures
- Myoclonus (rhythmic)
- Tardive sterotypy
- Myokymia
- Tic status

Differentiating from other MD



Describe the movement



What is a tic?

A “tic” is a sudden, rapid, recurrent, non-rhythmic, stereotyped motor movement or vocalization

May appear as exaggerated fragments of ordinary motor or phonic behaviors that occur out of context

Classification of tics

Simple

few muscles

Complex

multiple groups of muscles

Simple

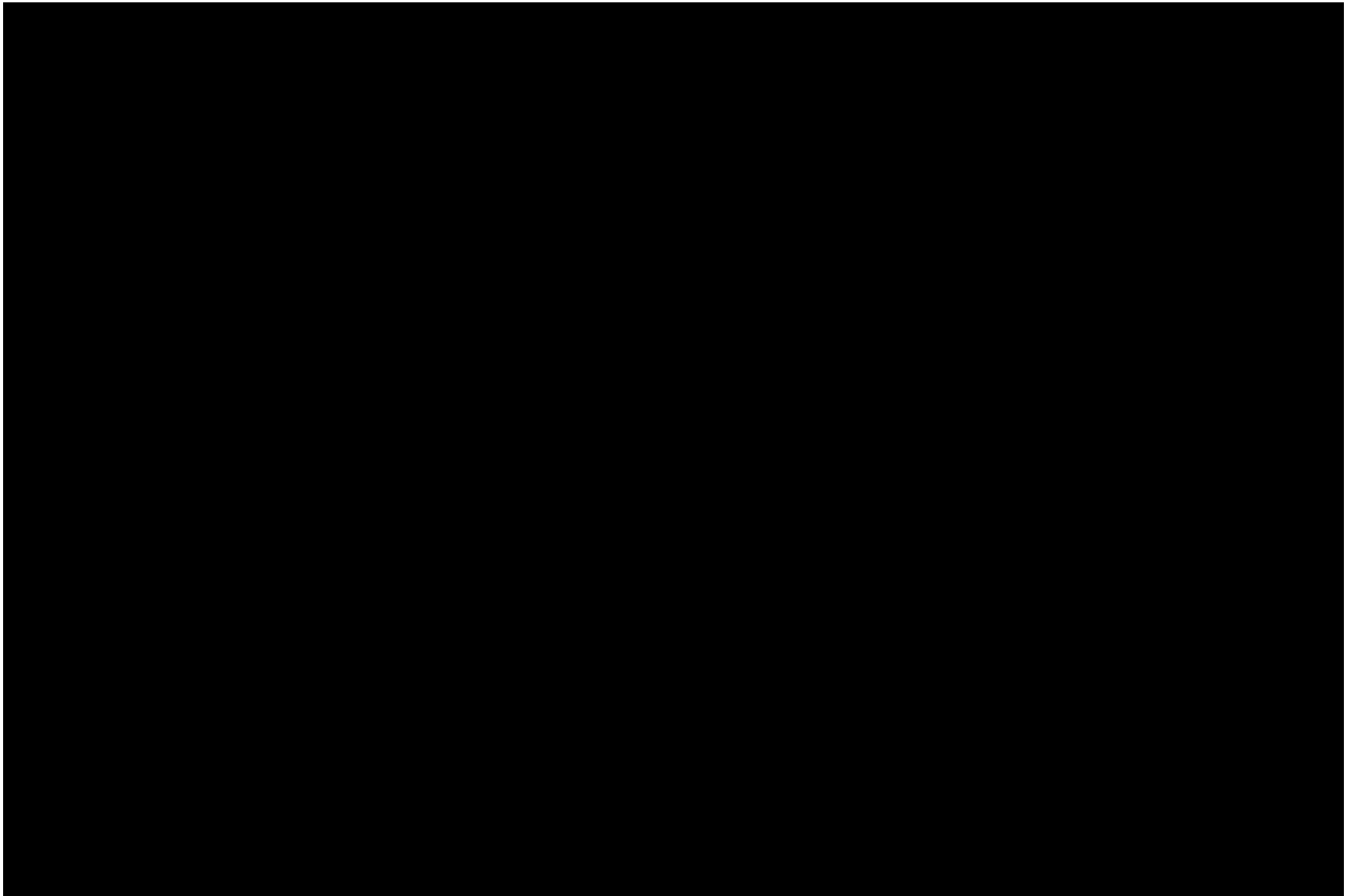
sounds

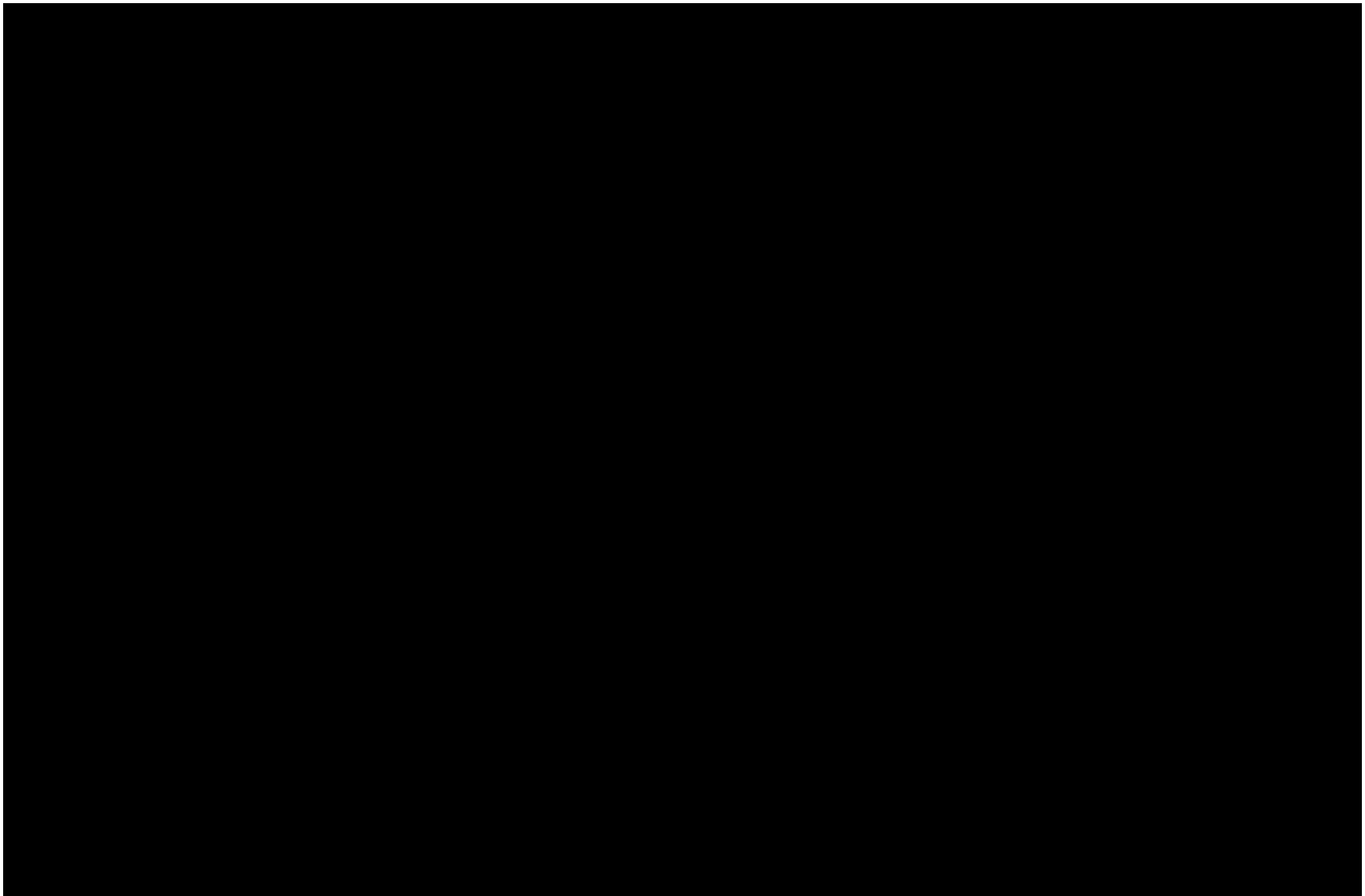
Complex

words or sentences

Motor Tics (Simple)

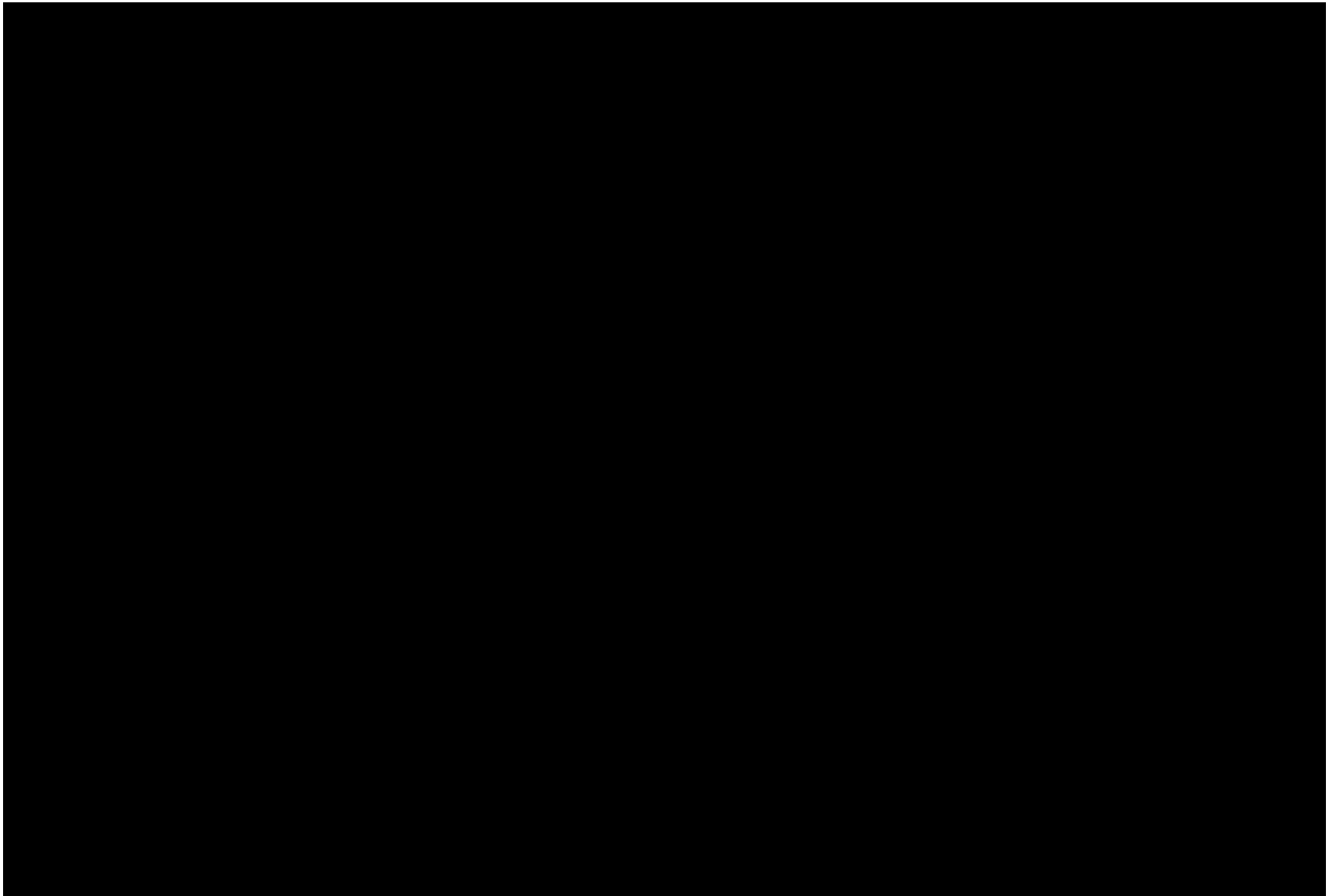
- Generally lasting less than several hundred milliseconds
- Examples include:
  - eye blinking
  - nose wrinkling
  - neck jerking
  - shoulder shrugging
  - facial grimacing
  - abdominal tensing





Motor Tics (Complex)

- Longer in duration than simple tics; usually lasting seconds or longer
- Examples include:
  - hand gestures
  - jumping, touching, pressing, or stomping
  - facial contortions
  - repeatedly smelling an object
  - squatting and/or deep knee bends
  - retracing steps and/or twirling when walking
  - assuming and holding unusual positions (including "dystonic" tics, such as holding the neck in a particular tensed position)

