PSYHIATRY

Schizophrenia Spectrum and Other Psychotic Disorders

Color Index:

- Important
- Extra Explanation

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Schizophrenia

It is not a single disease but a group of disorders with heterogeneous etiologies.
- Found in all societies and countries with equal prevalence & incidence worldwide.
- A life prevalence of 0.6 – 1.9 %
- Annual incidence of 0.5 – 5.0 per 10,000
- Peak age of onset are 10-25 years for ♂ & 25-35 years for ♀ (not common in elderly)

(risk of the disease increases with smoking, and weeds)

Crash course explaining schizophrenia

Schizophrenia Spectrum and Other Psychotic Disorders

- Schizophrenia
- Brief Psychotic Disorder
- Schizophreniform Disorder
- Delusional Disorder
- Schizoaffective Disorder
- Schizotypal (Personality) Disorder
- Substance/Medication -Induced Psychotic Disorder
- Psychotic Disorder Due to Another Medical Condition

Clinical Features

- No clinical sign or symptom is pathognomonic for schizophrenia, patient's history & mental status examination are essential for diagnosis.
- Picture of schizophrenia includes positive and negative symptoms:

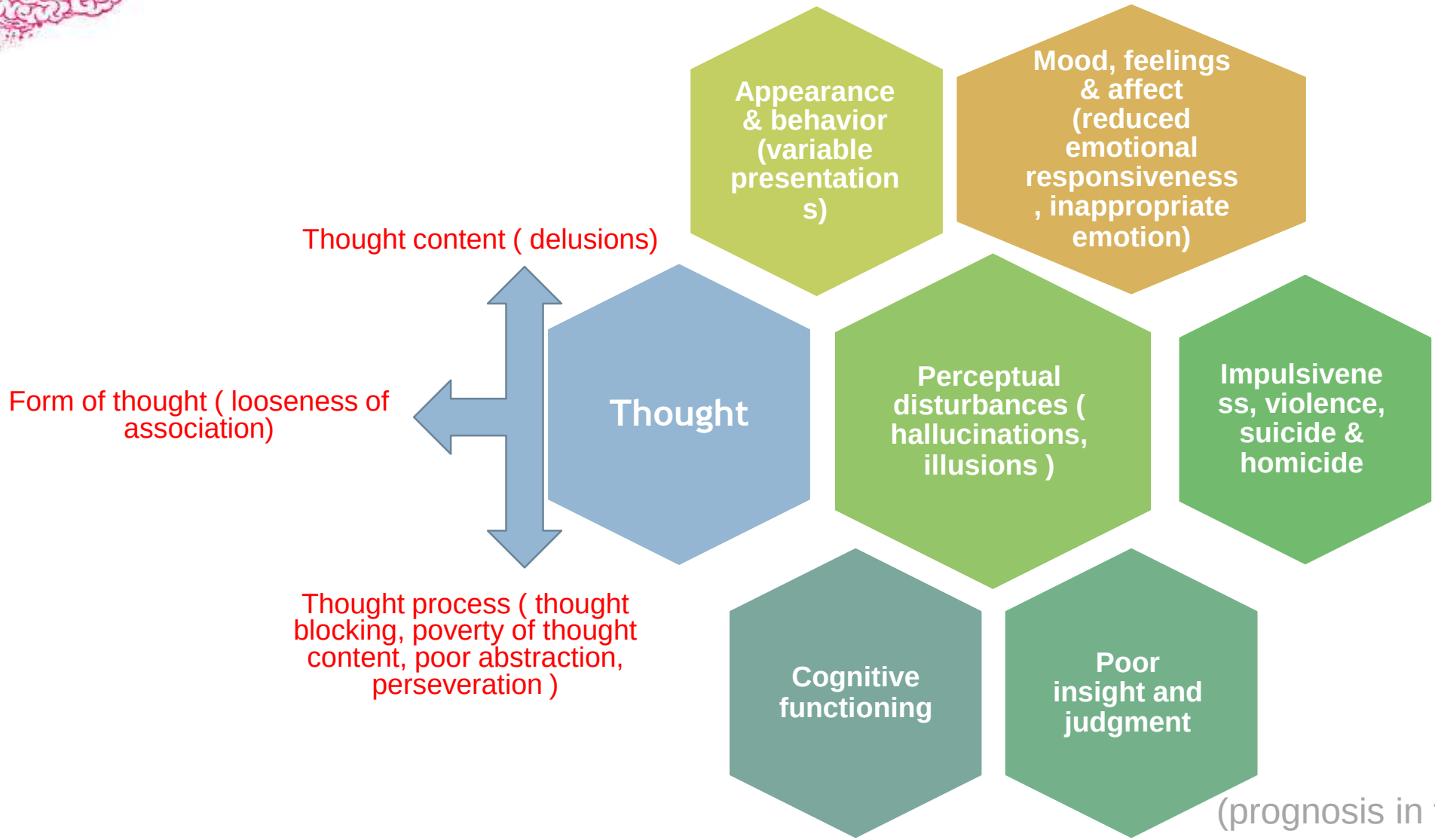
*Positive signs: are signs which add something to the experience of the patient
^crash course definition

