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**PSYCHIATRY AND NARCOLOGY**  
**ПСИХИАТРИЯ И НАРКОЛОГИЯ**

Workbook for skills-building sessions on psychiatry and narcology  
for the 4th- and 5th-year students of the Faculty of General Medicine  
for Overseas Students

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Учебно-методическое пособие содержит информацию по пропедевтике психических и поведенческих расстройств, основных психопатологических синдромах, клинике, лечению и профилактике основных психических расстройств и поведенческих расстройств по циклу «Психиатрия и наркология» высших медицинских учебных заведений для студентов 4 и 5 курсов факультета подготовки студентов зарубежных стран (на английском языке)

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## **1. Introduction**

Psychiatry is one of the rapidly developing branches of medical knowledge. In particular, significant progress has been made towards understanding of the role of neurochemistry and brain morphology, the bio- and psychosocial model of mental disorders origin has been formed.

Instruction in psychiatry and narcology in the Faculty of General Medicine for Overseas Students is an integral part of professional training due to significant medical, economic and social effects of mental disorders.

This workbook is composed according to the typical programme of the Ministry of Health of the Republic of Belarus, Reg. no. TD-L 254/typ dated 14th June 2011 on psychiatry and narcology for higher educational institutions on speciality 1-79 01 01 “Medical business” of the Training and Methodology Association of Medical Higher Educational Institutions of the Republic of Belarus.

## 2. General psychopathology

**Topic № 1: Technological aspects of interviewing patients with mental and behavioral disorders. Curation of patients. Considering the real environment and doctor–patient relationship. Making contact and identifying goals, objectives and motives of the doctor–patient interaction. Patient self-disclosure, active listening, succession of open and closed questions, and interpretation of the patient's answers. Planning further interaction and treatment recommendations.**

The following text provides an overview of the basic components and key concepts of the psychiatric interview.

1. What are the primary aims of the first psychiatric interview?

To make an initial differential diagnosis and to formulate a treatment plan.

These goals are achieved by:

Gathering information

Chief complaint: history of current and past suicidal and homicidal ideation, history of presenting problem(s);

Current and past history of victimization (e.g., domestic violence, child abuse)

Precipitating factors: history of psychiatric problems, including treatment and response

Social and developmental history;

Family psychiatric and social history;

Mental history Symptoms (Affective Cognitive Physical);

Medical history;

Substance use and abuse;

Changes in role and social functioning.

Arriving at an empathic understanding of how the patient feels. This understanding is a critical base for establishing rapport with the patient. When the clinician listens carefully and then communicates an appreciation of the patient's worries and concerns, the patient gains a sense of being understood. This sense of being understood is the bedrock of all subsequent treatment, and allows the clinician to initiate a relationship in which an alliance for treatment can be established.

2. That's a lot to focus on in the first meeting. What about helping the patient?

The initial diagnosis and treatment plan may be rudimentary. Indeed, when patients present in a crisis, the history may be confused, incomplete, or narrowly focused. As a result, some interventions are started even when basic information about history, family relationships, and ongoing stressors is being gathered. It is

critical to remember that emotional difficulties often are isolating. The experience of sharing one's problem with a concerned listener can be enormously relieving in and of itself. Thus, the initial interview is the start of treatment even before a formal treatment plan has been established.

3. How should the initial interview be organized? There is no single ideal, but it is useful to think of the initial interview as having three components:

Establish initial rapport with the patient, and ask about the presenting complaint or problems, i.e., what has brought the patient to the first meeting. Some patients tell their stories without much guidance from the interviewer, whereas others require explicit instructions in the form of specific questions to help them organize their thoughts. During this phase of the first interview, the patient should be allowed to follow his or her own thought patterns as much as possible.

Elicit specific information, including a history of the presenting problems, pertinent medical information, family background, social history, and specific symptom and behavioral patterns. Formally test mental status.

Ask if the patient has any questions or unmentioned concerns. Initial recommendations are then made to the patient for further evaluation and/or beginning treatment.

Although the three parts of the interview can be considered separately, they often weave together, e.g., mental status observations can be made from the moment the clinician meets the patient. Pertinent medical and family history may be brought up in the course of presenting other concerns, and patients may pose important questions about treatment recommendations as they present their initial history.