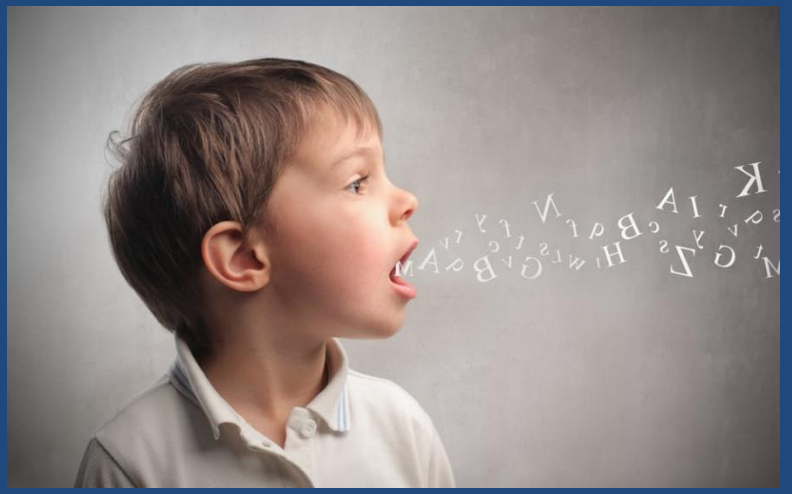


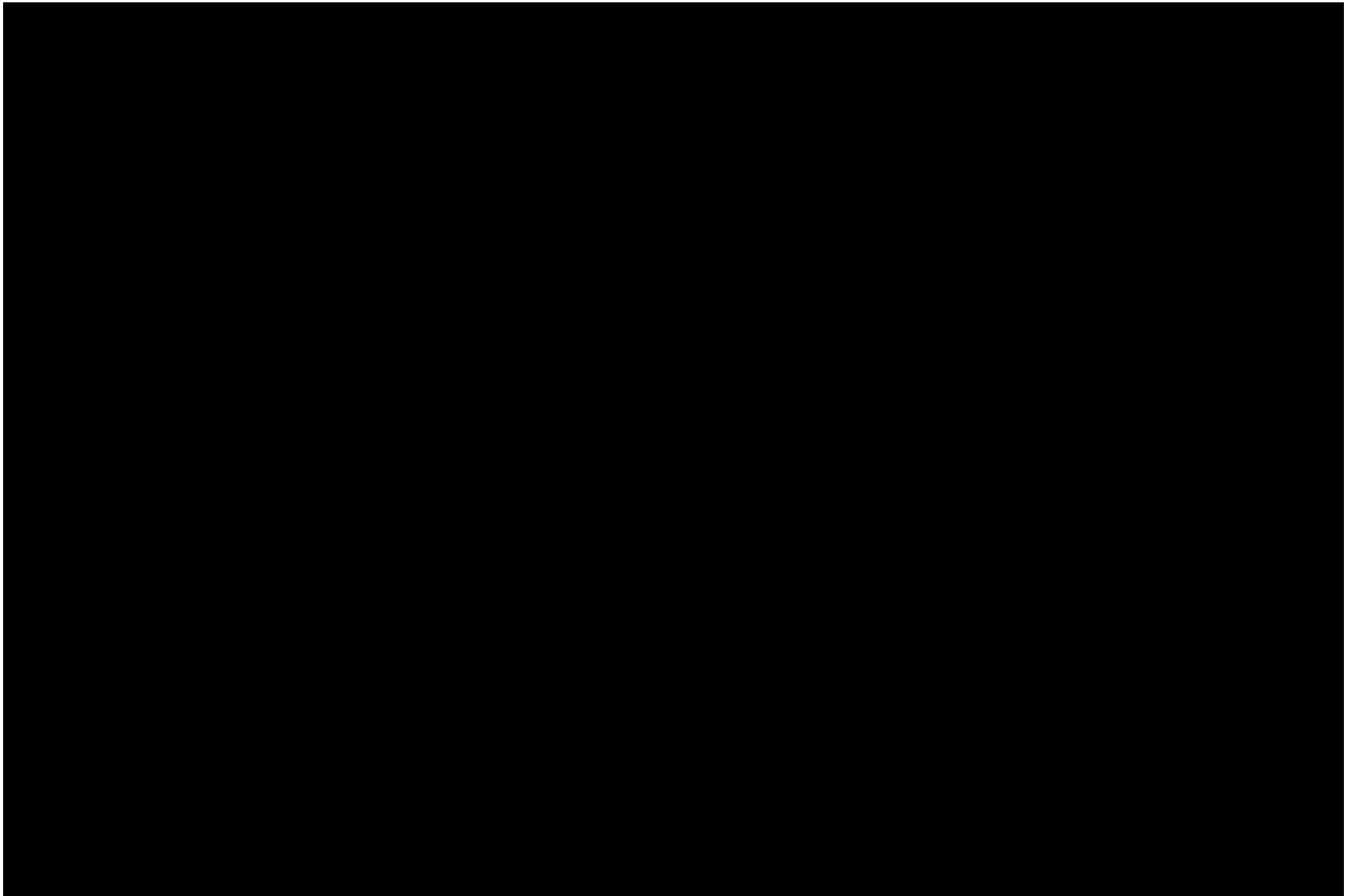
Copropraxia & Echopraxia

- Both are considered complex motor tics
- Copopraxia = a sudden, tic-like vulgar, sexual, or obscene gesture
- Echopraxia = a mirror phenomena, such as involuntary, spontaneous imitation of someone else's movements

Vocal Tics (Simple)

- Meaningless brief sounds
- Examples include:
  - Throat clearing
  - Grunting
  - Sniffing
  - Snorting
  - Chirping





The "Lalias"

- **All are considered complex vocal tics**
- **Palilalia** = repeating one's own sounds or words
- **Echolalia** = repeating the last heard sound, word, or phrase
- **Coprolalia** = the sudden, inappropriate expression of a socially unacceptable word or phrase that may include obscenities as well as specific ethnic, racial, or religious slurs (found in fewer than 10% of individuals with tic disorders)

	Simple	Complex
Motor tics	Eye blinking Nose wrinkling Jaw thrusting Shoulder shrugging Wrist snapping Neck jerking Limb jerking Abdominal tensing	Hand gestures Facial contortions Jumping Touching Repeatedly smelling object Squatting Copropraxia Echopraxia
Phonic tics	Sniffing Barking Grunting Throat clearing Coughing Chirping Screaming	Single words or phrases Partial words or syllables Repeated use of word or words out of context Palilalia Echolalia Coprolalia

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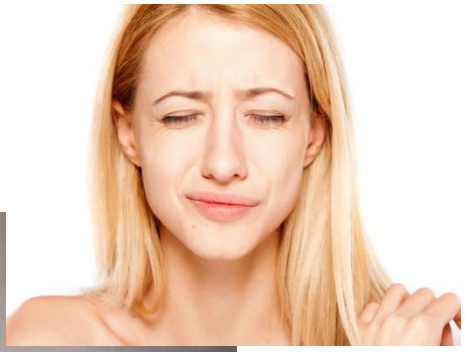
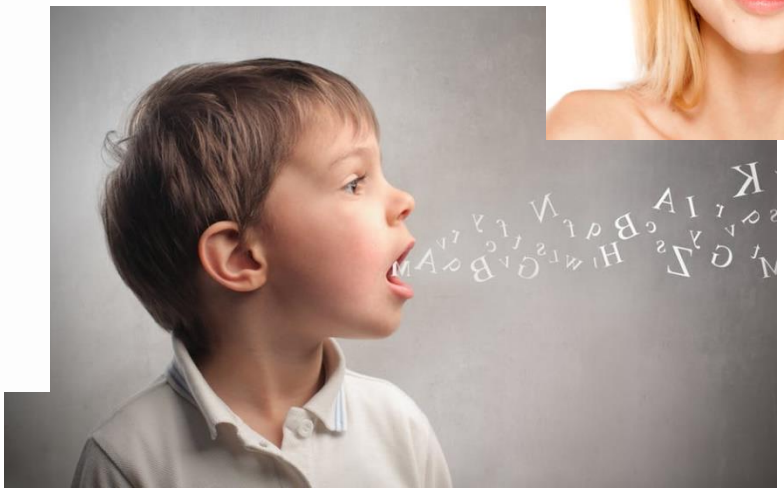
Distribution

- Predominate in the face, upper arms and neck.





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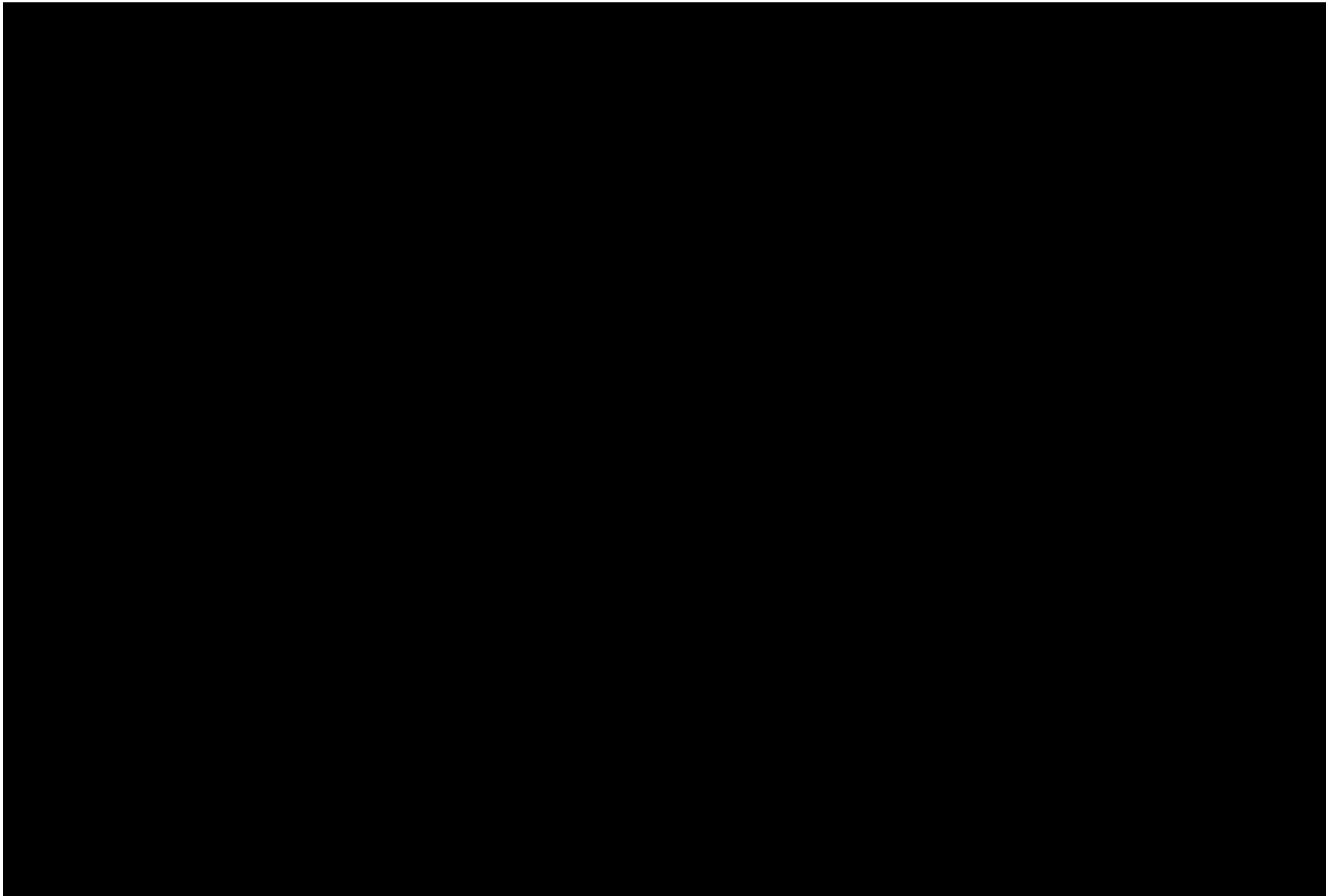
Decreased by , Increased by

- Aggravated by stress / anxiety.
- Often occur while relaxing, and may increase during relaxation after stress.

Decreased by , Increased by

- Tics are often more frequent when an individual relaxes in private (e.g., watching TV).
- Less frequent when an individual engages in directed, effortful activity (e.g., reading).