Positive signs	Negative signs
☐ Delusions	☐ Affective flattening or blunting
☐ Hallucinations	☐ Poverty (lack) of speech
☐ Disorganized speech	☐ Poor grooming (cleaning up one's self)
☐ Disorganized behavior	☐ Lack of motivation
	☐ Social withdrawal

*Negative signs: are signs which subtract from normal behavior
^ crash course definition

- Duration of at least 6 months
- Premorbid history includes schizoid or schizotypal personalities (few friends & exclusion of social activities) .
- Prodromal (before the illness) features include obsessive compulsive behaviors

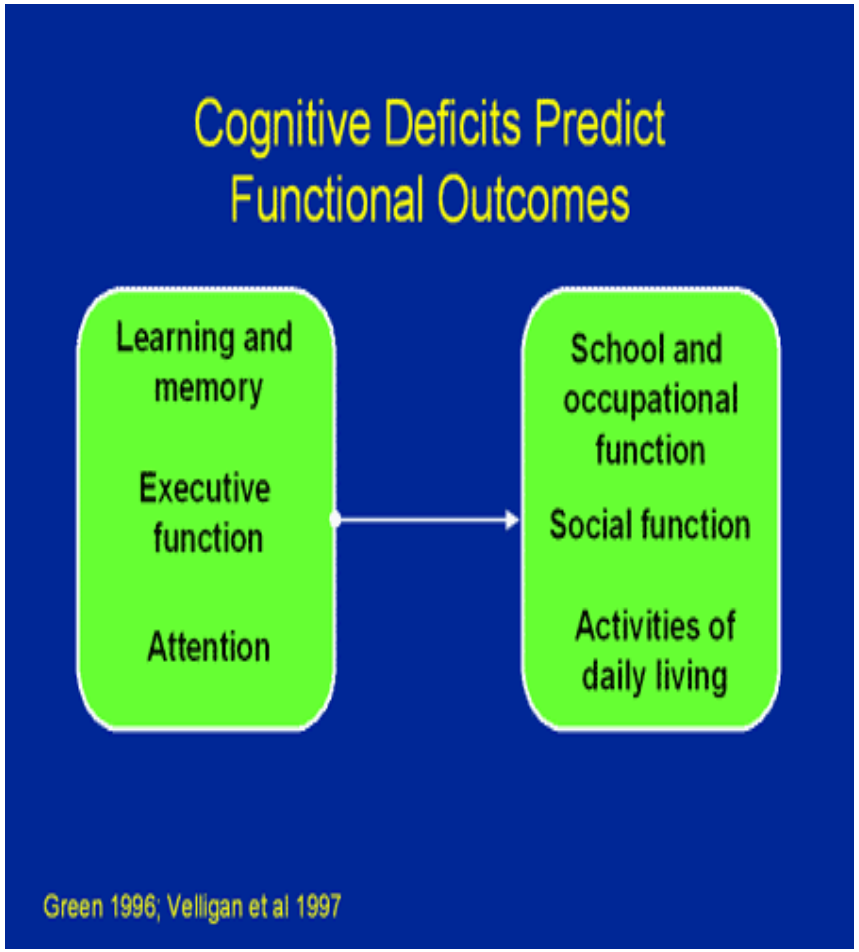
Mental status examination

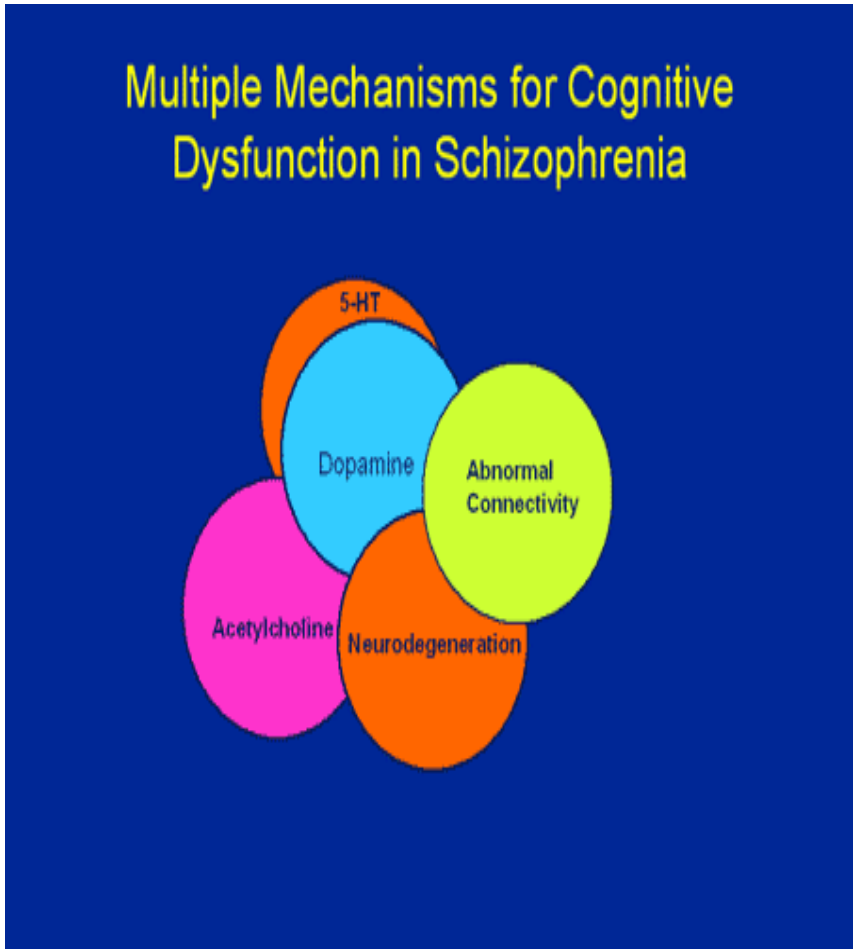


(prognosis in females is better)