4. Is the initial assessment different for complex situations?

The initial psychiatric assessment may require more than one session for complex situations – for example, when evaluating children or families, or when assessing a patient's suitability for a particular therapeutic approach, such as brief psychotherapy. The initial assessment also may require information gathering from other sources: parents, children, spouse, best friend, teacher, police officers, and/or other healthcare providers. These contacts may be incorporated into the first visit, or may occur later. The first step in making such arrangements is to explain the reason for them to the patient and to obtain explicit, written permission for the contact.

5. How should a referral source be approached?

It is almost always appropriate to call the referral source to gather information and to explain the initial diagnostic impressions and treatment plans. Exceptions may occur when the referral comes from other patients, friends, or other nonprofessionals, whom the patient wishes to exclude from treatment.

6. Are there any variations on these guidelines for an initial assessment?

Specific theoretical orientations may dictate important variations in the initial assessment. For example, a behavioral therapist guides discussion to specific analyses of current problems and spends little time on early childhood experiences. The psychopharmacologic evaluation emphasizes specific symptom patterns, responses to prior medication treatment, and family history of psychiatric illness. The approach presented in this chapter is a broadly applicable set of principles that can be used in evaluating most patients.

7. How is information gathered from an interview? The interviewer must discover as much as possible about how the patient thinks and feels. During the clinical interview, information is gathered from what the patient tells the interviewer; critically important clues also come from how the history unfolds. Thus, both the content of the interview (i.e., what the patient says) and the process of the interview (i.e., how the patient says it) are important routes to understanding the patient's problems. Consider the order of information, the degree of comfort in talking about it, the emotions associated with the discussion, the patient's reactions to questions and initial comments, the coherence of the presentation, and the timing of the information. The full elaboration of such information may take one or several sessions over the course of days, weeks, or months, but in the first interview hints of deeper concerns may be suggested.

For example, a 35-year-old woman presented with worries about her son's recurrent asthma and associated difficulties in school. She talked freely about her worries and sought advice on how to help her son. When asked about her husband's thoughts, she became momentarily quiet. She then said that he shared her concern and switched the discussion back to her son. Her hesitancy hinted at other problems, which were left unaddressed in the initial session. Indeed, she began the next session by asking, "Can I talk about something else besides my son?" After being reassured, she described her husband's chronic anger at their son for his "weakness." His anger and her feelings in response became an important focus of subsequent treatment.

8. How should the interview be started? The here and now is the place to begin all interviews. Any one of a number of simple questions can be used: "What brings you to see me today? Can you tell me what has been troubling you? How is it that you decided to make this appointment?" For anxious patients, structure is useful: early inquiry about age, marital status, and living situation may give them time to become comfortable before embarking on a description of their problems. If the anxiety is evident, a simple comment about the anxiety may help patients to talk about their worries.

9. Is a highly structured format important? No. Patients must be given some opportunity to organize their information in the way that they feel most comforta-

ble. The interviewer who prematurely subjects the patient to a stream of specific questions limits information about the patient's own thinking process, does not learn how the patient handles silences or sadness, and closes off the patient's opportunities to hint at or introduce new topics. Furthermore, the task of formulating one specific question after another may intrude on the clinician's ability to listen and to understand the patient. This does not mean that specific questions should be avoided. Often, patients provide elaborate answers to specific questions such as "When were you married?" Their responses may open new avenues to the inquiry. The key is to avoid a rapid-fire approach and to allow patients to elaborate their thoughts.

10. How should questions be asked? Questions should be phrased in a way that invites patients to talk. Open-ended questions that do not indicate an answer tend to allow people to elaborate more than specific or leading questions. In general, leading questions (e.g., "Did you feel sad when your girlfriend moved out?") can be conversation stoppers, because they may give the impression that the interviewer expects the patient to have certain feelings. Non-leading questions ("How did you feel when your girlfriend moved out?") are as direct and more effective.