Decreased by , Increased by

- May persist during all sleep stages, but not common during sleep.

Decreased by , Increased by

May diminish in situations where might be embarrassing, including doctor's visits

Clinical assessment of AIM

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Diurnal variation

- Tics generally change in severity over the course of a day and may change in location over time

- Tics may vary in their frequency and disruptivity depending upon the circumstance (e.g., school, home, work, etc.)

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Duration

- Tics are often emitted in bouts of one or several tics, separated by periods without tics lasting seconds to hours

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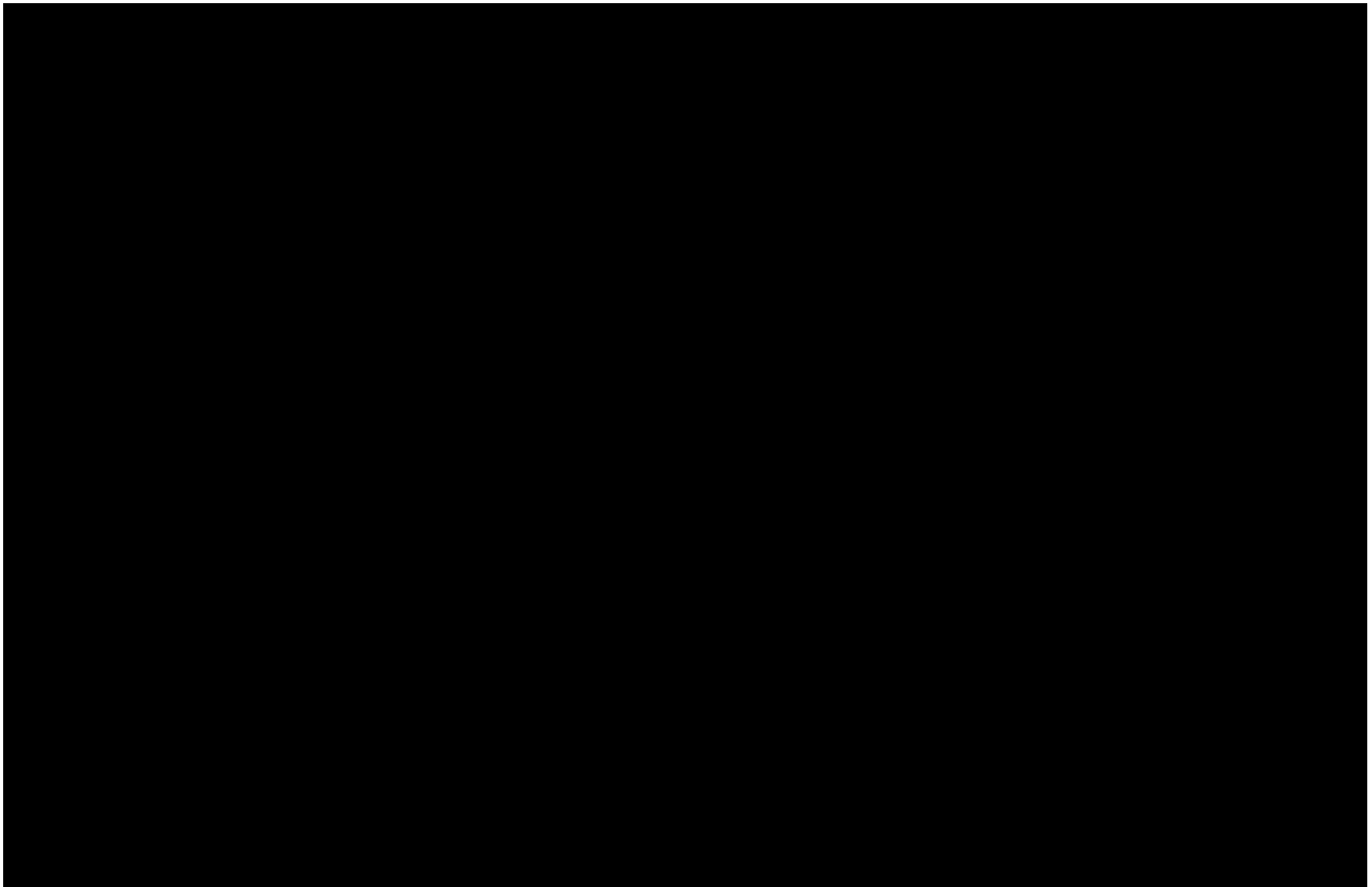
Distinguished phenomenon

- Irresistible but suppressible.
- Suggestibility in some individuals

Distinguished phenomenon



Distinguished phenomenon

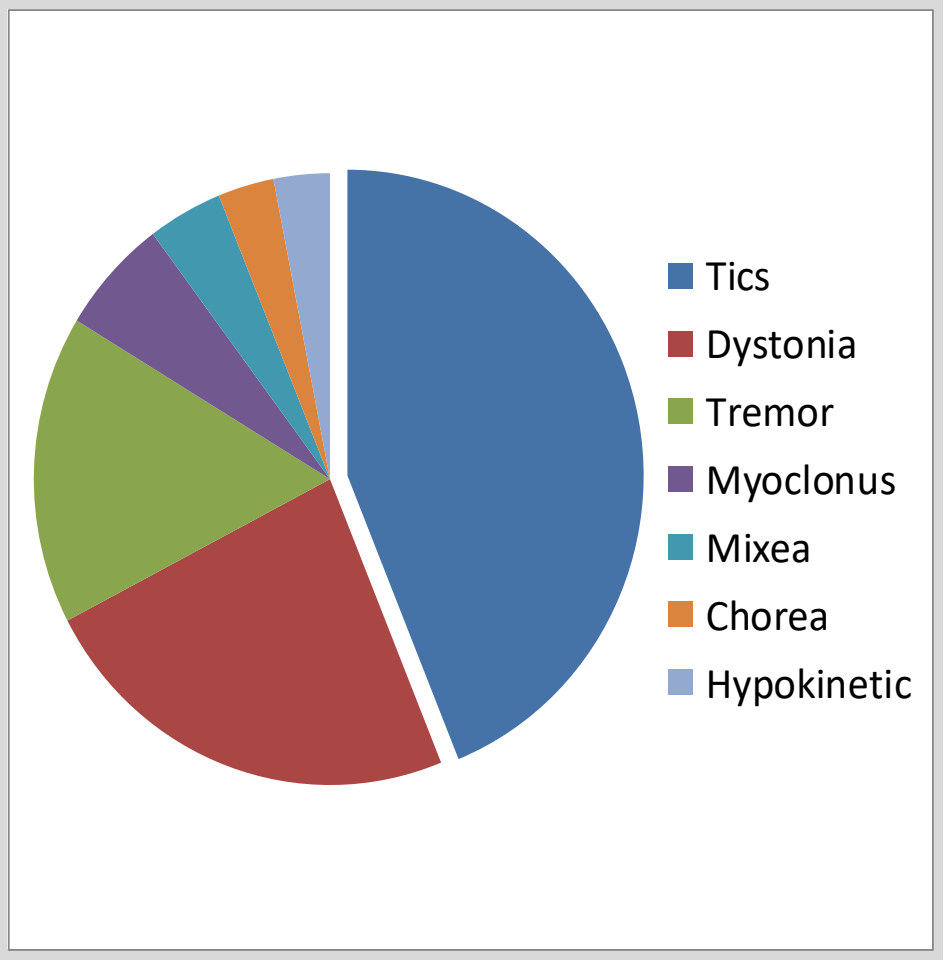
- Premonitory urge (a rising tension or somatic sensation that is relieved when the tic occurs).
- Tics are preceded by rising discomfort or urge (*sensory tic*) that is relieved by the movement.

(***itch and scratch***)

Fernandez alvarez, 2005

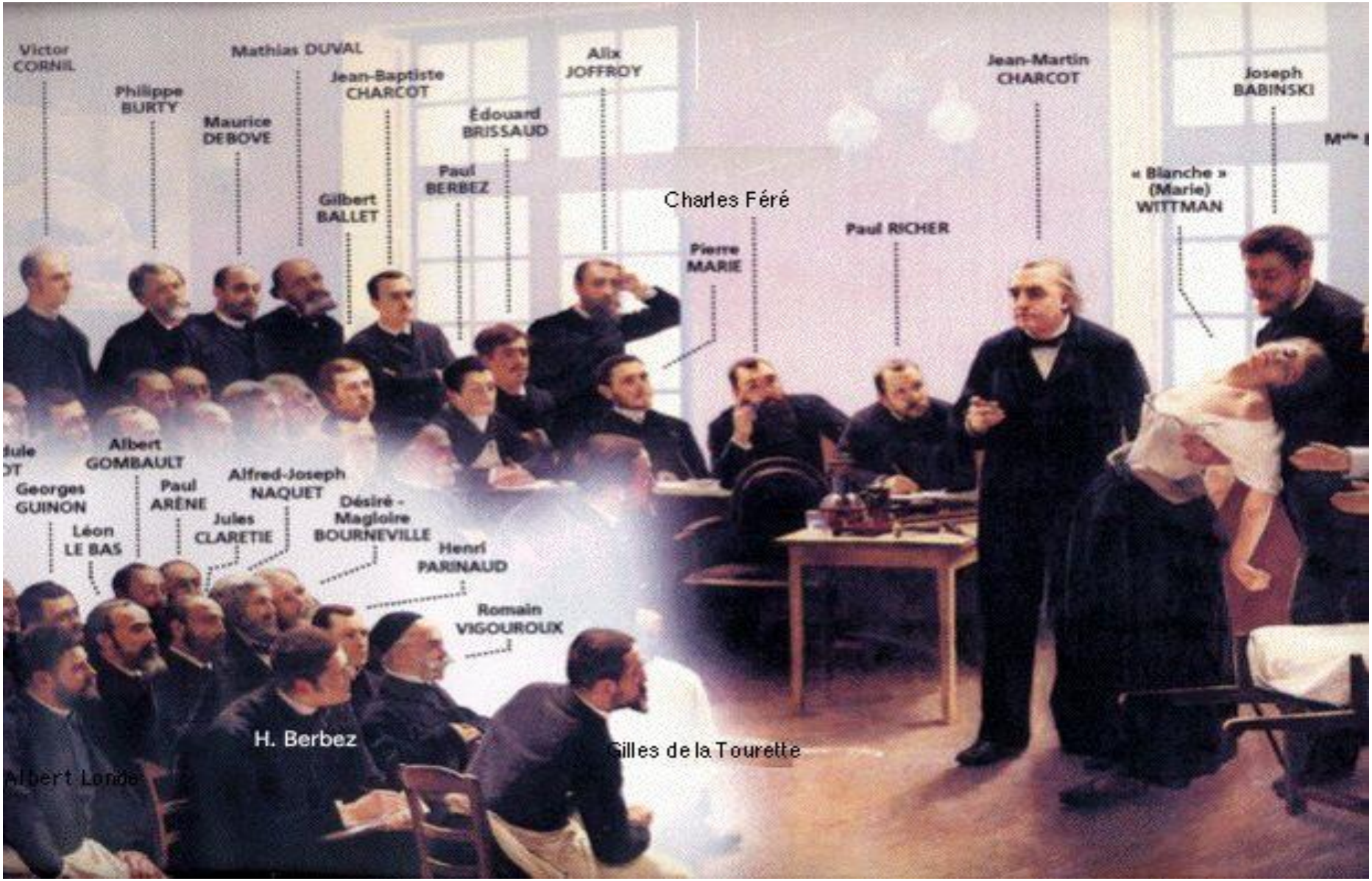
684 patient< 18 years

- Tics - 43%
- Dystonia- 23%
- Tremor- 16%
- Myoclonus 6%
- Mixea- 4%
- Chorea- 3%
- Hypokinetic 3%