*الجرائم العامه مالها دخل بالمرض تقريبا
لكن الجرائم مثلا بين الاب و ابنه قد تكون سببها المرض هذا

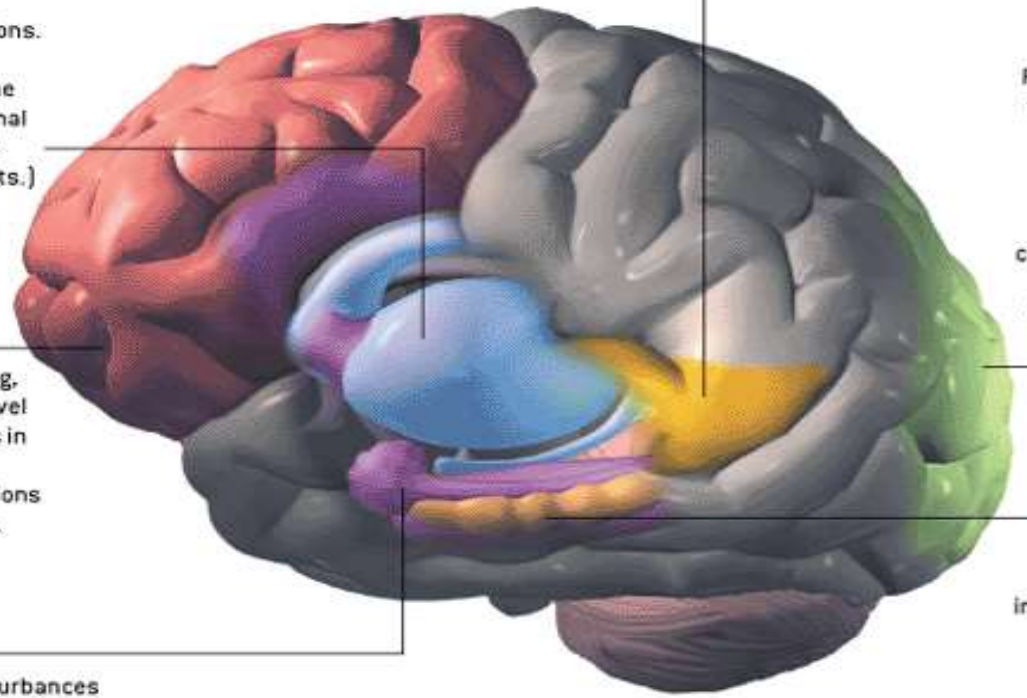
Cognitive deficits in schizophrenia





The Brain in Schizophrenia

MANY BRAIN REGIONS and systems operate abnormally in schizophrenia, including those highlighted below. Imbalances in the neurotransmitter dopamine were once thought to be the prime cause of schizophrenia. But new findings suggest that impoverished signaling by the more pervasive neurotransmitter glutamate—or, more specifically, by one of glutamate's key targets on neurons (the NMDA receptor)—better explains the wide range of symptoms in this disorder.



BASAL GANGLIA
Involved in movement and emotions and in integrating sensory information. Abnormal functioning in schizophrenia is thought to contribute to paranoia and hallucinations. (Excessive blockade of dopamine receptors in the basal ganglia by traditional antipsychotic medicines leads to motor side effects.)

AUDITORY SYSTEM
Enables humans to hear and understand speech. In schizophrenia, overactivity of the speech area (called Wernicke's area) can create auditory hallucinations—the illusion that internally generated thoughts are real voices coming from the outside.

OCCIPITAL LOBE
Processes information about the visual world. People with schizophrenia rarely have full-blown visual hallucinations, but disturbances in this area contribute to such difficulties as interpreting complex images, recognizing motion, and reading emotions on others' faces.

FRONTAL LOBE
Critical to problem solving, insight and other high-level reasoning. Perturbations in schizophrenia lead to difficulty in planning actions and organizing thoughts.

HIPPOCAMPUS
Mediates learning and memory formation, intertwined functions that are impaired in schizophrenia.

LIMBIC SYSTEM
Involved in emotion. Disturbances are thought to contribute to the agitation frequently seen in schizophrenia.

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Etiology

Exact etiology is unknown..

1- Stress-Diathesis Model

Integrates biological, psychosocial and environmental factors in the etiology of schizophrenia.

Symptoms of schizophrenia develop when a person has a specific vulnerability that is acted on by a stressful influence.

Predisposing factors → precipitating factors

2- Neurobiology

- Certain areas of the brain are involved in the pathophysiology of schizophrenia:

The limbic system, the frontal cortex, cerebellum, and the basal ganglia. (more details in the next slide)

3- Genetic Factors

- A wide range of genetic studies strongly suggest a genetic component to the inheritance of schizophrenia that outweighs the environmental influence.
- These include: family studies, twin studies and chromosomal studies.