11. What is an effective way to deal with patient hesitancy? When patients need help in elaborating, a simple statement and/or request may elicit more information: "Tell me more about that." Repeating or reflecting what patients say also encourages them to open up (e.g., "You were talking about your girlfriend."). Sometimes comments that specifically reflect the clinician understands of the patient's feelings about events may help the patient to elaborate. This approach provides confirmation for both the interviewer and the patient that they are on the same wavelength. When the interviewer correctly responds to their feelings, patients frequently confirm the response by further discussion. The patient whose girlfriend left may feel understood and freer to discuss the loss after a comment such as "You seem discouraged about your girlfriend moving out."

12. Give an example of how comprehensive information gathering can pinpoint a problem. An elderly man was referred for increasing despondency. In the initial interview, he first described financial difficulties and then brought up the recent development of medical problems, culminating with the diagnosis of prostatic carcinoma. As he began talking about the cancer and his wish to give up, he fell silent. At this point in the interview, the clinician expressed his recognition that the patient seemed to feel overwhelmed by the build up of financial and, most of all, medical reversals. The patient nodded quietly and then elaborated his particular concerns about how his wife would get on after he died. He did not feel that his children would be helpful to her. It was not yet clear whether his pessimism reflected a depressive overreaction to the diagnosis of cancer or an accurate appraisal



of the prognosis. Further assessment of his symptoms and mental state and a brief discussion with his wife later in the meeting revealed that the prognosis was quite good. The treatment then focused on his depressive reactions to the diagnosis.

13. How are questions best worded? The interviewer should use language that is not technical and not overly intellectual. When possible, the patient's own words should be used. This is particularly important in dealing with intimate matters such as sexual concerns. People describe their sexual experience in language that is quite varied. If a patient says that he or she is gay, use that exact term rather than an apparently equivalent term such as homosexual. People use some words and not others because of the specific connotations that different words carry for them; at first, such distinctions may not be apparent to the interviewer.

14. What about patients who are unable to communicate coherently? The interviewer must remain aware at all times of what is going on during the interview. If the patient is hallucinating or intensely upset, failure to acknowledge the upset or the disconcerting experience may elevate the patient's anxiety. Discussing the patient's current upset helps to alleviate tension and tells the patient that the clinician is listening. If the patient's story rambles or is confusing, acknowledge the difficulty of understanding the patient and evaluate the possible reasons (e.g., psychosis with loosened associations vs. anxiety about coming to the visit). When general questions (e.g., "Tell me something about your background.") are ineffective, it may be necessary to ask specific questions about parents, schooling, and dates of events. Realize, however, that it can be tempting to ask endless questions to alleviate your own anxiety rather than the patient's.

15. Summarize key points to remember about the initial interview. Allowing the patient freedom to tell his or her own story must be balanced by attending to the patient's ability to focus on relevant topics. Some people require guidance from the interviewer to avoid getting lost in tangential themes. Others may need consistent structure because they have trouble ordering their thoughts, perhaps due to a high degree of anxiety. An empathic comment about the patient's anxiety may reduce it and thus lead to clearer communication.

#### Some Interviewing Guidelines

- Let the first part of the initial interview follow the patient's train of thought.
- Provide structure to help patients who have trouble ordering their thoughts or to finish obtaining specific data.
- Phrase questions to invite the patient to talk (e.g., openended, non-leading questions).
- Use the patient's words.

- Be alert to early signs of loss of behavioral control (e.g., standing up to pace).
- Identify the patient's strengths as well as problem areas.
- Avoid jargon and technical language.
- Avoid questions that begin with "why".
- Avoid premature reassurance.
- Do not allow patients to act inappropriately (e.g., break or throw an object).
- Set limits on any threatening behavior, and summon help if necessary.

#### 16. What specific pitfalls should be avoided during the initial interview?

Avoid jargon or technical terms, unless clearly explained and necessary. Patients may use jargon, for example, "I was feeling paranoid." If patients use a technical word, ask about their meaning for the term. You may be quite surprised by the patient's understanding. For example, patients may use "paranoid" to suggest fear of social disapproval or pessimism about the future. Also, be careful about assigning a diagnostic label to the patient's problems during the interview. The patient may be frightened and confused by the label.

In general, avoid asking questions that begin with "why". Patients may not know why they have certain experiences or feelings, and can feel uncomfortable, even stupid, if they believe their answers aren't "good". Asking why also implies that you expect the patient to provide quick explanations. Patients discover more about the roots of their problems as they reflect on their lives during the interview and in subsequent sessions. When tempted to ask why, rephrase the question so that it elicits a more detailed response. Alternatives include "What happened?" "How did that come about?" or "What thoughts do you have about that?"