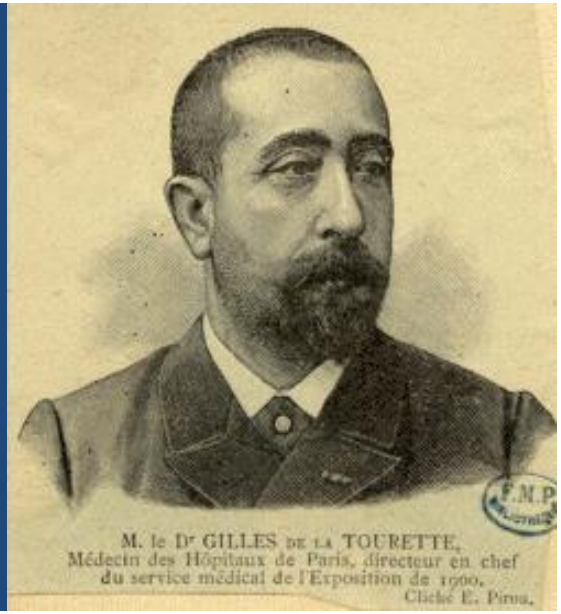
Tourette Syndrome: History

Tourette Syndrome: History



Tourette Syndrome: History

- Georges Gilles de la Tourette
- French neurologist, student of Charcot
- Interest in hysteria, hypnotism
- In 1885, published paper describing *malidie des tics*



- Study of 9 patients
- Patients characterized by convulsive tics, obscene utterances, repetition of others' words
- Charcot renamed it "Gilles de la Tourette Syndrome"

Prevalence and Incidence

- Originally thought to be rare, but now recognized to be more prevalent
- 20% of children experience tics, mostly transient
- Prevalence estimates vary greatly
  - .05% to 3% of all children
  - Majority suggest 1% of general population
- ~750,000* children in US, although many undiagnosed
- Occurs in all races and ethnicities
- Males 3-4x > females

*Tourette Syndrome Association, www.tsa-usa.org

0.1%

Worldwide incidence

400, 000 people in US have MS

MULTIPLE SCLEROSIS AFFECT:

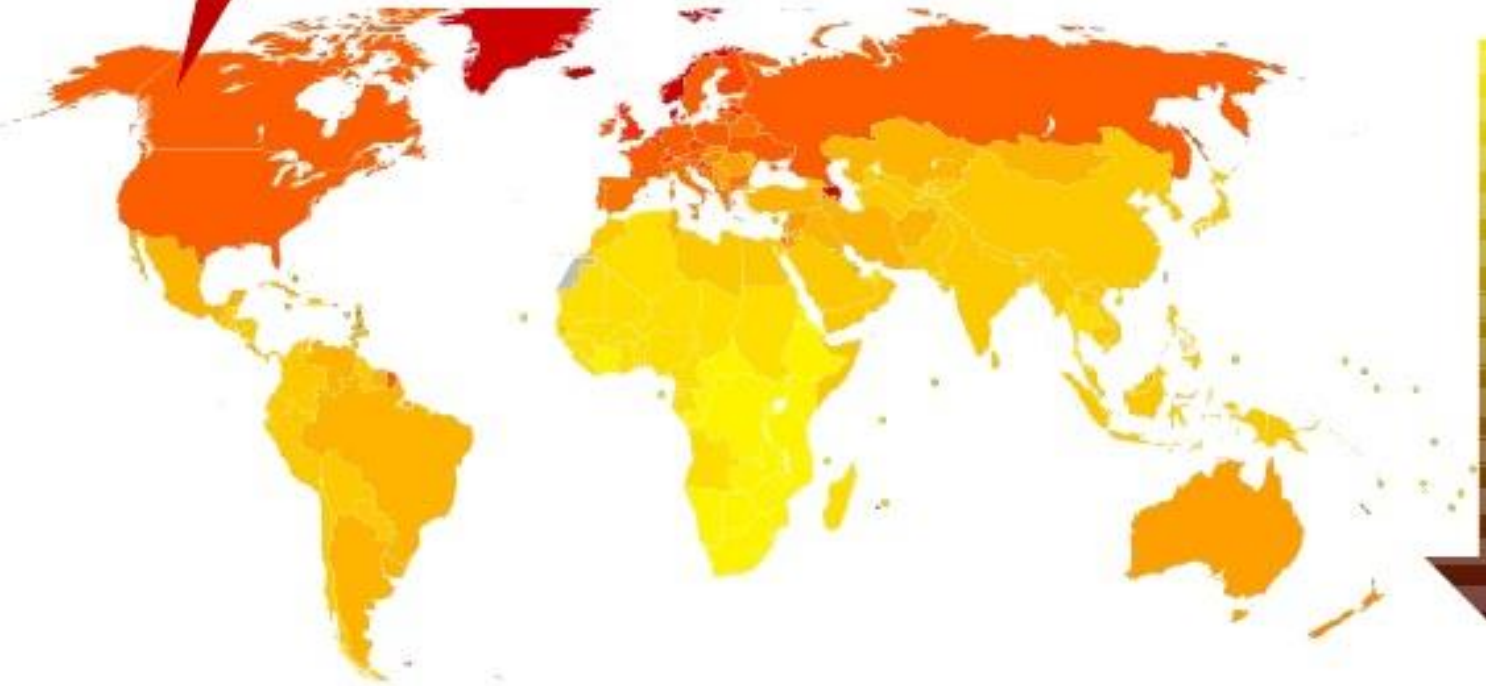
The ratio is increasing now

worse prognosis

Predominant age: **20-40**

1–3% risk of MS among 1st-degree relatives

Highly variable and unpredictable



Higher incidence in **Northern European** descent and in temperate climate, but the latitude gradient is **decreasing**

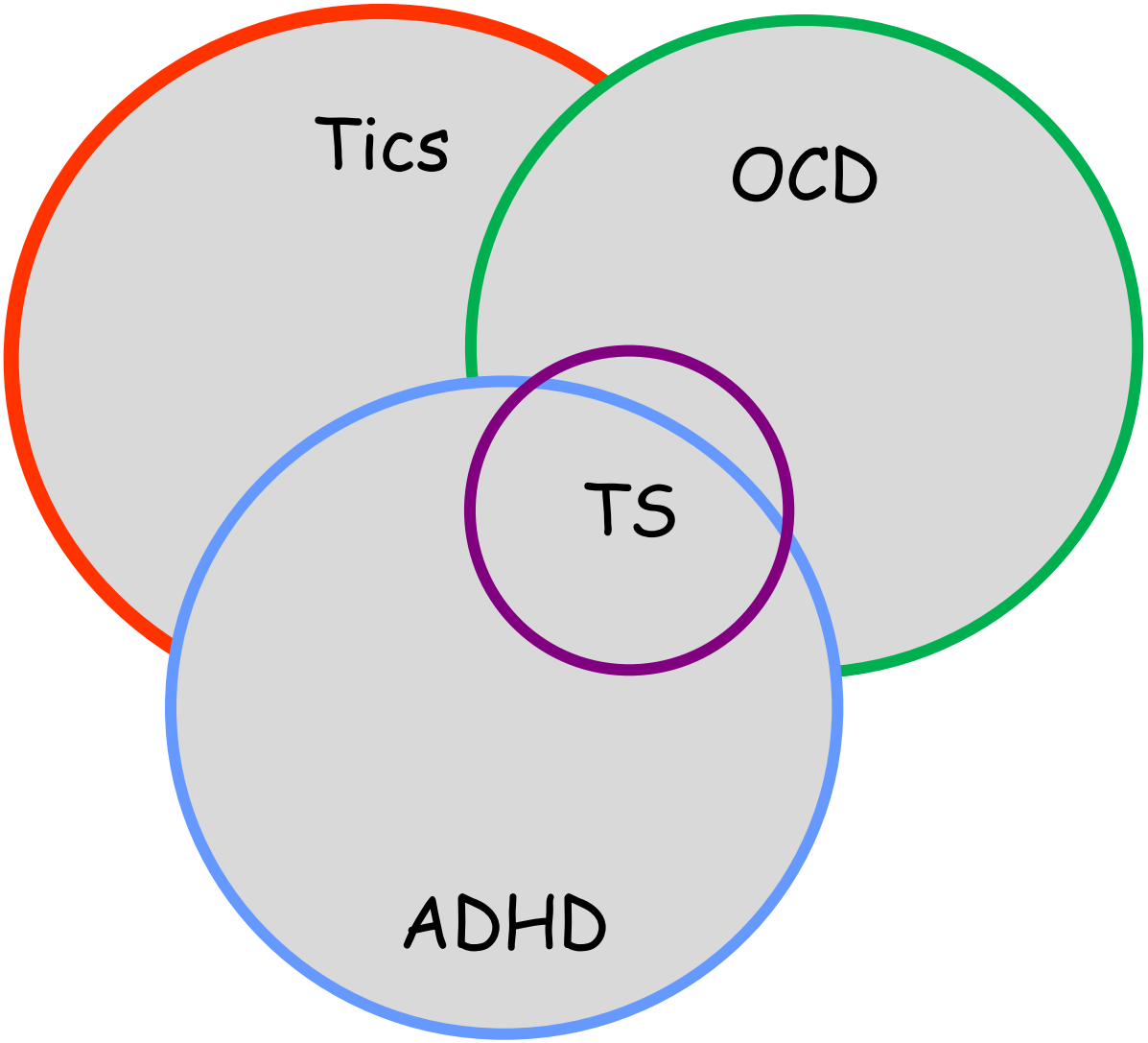
Tourette Syndrome: OCD

- Tics typically appear in early childhood (most often by age 6 or 7)
- In 96% of patients, disorder manifested by age 11
- Simple motor tics often initial symptom
  - eye blinking and neck movements common
- Phonic tics and more complex motor tics follow in next two years, but may appear later in adolescence
  - Motor tics tend to progress top-to-bottom and central-to-peripheral
  - Phonic tics also progress in complexity

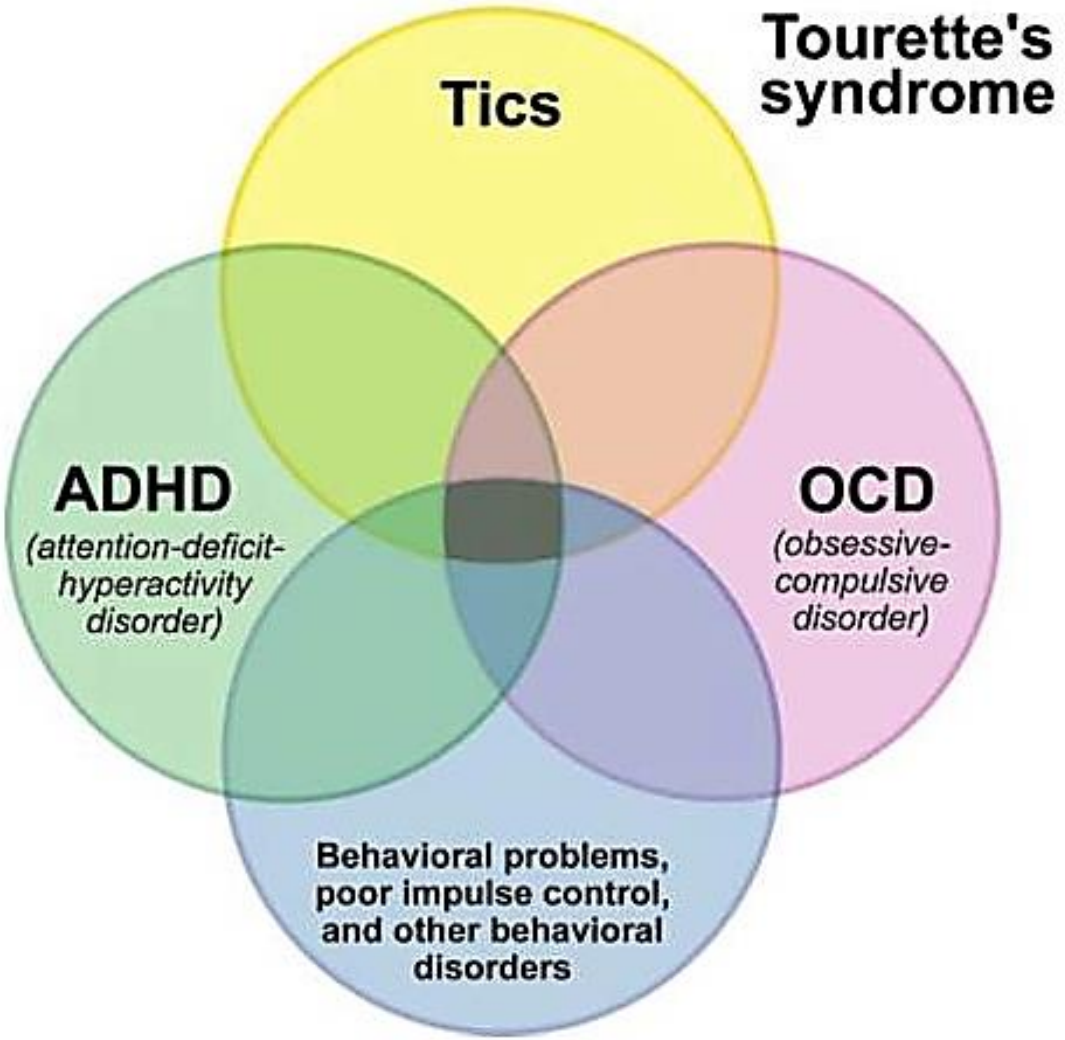
Tourette Syndrome: OCD

- Tics generally occur daily, but tend to wax and wane in frequency and intensity
- Type, location, and severity may change over time
  - Tics usually most severe at ~10 years of age
- By age 18 years, half of patients are free of tics
- For those whose tics persist, severity typically diminishes in adulthood