4- Psychosocial Factors

- In family dynamics studies, no well-controlled evidence indicates specific family pattern plays a causative role in the development of schizophrenia.
- High Expressed Emotion family : increase risk of relapse.

Etiology (Neurobiology)

A. Dopamine Hypothesis: (the main)

- Too much dopaminergic activity (whether it is ↑ release of dopamine, ↑ dopamine receptors, hypersensitivity of dopamine receptors to dopamine, or combinations is not known).

B. Other Neurotransmitters:

- Serotonin, Norepinephrine, GABA, Glutamate & Neuropeptides

C. Neuropathology:

- Neuropathological and neurochemical abnormalities have been reported in the brain particularly in the limbic system, basal ganglia and cerebellum. Either in structures or connections (as shown in next slide)

D. Psychoneuro-immunology:

- ↓ T-cell interlukeukin-2 lymphocytes, abnormal cellular and humoral reactivity to neurons and presence of anti-brain antibodies.
- These changes are due to neurotoxic virus ? or endogenous autoimmune disorder ?

E. Psychoneur-endocrinology:

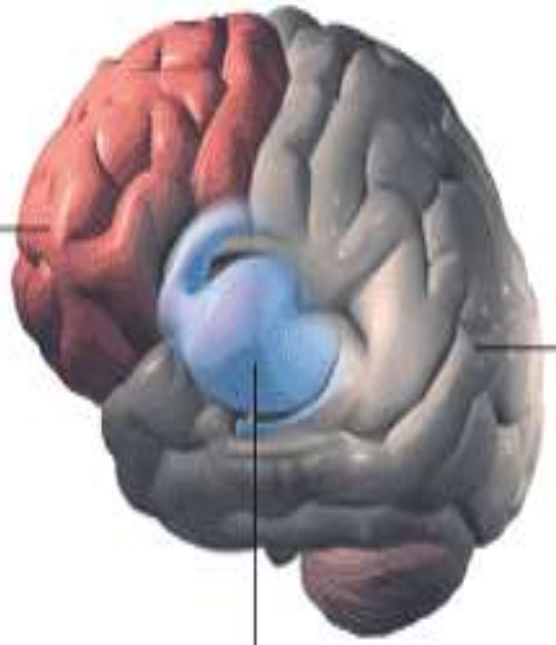
- Abnormal dexamethasone-suppression test
- ↓ LH/FSH
- A blunted release of prolactin and growth hormone on stimulation.

Different Neurotransmitters, same results

Susceptibility to schizophrenia

Schizophrenia is mostly caused by various possible combinations of many different genes (which are involved in neurodevelopment, neuronal connectivity and synaptogenesis) plus stressors from the environment conspiring to cause abnormal neurodevelopment.
There is also abnormal neurotransmission at glutamate synapses, possibly involving hypofunctional NMDA receptors .

SOME SCIENTISTS have proposed that too much dopamine leads to symptoms emanating from the basal ganglia and that too little dopamine leads to symptoms associated with the frontal cortex. Insufficient glutamate signaling could produce those same symptoms, however.



IN THE REST OF THE CORTEX, glutamate is prevalent, but dopamine is largely absent.

IN THE FRONTAL CORTEX, where dopamine promotes cell firing (by acting on D1 receptors), glutamate's stimulatory signals amplify those of dopamine; hence, a shortage of glutamate would decrease neural activity, just as if too little dopamine were present.

IN THE BASAL GANGLIA, where dopamine normally inhibits cell firing (by acting on D2 receptors on nerve cells), glutamate's stimulatory signals oppose those of dopamine; hence, a shortage of glutamate would increase inhibition, just as if too much dopamine were present.