Avoid premature reassurance. When patients are upset, as they often are during first inter-views, the interviewer may be tempted to allay the patient's fear by saying "Everything will be fine" or "There is nothing really seriously wrong here." However, reassurance is genuine only when the clinician (1) has explored the precise nature and extent of the patient's problems and (2) is certain of what he or she is telling the patient. Premature reassurance can heighten the patient's anxiety by giving the impression that the clinician has jumped to a conclusion without a thorough evaluation or is just saying what the patient wants to hear. It also leaves patients alone with their fears about what is really wrong. Furthermore, premature reassurance tends to close off discussion rather than encourage further exploration of the problem. It may be more reassuring to ask what the patient is concerned about. The process (i.e., the nature of the interaction) comforts the patient more than any single thing the interviewer may say. Set limits on behavior. Because of their psychiatric problems, some patients may lose control in the session. Although the approach described here emphasizes letting the patient direct much of the verbal dis-

cussion, at times limits must be set on inappropriate behavior. Patients who are aroused and want to take off their clothes or threaten to throw an object need to be controlled. This goal is most often accomplished by commenting on the increasing arousal, discussing it, inquiring about sources of upset, and letting patients know the limits of acceptable behavior. On rare occasions outside help may be necessary (e.g., security guards in an emergency department), especially if the behavior is escalating, and if the interviewer senses danger. The interview should be stopped until the patient's behavior can be managed so that it is safe to proceed.

17. What is commonly forgotten in evaluating patients? The new patient initiates contact with the clinician because of problems and worries; these are the legitimate first topics of the interview. It also is helpful to gain an understanding of the patient's strengths, which are the foundation on which treatment will build. Strengths include ways in which the patient has coped successfully with past and current distress, accomplishments, sources of inner value, friendships, work accomplishments, and family support. Strengths also include hobbies and interests that patients use to battle their worries. Questions such as "What are you proud of?" or "What do you like about yourself?" may reveal such information. Often the information comes out as an afterthought in the course of conversation. For example, one patient took great pride in his volunteer work through the church. He mentioned it only in passing as he discussed his activities of the week before the meeting. Yet this volunteer work was his only current source of personal value. He turned to it when he became upset about his lack of success in his career.

18. What is the role of humor in the interview? Patients may use humor to deflect the conversation from anxiety-provoking or troubling topics. At times, it may be useful to allow such deviations to help patients maintain emotional equilibrium. However, probe further if the humor seems to lead to a radical change in focus from a topic that seemed important and/or emotionally relevant. Humor also can direct the interviewer toward new areas for investigation. A light joke by the patient (e.g., about sex) may be the first step in introducing a topic that later takes on importance. On the part of the interviewer, humor may be protective and defensive. Just as the patient can feel anxious or uncomfortable, so can the interviewer. Be careful, because humor can backfire. It may be misunderstood as ridicule. It also can allow both patient and interviewer to avoid important topics. Sometimes humor is a wonderful way to show the human qualities of the interviewer and thus build a therapeutic alliance. Nonetheless, keep in mind the problematic aspects of humor, especially when you and your patient don't know each other well.

19. How is suicidal intent assessed? Because of the frequency of depressive disorders and their association with suicide, it always is necessary to address the possibility of suicidal intent in a first interview. Asking about suicide will not pro-

voke the act. If the subject does not arise spontaneously, several questions can be used to draw out the patient's thoughts on suicide (listed in the order that they may be used for beginning a discussion):

- How badly have you been feeling?
- Have you thought of hurting yourself?
- Have you wanted to die?
- Have you thought of killing yourself?
- Have you tried?
- How, when, and what led up to your attempt?
- If you have not tried, what led you to hold back?
- Do you feel safe to go home?
- What arrangements can be made to increase your safety and to decrease your risk of acting on suicidal feelings?

Such discussion may need to be extended until it is clear whether the patient may safely leave or needs hospital admission).