Tourette Syndrome: OCD



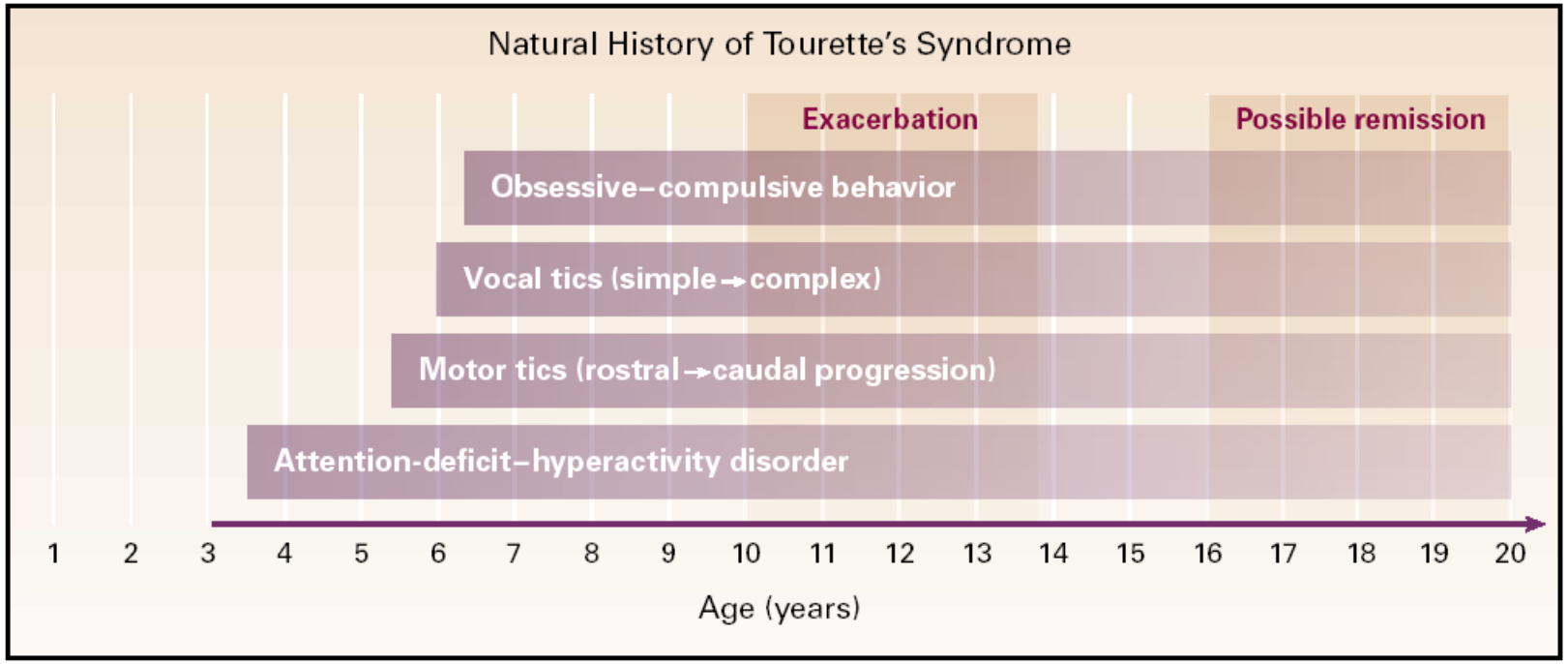
Tourette Syndrome: Associations



Tourette Syndrome: Associations

- Other abnormal movements and behavior patterns can also develop:
  - Stuttering
  - Sticking out the tongue
  - Smelling objects
  - Pounding the chest or body
  - Grabbing at one's genitals
  - Compulsive touching
  - Bruxism
  - Echopraxia

Course with Comorbidities



Jancovic, 2001

DSM-IV Diagnostic Criteria

- Both multiple motor and one or more vocal tics have been present at some time during the illness, although not necessarily concurrently. (A tic is a sudden, rapid, recurrent, nonrhythmic, stereotyped motor movement or vocalization.)
- The tics occur many times a day (usually in bouts) nearly every day or intermittently throughout a period of more than 1 year, and during this period there was never a tic-free period of more than 3 consecutive months.
- The onset is before age 18 years.
- The disturbance is not due to the direct physiological effects of a substance (e.g., stimulants) or a general medical condition (e.g., Huntington's disease or postviral encephalitis).

Differential Diagnosis of Tourettes

- Transient tics of childhood
- Prenatal/perinatal insults
  - Congenital CNS defects
  - Birth defects
- Infections/post-infectious
  - Post-viral encephalitis
  - HIV infections of CNS
  - Lyme disease
  - PANDAS

Differential Diagnosis of Tourettes

- Head trauma
- Toxin exposure
- Drugs
  - Neuroleptics, levodopa, opiate withdrawal, amphetamines, lamotrigine
- Chromosomal abnormalities
  - XYY
  - XXY
- Fragile X syndrome

Transient	Chronic	Tourette's
Single/multiple motor AND/OR vocal	Single/multiple motor OR vocal	Multiple motor AND single/multiple vocal
< 1 year =/> 4 weeks	> 1 year Not tic free > 3 months	> 1 year Not tic free > 3 months

PANDAS

- Pediatric Autoimmune Neuropsychiatric Disorders Associated with Strep
- A possible cause of OCD and Tourettes
  - Group A β-hemolytic streptococcal infection in select individuals may induce neuronal damage

Structural Neuroimaging Findings

- General neuroimaging and neuropathological examination of TS brains is normal
- However, morphological abnormalities have been reported in volumetric MRI studies:
  - A loss or reversal of normal asymmetries of the putamen and lenticular nucleus has been noted
  - Corpus callosum morphology (and therefore interhemispheric connectivity) appears to be altered in (at least) males with TS

Functional Neuroimaging Findings

- SPET studies have detected hypoperfusion in various brain structures bilaterally (including the BG, orbitofrontal cortex, and temporal lobes)
- PET scans have shown decreased activity in prefrontal cortices and striatum

Etiology of TS

Precise etiology unknown

May be inherited in ~80% of cases

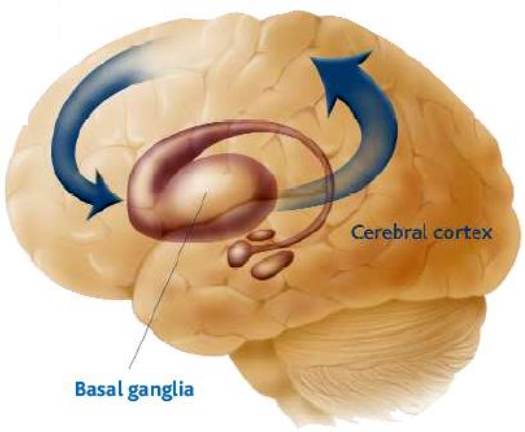
Support for developmental disorder of synaptic neurotransmission involving cortical-subcortical circuitry

Pathogenesis of TS

Support for TS as a developmental disorder of synaptic neurotransmission

Involves basal ganglia and related neural pathways

Failure in filtering (disinhibition) along striatal-thalamic-cortical circuit, resulting in ineffective removal of unwanted, interfering information



Same circuits and structures involved in OCD, ADHD

Yale Global Tic Severity Scale

A simple tracking device for assessing the nature and severity of tics

Addresses the following categories:

- Simple motor
- Complex motor
- Simple phonic
- Complex phonic
- Behavior

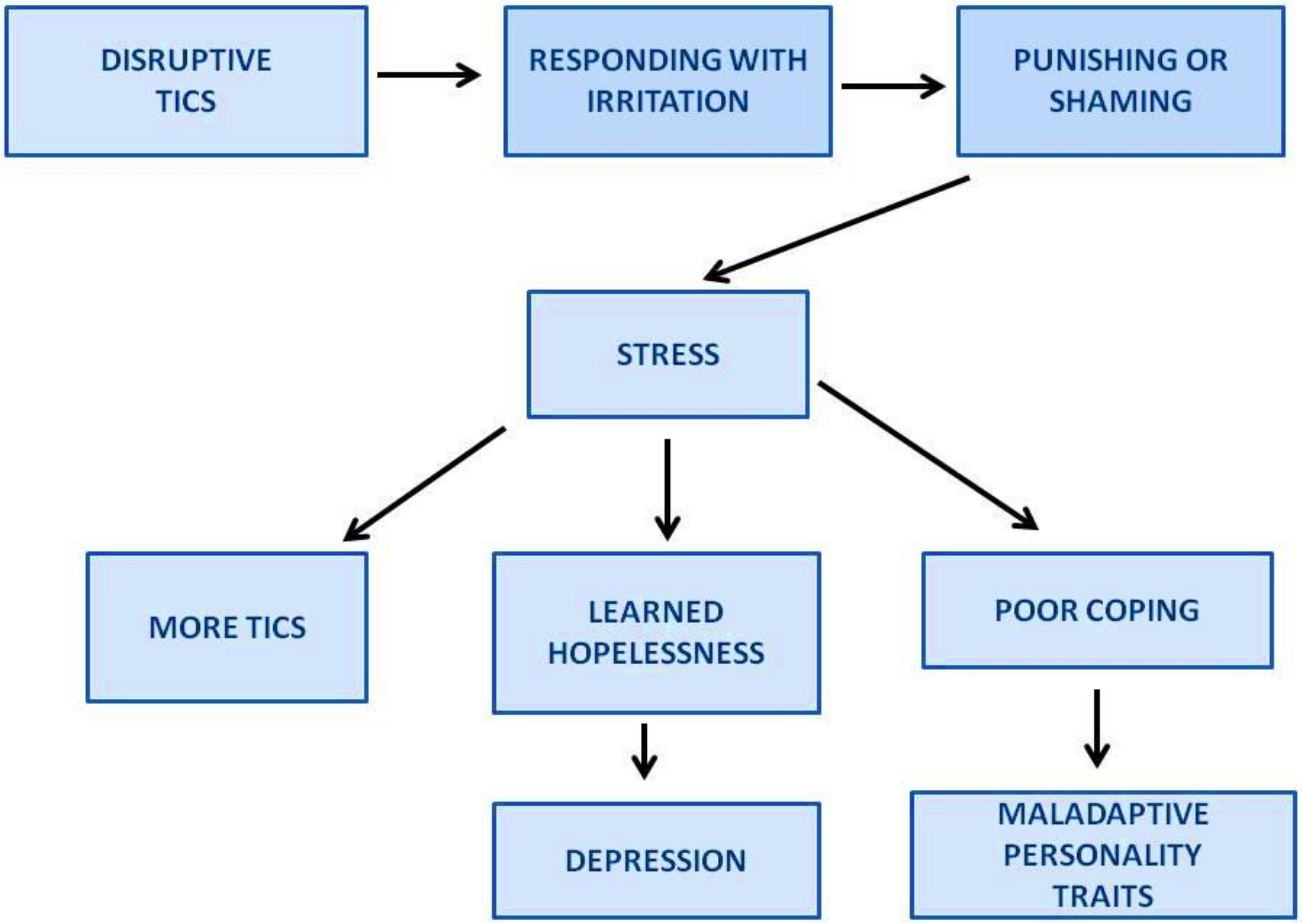
Uses a Likert Scale

Most tic exacerbations will be identified with the change on the YGTSS is greater than “9” and the total current YGTSS score exceeds “19”

Social Impact of TS

- Increased self-consciousness and poor self-esteem
- Often targets for mocking and bullying
- Withdrawal from social situations
- Difficulties in school or workplace
- Comorbid ADHD or other disorders increases likelihood of social problems.

Social Impact of TS



Management considerations

- No standard practice guidelines for physicians.
- Highly individualized to patient.
- Tic control not sole focus of treatment.
- Determine areas of functional and psychosocial impairment imposed by tics and comorbid conditions.