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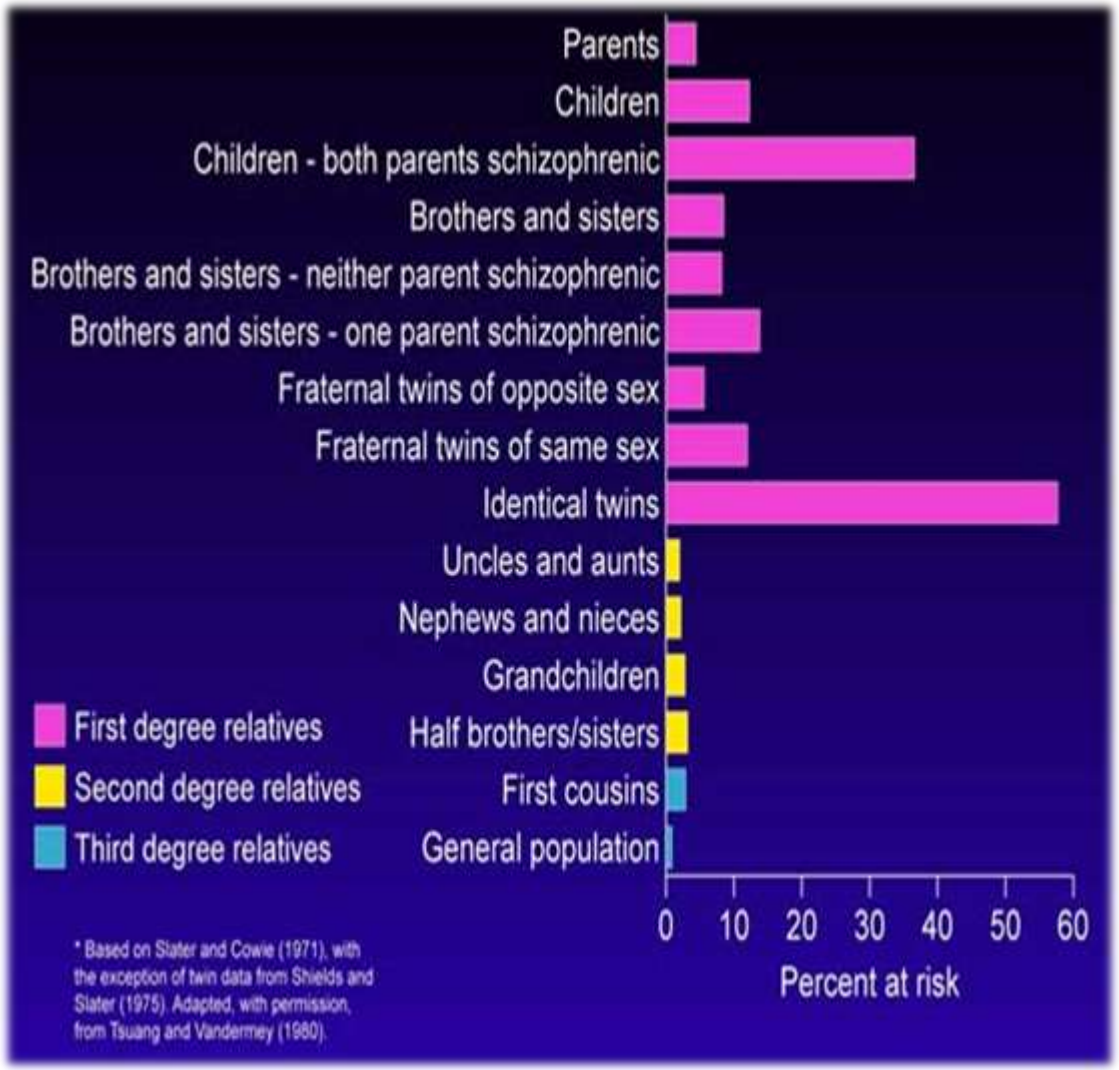
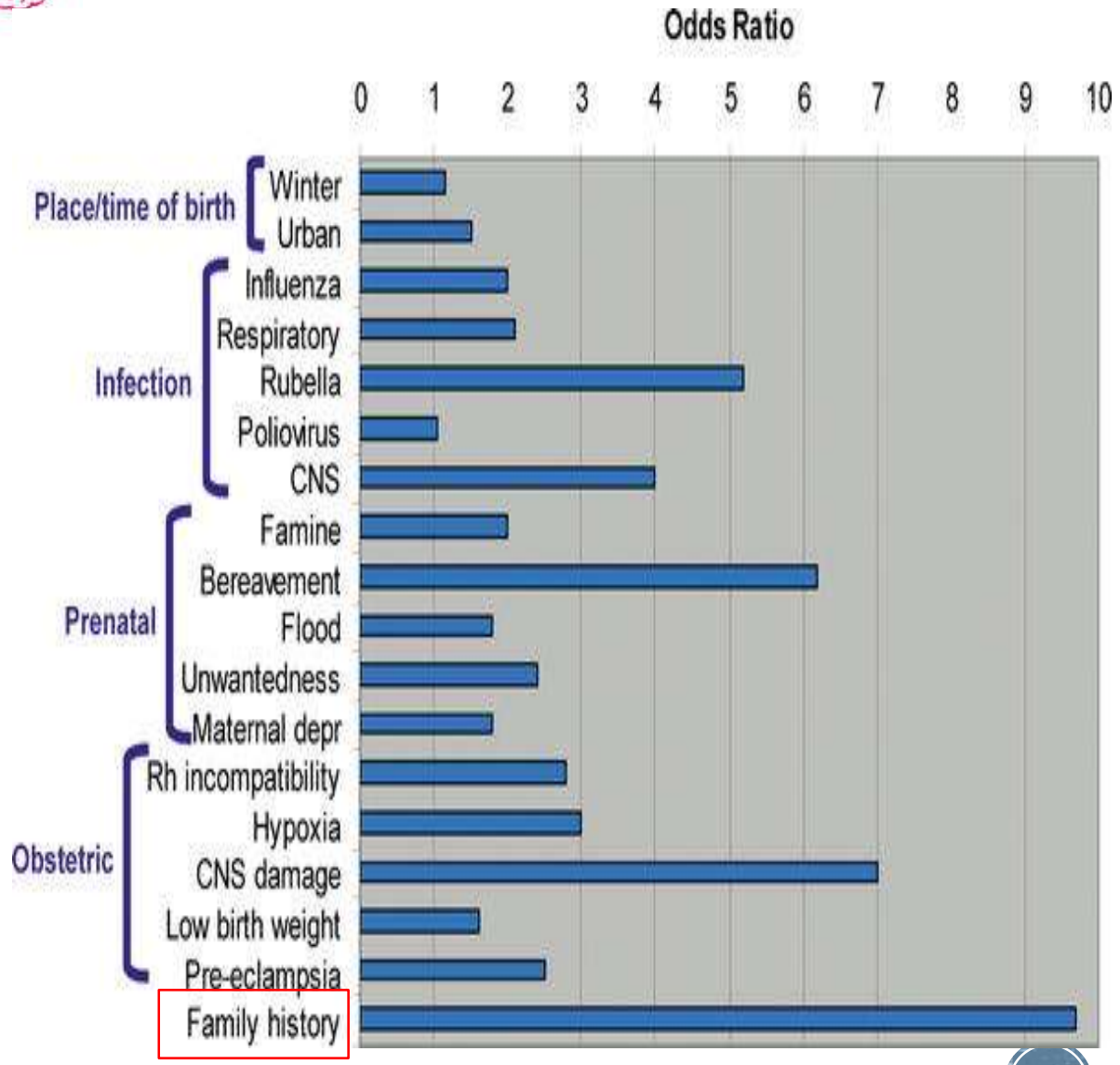
Rates of Schizophrenia among relatives of schizophrenic patients



Risk Factors



Course

- Acute exacerbation with increased residual impairment
- Full recovery: very rare
- Longitudinal course: downhill

Differential Diagnosis

Nonpsychiatric disorders:

- Substance-induced disorders
- Epilepsy (TLE)
- CNS diseases
- Trauma
- Others

Psychiatric disorders:

- Schizophreniform disorder
- Brief psychotic disorder
- Delusional disorder
- Affective disorders
- Schizoaffective disorder
- Personality disorders:

(schizoid, schizotypal & borderline personality)

- Malingering & Factitious disorders

Treatment

1. Biological therapies
2. Hospitalization
3. Psychosocial therapies

Biological therapies

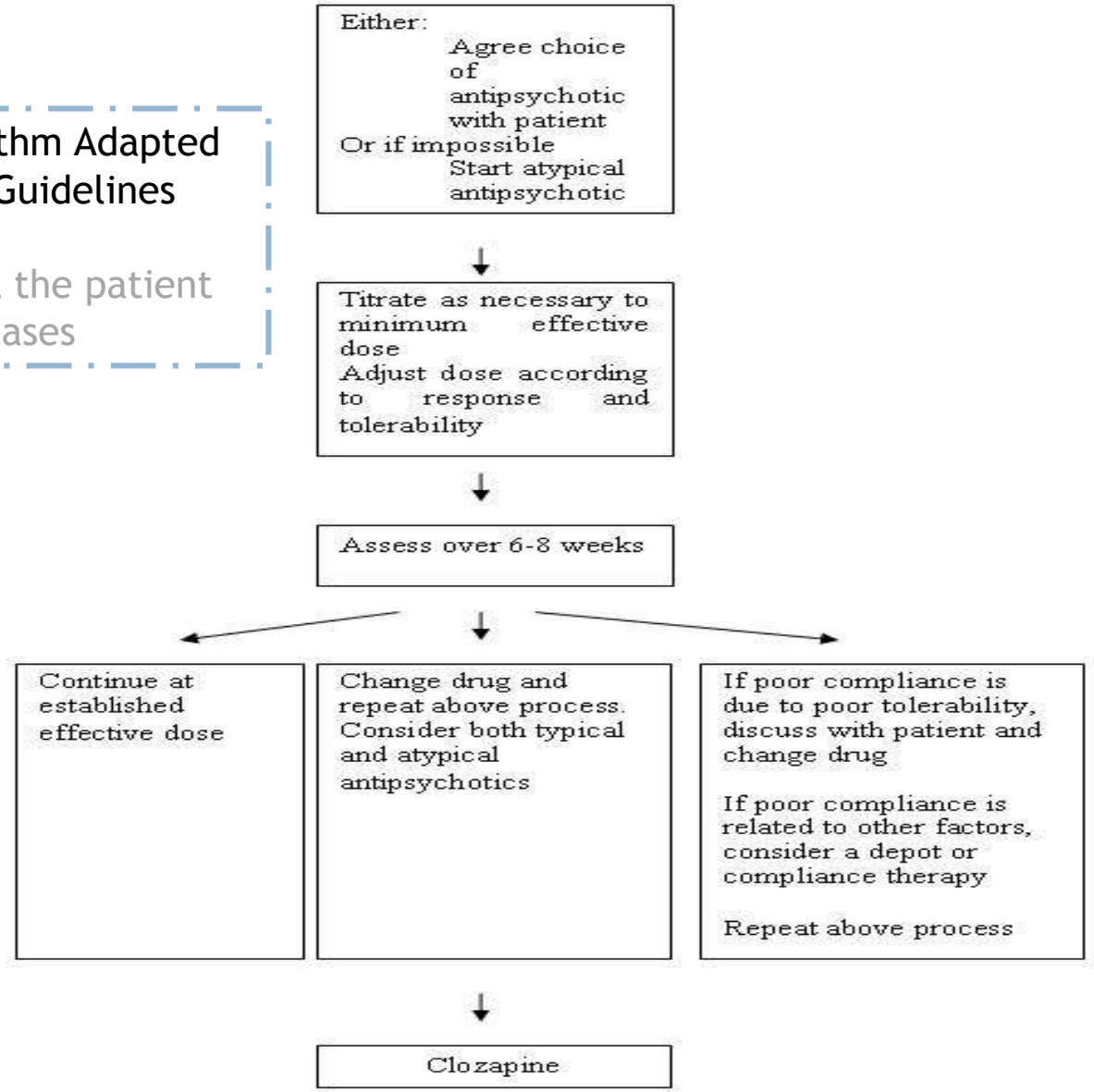
Antipsychotic medications are the mainstay of the treatment of schizophrenia.
Generally, they are remarkably safe.
Two major classes:

- ❖ Dopamine receptor antagonists (haloperidol, chlorpromazine)
- ❖ Serotonin-dopamine receptor antagonists (Risperidone, clozapine, olanzapine).
- ❖ Other drugs:
  - Anticonvulsants
  - Lithium
  - Benzodiazepines
  - Depot forms of antipsychotics eg. Risperidone Consta is indicated for poorly compliant patients
  - Electroconvulsive therapy (ECT) for catatonic or poorly responding patients to medications

Pharmacological Treatment

Pharmacological Treatment Algorithm Adapted from the Maudsley prescribing Guidelines

The algorithm is the plan to treat the patient
Clozapine for resistance cases



Common side effects of antipsychotic medication

First generation antipsychotics	*Second generation antipsychotics*	*Clozapine*
Extrapyramidal effects Dystonia Pseudoparkinsonism Akathisia Tardive dyskinesia	Olanzapine Weight gain Sedation Glucose intolerance and frank diabetes mellitus Hypotension	Sedation
Sedation		Hypersalivation
Hyperprolactinaemia	Risperidone Hyperprolactinaemia Hypotension EPS at higher doses Sexual dysfunction	Constipation
Reduced seizure threshold		Reduced seizure threshold
Postural hypotension	Amisulpiride Hyperprolactinaemia Insomnia Extrapyramidal effects	Hypo & hypertension
Anticholinergic effects Blurred vision Dry Mouth Urinary Retention	Quetiapine Hypotension Dyspepsia Drowsiness	Tachycardia
Neuroleptic malignant syndrome		Pyrexia
Weight gain		Weight gain
Sexual dysfunction		Glucose intolerance and diabetes mellitus
Cardio-toxicity (including prolonged QTc)		Nocturnal enuresis
		Rare serious side effects Neutropaenia 3% Agranulocytosis 0.8% Thromboembolism Cardiomyopathy Myocarditis Aspiration pneumonia

Hospitalization

Indications:

- Diagnostic purpose
- Patient & other's safety
- Initiating or stabilizing medications
- Establishing an effective association between patient & community supportive systems

Psychosocial therapies

Social skills training

- Family oriented therapies
- Group therapy
- Individual psychotherapy
- Assertive community treatment
- Vocational therapy

Prognosis

Good P.F	Poor P.F
1. Late age of onset 2. Acute onset 3. PPT factor 4. Presence of mood component 5. Good response to TTT 6. Good supportive system	1. Young age of onset 2. Insidious onset 3. Lack of P.T. 4. Multiple relapses 5. Low IQ 6. Pre-morbid personality 7. Negative symptom 8. Positive family history

This slide is Important

The patient should complain and his own response to the drug

Insidious onset is worse because the patient comes late so he don't take treatment early

Social support will make the prognosis better

Quiz yourself (MCQs)

* = Right answer

Q1: Define schizophrenia?

- psychological disease.
- symptom of homogeneous etiology .
- group of disorder with heterogeneous etiologies.*
- syndromes of homogeneous etiology.

Q2: Positive symptoms of schizophrenia?

- Depression
- alogia
- hallucinations *
- executive

Q3: Which one related to Factitious disorder?

-primary psychiatric disorder*
-schizophreniform disorder
-secondary psychiatric disorder
-substance-inducer

Q4: In relatives which one at highest risk ?

- parents
- children
- sister
- identical twins *

Q4: Which one is not involved with pathophysiology of schizophrenia ?

- limbic system
- cerebellum
- mid brain*
- frontal cortex

Q5: In schizoaffective disorder :

-there is a major mood episode *
-there is sharp increasing of relapses
-schizophrenia is clear
-patients could have coma

Q6: What is the mainstay treatment of schizophrenia?

-ECT
-hospitalization
-antipsychotic medication *
-antidepressants medication

Q7: In first generation antipsychotic drugs the related side effects could be :

-dyspepsia
-constipation
-sedation
-neuroleptic malignant syndrome *

Quiz yourself (SAQs)

Q8: What is schizophrenia?

It is not a single disease but a group of disorders heterogeneous etiologies

Q9: What are the disorganization features affected in schizophrenia?

-speech

-behavior

Q10: Explain the neurobiology of dopamine hypothesis related to schizophrenia:

-too much dopaminergic activity in form of =

Increase releasing of dopamine.

Increase dopamine receptors.

Hypersensitivity of dopamine receptors to dopamine.

Q11: Mention some of mental status examinations in schizophrenia:

-Appearance

-Mood,feelings

-cognitive functioning

-judgment

Q12: What is the 1st generation antipsychotic drugs related to schizophrenia?