Management considerations

- Medication may be prescribed for tics, comorbid disorders or both.
- Monotherapy ideal, but polypharmacy common.
- Most med use is off-label or not specifically approved for children.
- Several medication options have been used, representing variety of pharmacological classes.

Management considerations

- Simply having tics not indicator for medication.
- Medication usually considered when symptoms interfere with peer relationships, social interactions, academic or job performance, or ADLs.

Management considerations

No drug will entirely eliminate tics.

Goals: relieve tic-related discomfort or embarrassment and to achieve a degree of control of tics that allows the patient to function as normally as possible.

Management considerations

Multi-component management approach recommended

- Education for patient and others
- Behavioral approaches
- Medication
- Academic accommodations
- Psychosocial and psychological supports

Treatments: Psychosocial

- Counseling for family and patient
- Relaxation therapy
- Supportive therapy
- Habit Reversal Therapy

Management: Behavioral Approaches

- Only “habit reversal” has been shown effective in adults (limited data for children)
- Increase awareness of tics and premonitory urges and then performing competing responses
- Results in less noticeable tics and may decrease degree of urge

HRT

- 4 Components:
  1. Awareness training

     --learn to recognize when they're ticking
  2. Development of a competing response

     --less noticeable, can be carried out for more than a few minutes
  3. Building motivation

     --make a list of the problems caused by tics, all the bad things it brings
  4. Generalization of new skills

     --practice the skills in new contexts and locations

Management: Behavioral Approaches

- Other behavior-based strategies for tic control not well documented
- Anxiety reducing techniques (e.g., PMR), awareness increasing techniques (e.g., videotaping) may help reduce tics

Treatments: Medication

- Historically high potency first generation antipsychotics
  - Pimozide (Orap) best studied
  - Haloperidol
- Severe side effects has led to search for alternative 2nd generation antipsychotics

Treatments: Medication

For reducing tics:

- Clonidine, Guanfacine: may treat comorbid anxiety, ADHD, insomnia
- Atypcial neuroleptics (e.g., Risperdal)
- Conventional neuroleptics (e.g., Haldol,Pemozide)
- Botunlinum toxin A (Botox): for severe focal tics
- Benzodiazepine (e.g., Klonopin)
- Less common, but promising:
  - GABA agonist/muscle relaxant (Baclofen)
  - Dopamine agonist (Pergolide): may also help ADHD

Treatments: Medication

Comorbid disorders:

- Follow guidelines for individual disorders (e.g., ADHD, OCD, depression)
- Controversy regarding whether ADHD treatment with psychostimulants exacerbates tics
- SSRIs: Effective for comorbid obsessions and compulsions, anxiety, and, possibly, depression; mixed results about tics.

Treatments: Comorbid Conditions

- OCD
  - SSRIs +/- antipsychotics
- ADHD
  - History of concern that stimulants would "unmask" tics
  - Multicenter, RDBPC study of MTP and clonidine (alone and in combination) in 136 children with Tourettes demonstrated:
    - Significant improvement in ADHD with both treatments
    - Greatest benefit resulting from a combination of both
    - The proportion of subjects reporting a worsening of tics was no higher amongst those treated with MTP alone (20%) vs. clonidine (26%) vs. placebo (22%)

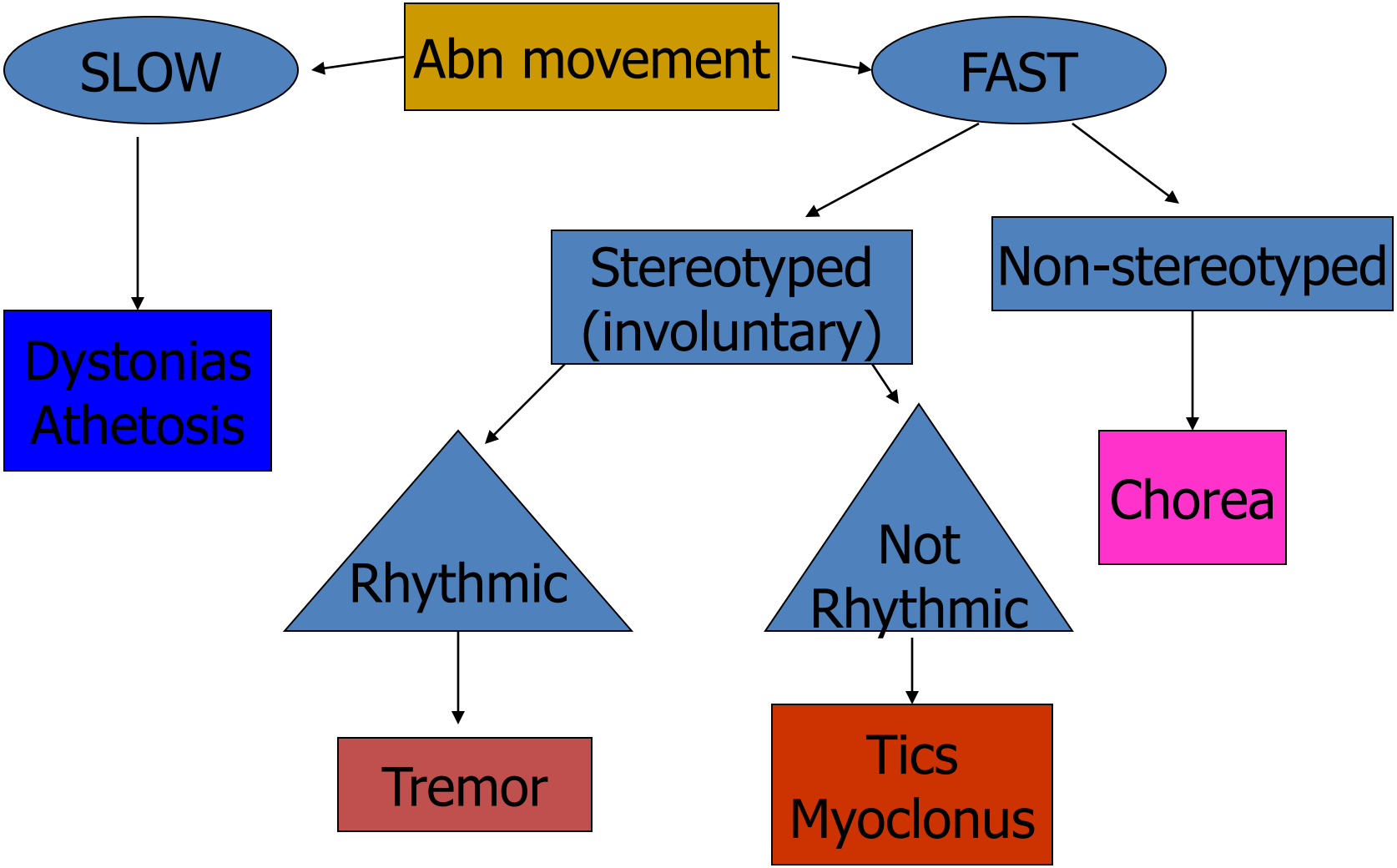
Treatments: Other Approaches

- Alternative approaches such as fish oil supplements are being investigated
- Dietary modification and allergy testing have been explored for tic management but not supported
- High frequency Deep Brain Stimulation (DBS) shown to be effective in small number of cases (no children)

Prognosis

50%	• Tics resolve (go away)
30-50%	• Tics get much better but are still displayed
5-10%	• Tics continue into adulthood

Differentiating between types



Paroxysmal Dyskinesias (PDys)

- A heterogeneous group of disorders characterized by the abrupt onset of abnormal involuntary movements.
- These usually arise out of a background of normal motor behavior.
- The attacks are often not witnessed by the physician and the movements are varied with a combination of chorea, ballism and dystonia.

Paroxysmal Dyskinesias (PDys)

What is not considered to be a Paroxysmal Dyskinesia?

1. Action/Task Specific Dystonia
2. Tics- can occur in bursts
3. Paroxysmal Exaggeration of Tremor
4. Action Myoclonus

Paroxysmal Kinesigenic Dyskinesia

- Attacks precipitated by ***s***udden movement or ***s***tartle and sometimes by ***s***tress
- Frequency up to 100 per day of ***s***hort lasting (Patients may have a sensory aura before the attack and there is often a refractory period
- Most have asymmetric dystonia while others may have chorea

PKD-Proposed Criteria

- Identified kinesigenic trigger for the attacks
- Short duration of attacks (<1 minute)
- No loss of consciousness or pain during attacks
- Exclusion of other organic diseases and normal neurologic examination
- Control of attacks with phenytoin or carbamazepine, if tried
- Age at onset between 1 and 20 years, if no family history of PKD

Bruno et al 2004

Paroxysmal NONKinesigenic Dyskinesia

- Often inherited as an autosomal dominant trait.
- More often in males (2:1).
- As in PKD attacks start in childhood and decrease in adulthood.

Paroxysmal NONKinesigenic Dyskinesia

- Attacks precipitated by alcohol,fatigue,and caffeine. In one series 98% of cases with MFR-1 mutation had this clinical correlate.(Bruno,2007)
- Some families have predominant dystonia while others have chorea.
- Frequency 3/day to 2/year.
- Duration minutes to hours rarely under 5 minutes.

Paroxysmal Exercise-Induced Dyskinesia

- Frequency 1 per day to two/month.
- The duration is intermediate between PKD and PNKD (5-30 minutes).
- Prolonged exercise precipitates attack.
- The legs are more affected but the exercise limited to upper extremity may involve upper limbs alone.

Paroxysmal Exercise-Induced Dyskinesia

- Usually,inherited in a dominant mode but sporadic attacks described as well
- In some families there is an overlap between PNKD and PED.
- PED may precede parkinsonism in familial PD (Bruno MK, 2004).

Paroxysmal Dyskinesias (PDys)

Disorder	Age of Onset	Reported Triggers	Duration of Episode	Treatment	Causative Gene	Allelic Disorders
Paroxysmal kinesogenic dyskinesia	Infancy to fourth decade	Sudden movement	Short: seconds to minutes	Carbamazepine, phenytoin	*PRRT2*	Infantile convulsions choreoathetosis, familial Benign paroxysmal torticollis of infancy Familial migraine
Paroxysmal nonkinesogenic dyskinesia	Infancy to fourth decade	Caffeine, alcohol, stress/anxiety, sleep deprivation	Longer: minutes to hours	Benzodiazepines	*MR1*	None known
Paroxysmal exercise-induced dyskinesia	Infancy to adulthood	Exercise, stress, fasting	Longer: minutes to hours	Ketogenic diet	*SLC2A1*	Glucose transporter deficiency phenotypes like absence epilepsy, myoclonic-atonic epilepsy, generalized epilepsy, early infantile epileptic encephalopathy

?Paroxysmal Hypnogenic Dyskinesia

First description by Joynt and Green in a patient with multiple sclerosis.

Attacks occur during Non-REM sleep.

Attacks represent medial frontal lobe seizures in most cases.

In rare cases the long lasting attacks may be basal ganglia origin.