-haloperidol

-chlorpromazine

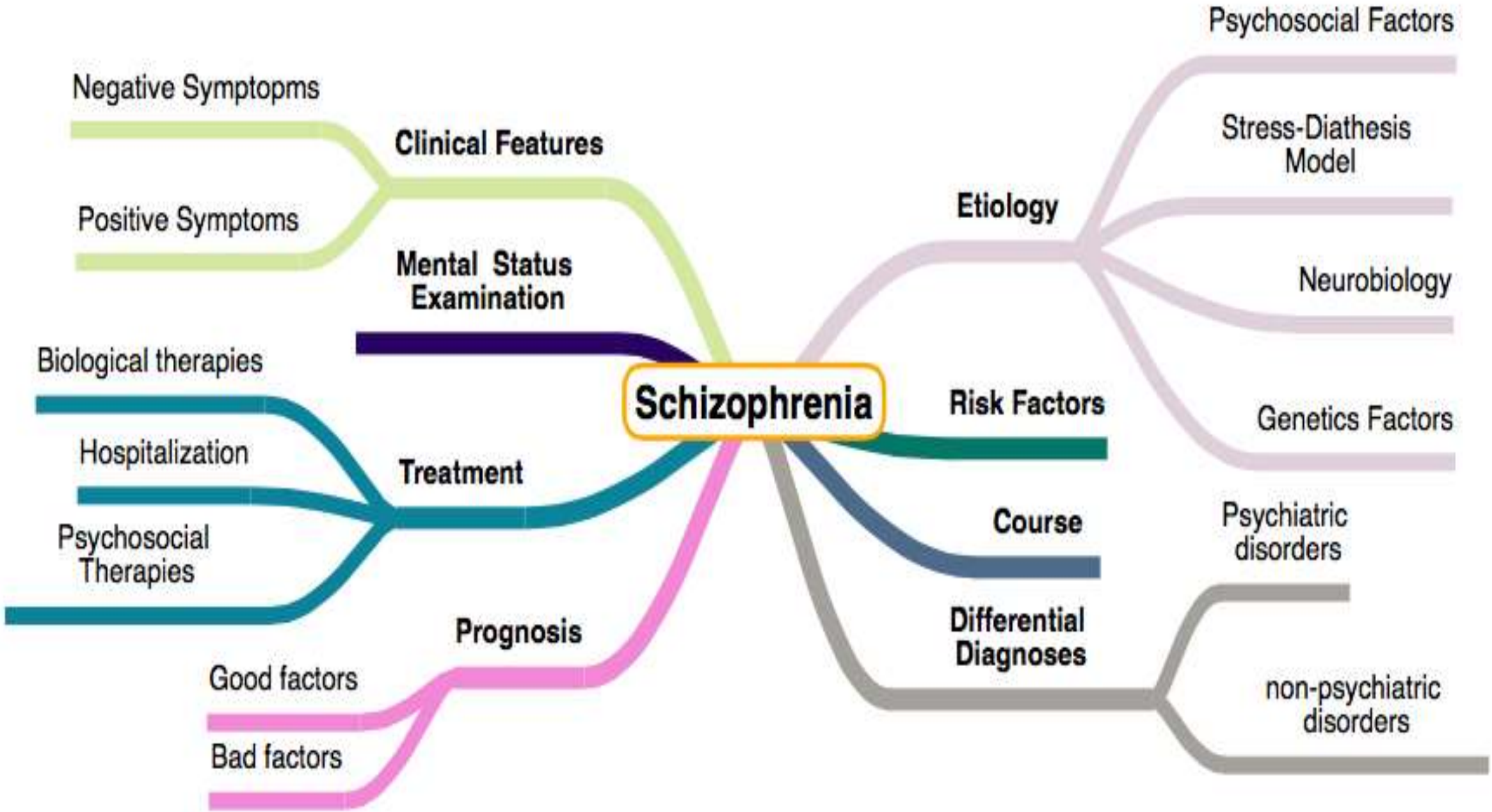
Case 1

- a 28 year-old single male who was brought to Emergency room by his family because of gradual changes in his behavior started 9 months ago. Since then, he became agitated; eat only canned food but not cooked food made by his family, afraid of being poisoned. He talks to himself and stares occasionally on the roof of his room.
- He had two brief psychiatric hospitalizations in last 3 years that were precipitated by anger at his neighbor and voices commenting about his behavior.
- His personal history indicated that he was a healthy child, but his parents reported that he was a bed wetter and seemed slower to develop than his brothers and sisters.
- he smokes tobacco frequently to calm himself. During his early adolescence he used to smokes Hash heavily plus occasional use of amphetamine. He stopped both Hash and Amphetamine use 5 years ago.

Case 2

260. A 25-year-old woman is diagnosed with schizophrenia when, after the sudden death of her mother, she begins complaining about hearing the voice of the devil and is suddenly afraid that other people are out to hurt her. Her history indicates that she has also experienced a 3-year period of slowly worsening social withdrawal, apathy, and bizarre behavior. Her family history includes major depression in her father. Which of the following details of her history leads the physician to suspect that her outcome may be poor?

a. She is female.
b. She was age 25 at diagnosis.
c. She had an acute precipitating factor before she began hearing voices.
d. She had an insidious onset of her illness.
e. There is a history of affective disorder in her family.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