Secondary Paroxysmal Dyskinesias

- Multiple sclerosis
- Cerebral Palsy
- Hypoparathyroidism and pseudohypoparathyroidism
- Hypoglycemia
- Head trauma
- Cerebrovascular disease
- Neuroacanthocytosis
- Psychogenic

Miscellaneous Causes of Sec PDx

- Cytomegalovirus Encephalitis
- Neurosarcoidosis
- Migraine
- Cervical Cord lesions
- Primary CNS Lymphoma
- Kernicterus
- Hypoglycemia

Miscellaneous Causes of Sec PDx

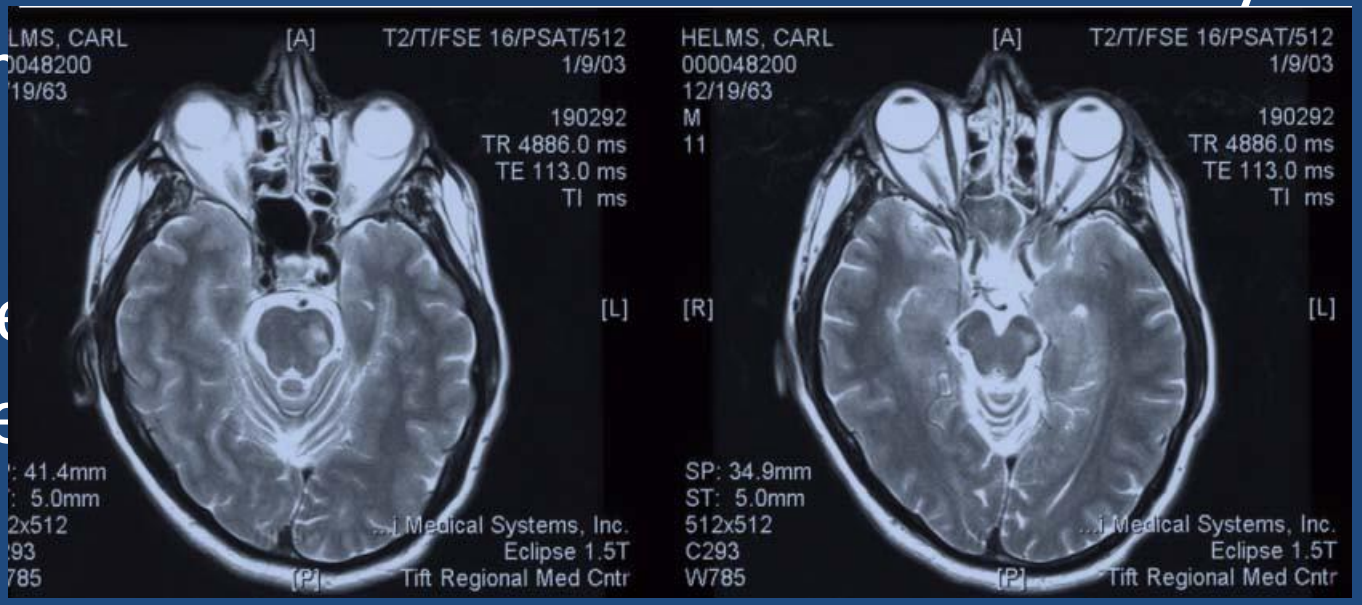
Also known as tonic seizures and may be the presentation of the disease.

Unilateral , bilateral attacks described more in the Japanese.

Hyperventilation precipitates the attack. Painful

Secondary Paroxysmal Dyskinesia- Multiple Sclerosis

Also known as tonic seizures and may be the preser



Unilate in the Japane

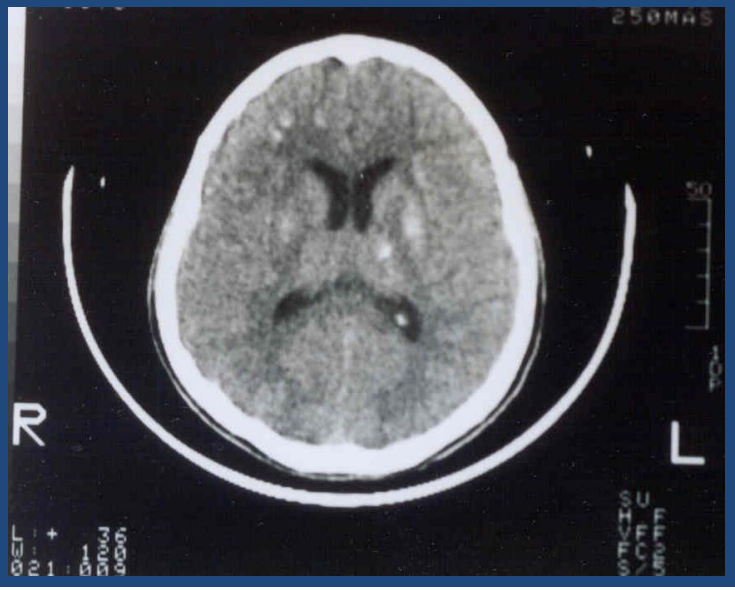
Hyperventilation precipitates the attack. Painful

Metabolic Disorders

PNKD may occur in Idiopathic hypoparathyroidism

PNKD and PKD reported in pseudohypoparathyroidism (Dure,1998)

The dyskinesia may respond to Vitamin D and Calcium



Overlaps

Episodic Ataxia 1

- Early childhood
- Provoked by startle
- Duration minutes
- Interictal myokymia
- Autosomal dominant
- Potassium channel gene mutation KCNA-1 –12P

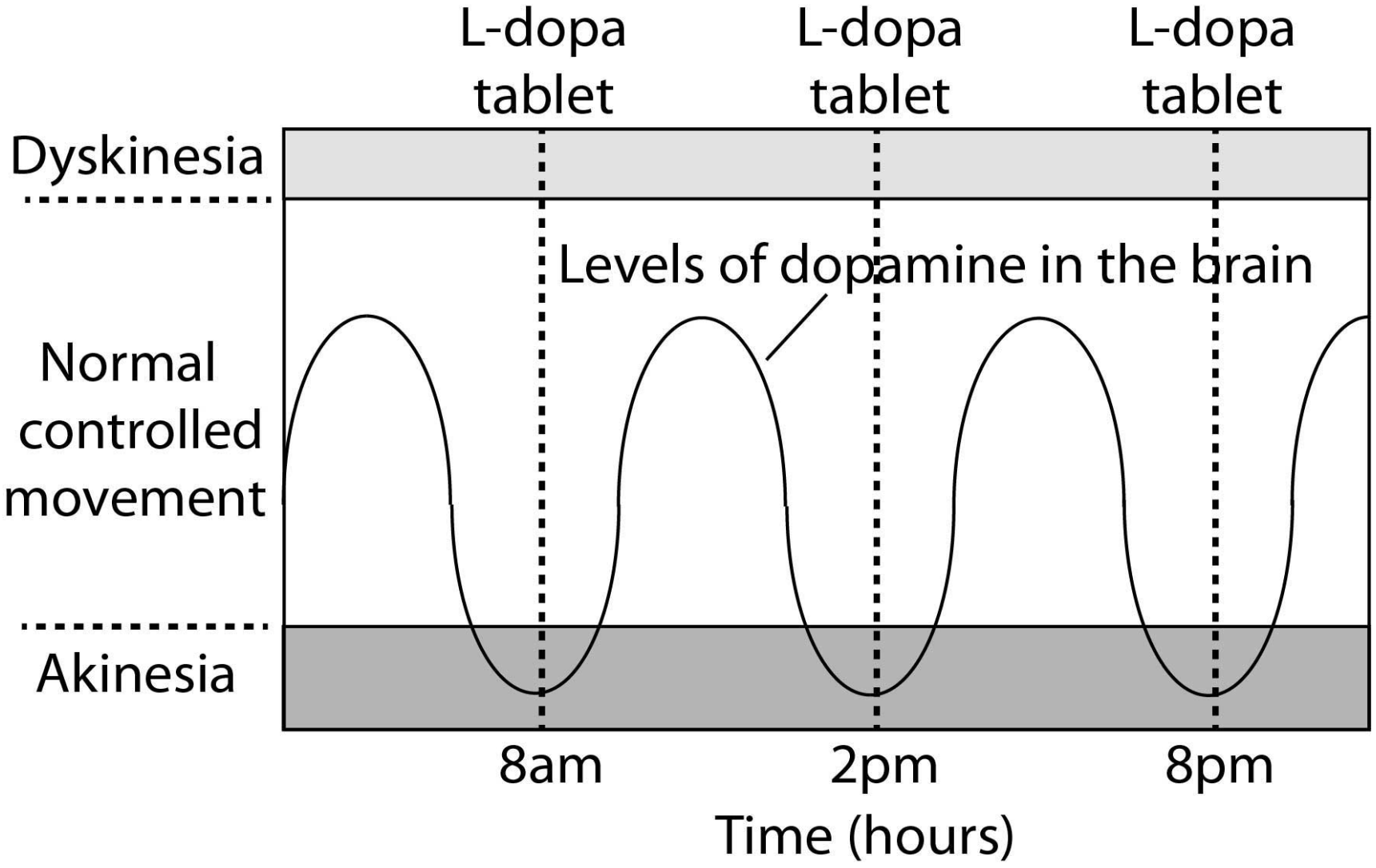
Episodic Ataxia 2

- Late childhood
- Stress,alcohol
- Minutes to hours
- Interictal nystagmus
- Autosomal dominant
- Calcium channel gene mutation CACNLIA—19P
- Rarely myasthenic syndrome (Jen et al ,02)

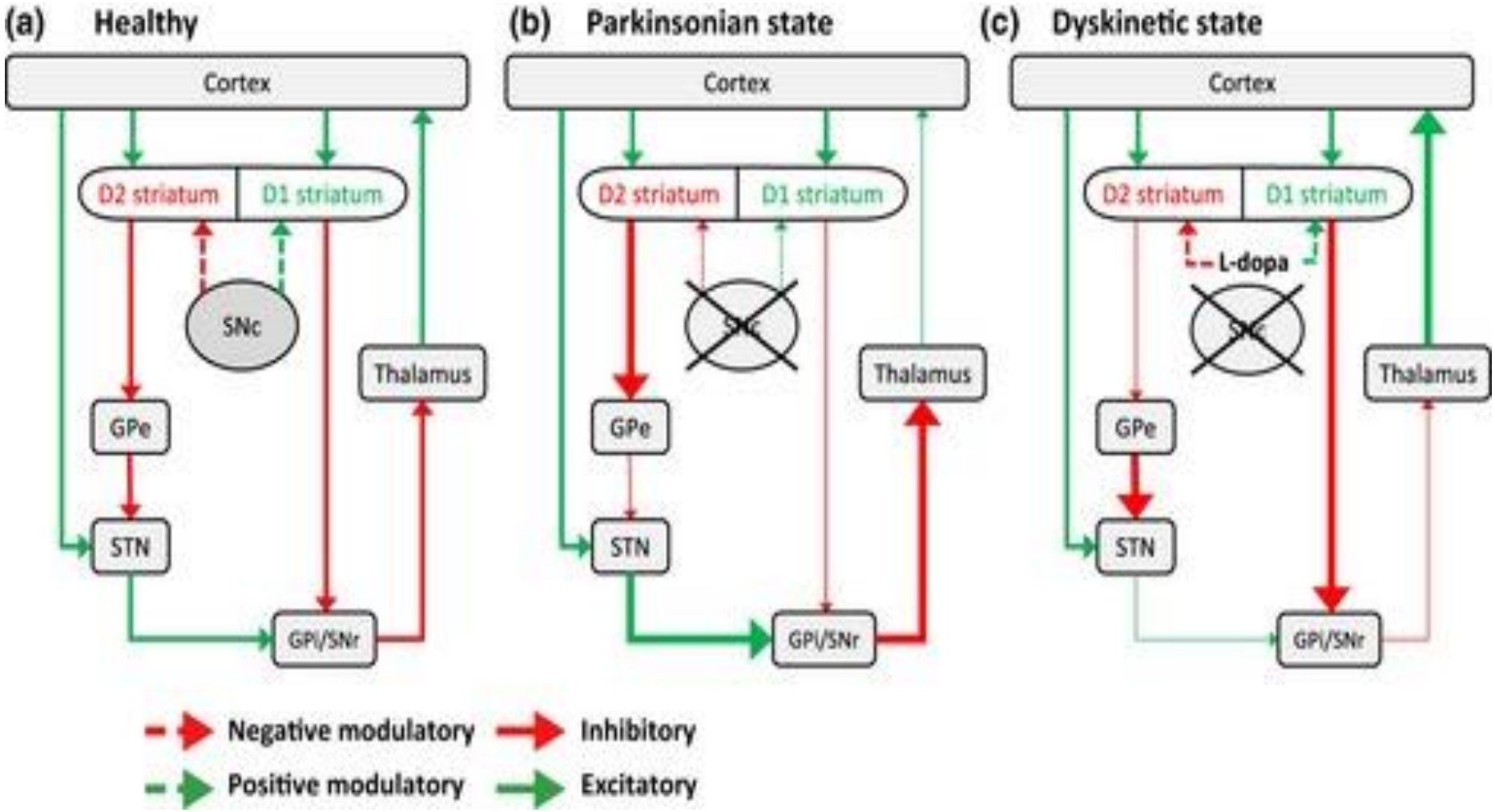
Overlaps

- Facial myokymia and dystonic/choreic movements (FDFM) is a dominant disorder with dyskinesia that is episodic but may become constant with increasing age.
- Localized to chromosome 3.(Raskind WH,2009).
- Ion channel dysfunction is a well known mechanism for myokymia.

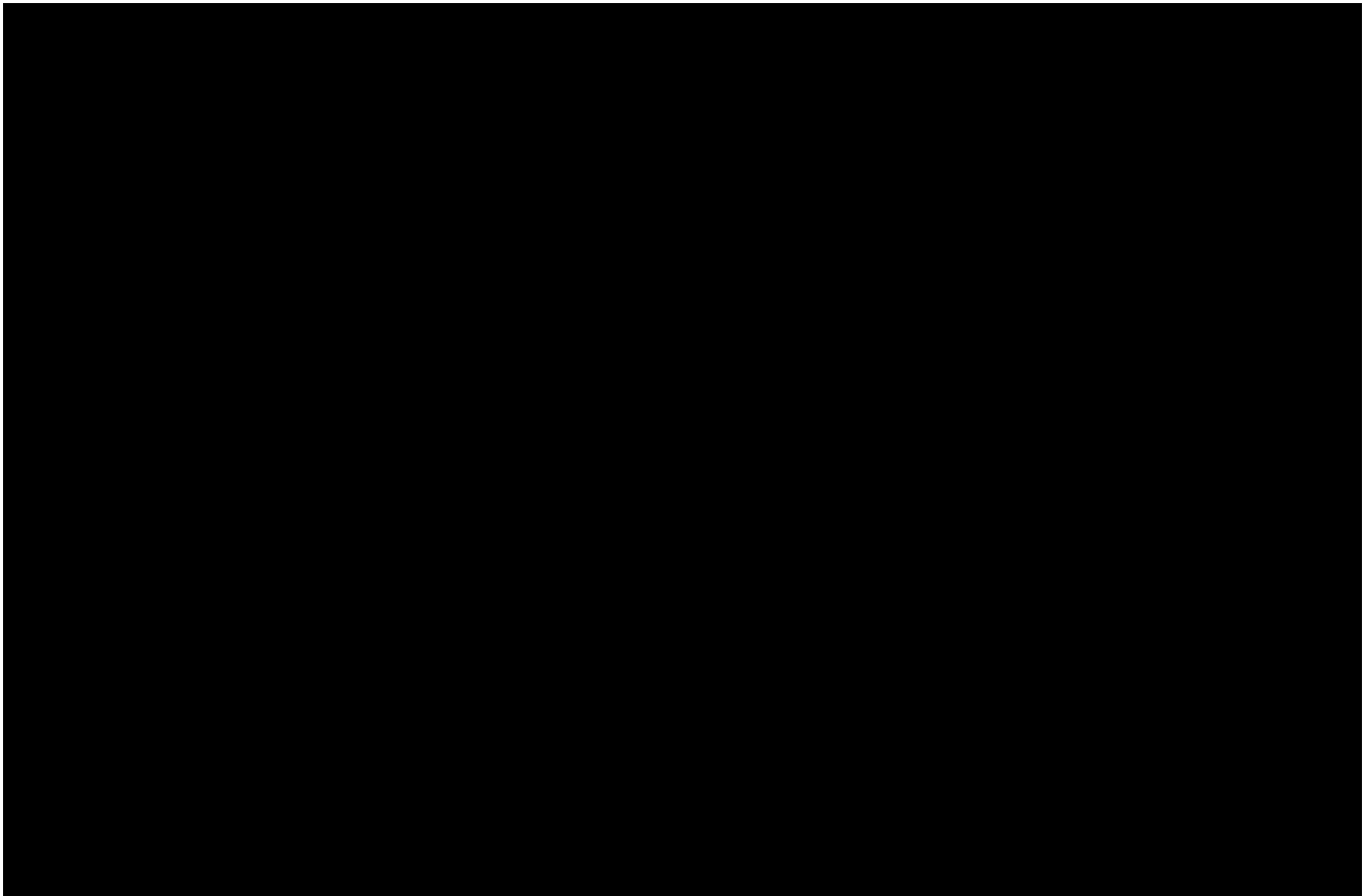
L- dopa dyskinesia



L- dopa dyskinesia



Adapted from Rodriguez et al., 2009



Tardive dyskinesia

TD is a syndrome of permanent, involuntary movements, is most commonly caused by the longterm use of typical antipsychotics.

Once it has developed, TD is irreversible.

Tardive dyskinesia

Symptoms of TD include:

– Involuntary movements of the tongue, facial and neck muscles, upper and lower extremities, and truncal musculature

– Tongue-thrusting and protrusion, lip-smacking, blinking, grimacing and other excessive, unnecessary facial movements

Tardive dyskinesia

Although TD is irreversible, its progression can be arrested by decreasing or discontinuing the antipsychotic medication.

Preventing the occurrence of TD is done by keeping maintenance dosages as low as possible, changing medications, and monitoring the client periodically for the initial signs of TD.

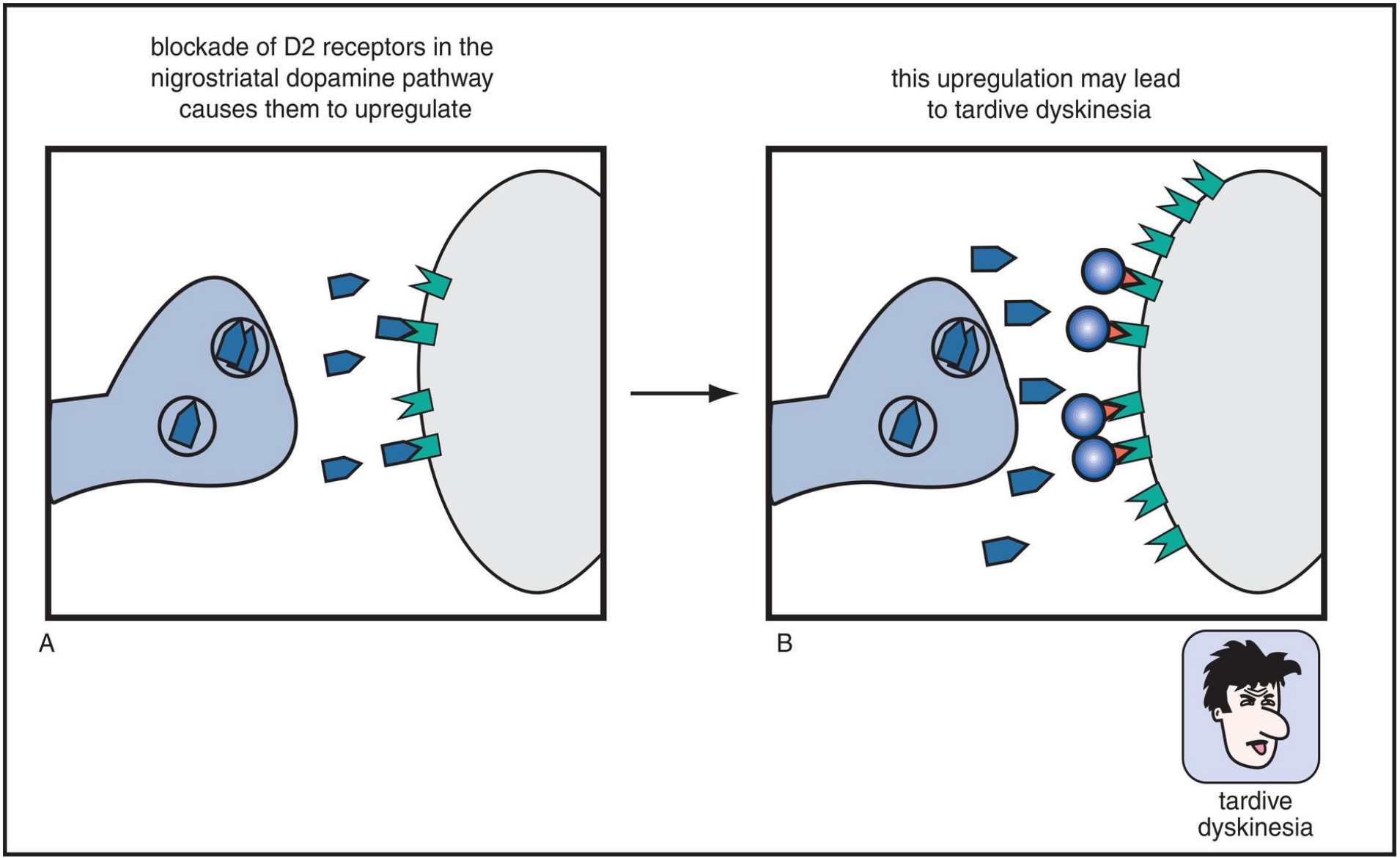
Tardive dyskinesia

Persons who have already developed signs of TD

Still need to take antipshychotic medication

Are often given clozapine.

Tardive dyskinesia

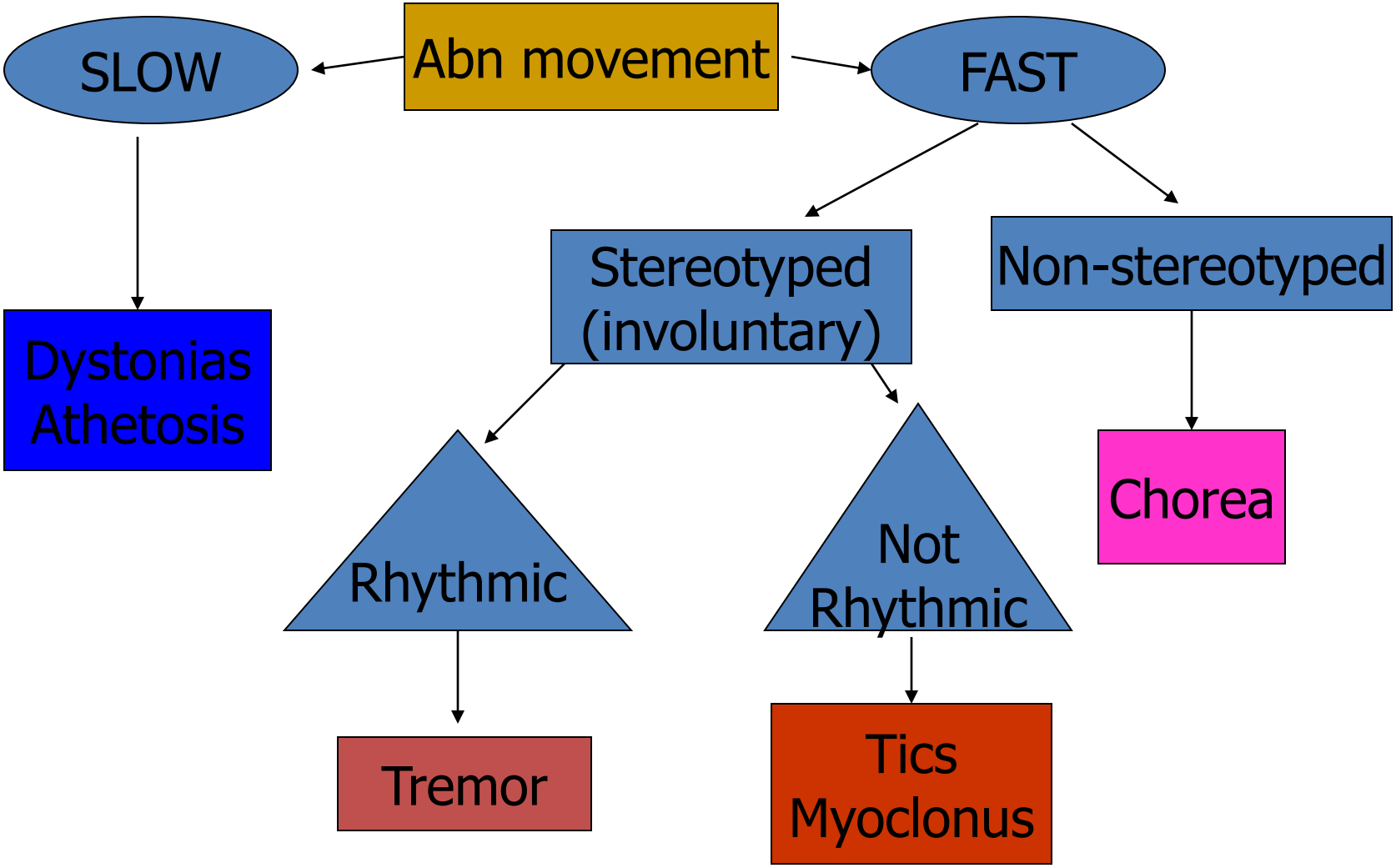


Clinical assessment of AIM

- **D**escribe the movement
- **D**ifferentiate from other MD
- **D**istribution
- **D**ecreased by , Increased by
- **D**iurnal variation
- **D**uration
- **D**istinguished phenomenon

Dr Amr Hassan 2017

Clinical assessment of AIM

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THANK YOU