20. What is the best way to bring a first evaluation interview to a close? One way is to ask the patient if he or she has any specific questions or concerns that have not been addressed. After addressing such issues, briefly summarize important impressions and diagnostic conclusions and then suggest the course of action. Be as clear as possible about the formulation of the problem, diagnosis, and next steps. This is the time to mention the need for any tests, including laboratory examinations and further psychological assessments, and to obtain permission for meeting or talking with important others who may provide needed information or should be included in the treatment plan.

Both clinician and patient should recognize that the plan is tentative and may include alternatives that need further discussion. If medication is recommended, the clinician should describe the specific benefits and expected time course as well as inform the patient about potential side effects, adverse effects, and alternative treatments. Often patients want to think over suggestions, get more information about medications, or talk with family members. In most instances, the clinical situation is not so clear that action must be taken in the first interview. However, be clear in presenting recommendations, even if they are tentative and primarily oriented to further diagnostic assessment.

At this point it is tempting to provide false reassurance, such as "I know everything is going to be okay." It is perfectly legitimate – and indeed better – to allow for uncertainty when uncertainty exists. Patients can tolerate uncertainty, if they see that the clinician has a plan to elucidate the problem further and to arrive at a sound plan for treatment.

## Topic № 2

a) **Disturbances of Perception: Delusions and hallucinations. Difference between true hallucinations and pseudohallucinations.**

b) **Memory disorders. Korsakoff Syndrome.**

c) **Psychopathology of emotions. The symptoms of emotional disorders. Depressive and manic syndromes.**

a) **Disturbances of Perception: Delusions and hallucinations. Difference between true hallucinations and pseudohallucinations.**

- Perception is a process of becoming aware of what is presented through the sense organs

- Imagery means an experience within the mind, usually without the sense of reality that is part of reality

- Pseudoillusions – distorted perception of objects which may occur when the general level of sensory stimulation is reduced

- Illusions are psychopathological phenomena; they appear mainly in conditions of qualitative disturbances of consciousness (missing insight)

- Hallucinations are percepts without any obvious stimulus to the sense organs; the patient is unable to distinguish them from reality. These sensory impressions are generated by the mind rather than by any external stimuli, and may be seen, heard, felt, and even smelled or tasted. A hallucination occurs when environmental, emotional, or physical factors such as stress, medication, extreme fatigue, or mental illness cause the mechanism within the brain that helps to distinguish conscious perceptions from internal, memory-based perceptions to misfire. As a result, hallucinations occur during periods of consciousness. They can appear in the form of visions, voices or sounds, tactile feelings (known as haptic hallucinations), smells, or tastes.

### **Hallucinations:**

- auditory (acousma, voices)
- visual
- olfactory
- gustatory
- tactile (or deep somatic)
- extracampine, inadequate
- intrapsychic (belong rather to disturbances of thinking)
- hypnagogic and hypnopompic (hypnagogic)

Pseudohallucinations – patient can distinguish them from reality

### **Clinical distinction of pseudo and true hallucinations**

<b>symptom</b>	<b>True hallucinations</b>	<b>pseudo hallucinations</b>
realization of the hallucinatory image	has a sense of objectivity and reality, pt. perceives it as a reality	a patient perceives hallucination as something subjective and abnormal, hallucination are distinct from real images, phantasies and true hallucinations
judgment on the means of perception hallucinatory image	conviction in usual way of "perception" through one of the analyzers	conviction in unusual "perception" through non-existing analyzer ("inner ear/eye")
Identification of the hallucinatory image with a real one	total identification with alienation of hallucinatory image	Absent, hallucinatory image holds special qualities allowing the subject to differentiate it from reality
projection of the hallucinatory image	as a rule, projection is into material, physical world within the reach of the analyzer	As a rule, projection is into subjective world, frequently out of the analyzer's reach
"feeling of madeness"	always absent	always present
Actual behavior (concordance of behavior to hallucinatory experience)	Observed most cases	behavior is almost always dissociated with the hallucinations content
conviction that others see the same images	Present in most cases	almost always absent
hallucinatory image is dangerous	Commonly, hallucinatory images present danger to patients' and their relatives' lives, health	Commonly, hallucinatory images present danger to patient psychic
Daily fluctuation of the symptom	usually, hallucinatory experiences increase and can reach twilight state at the evening, night time	usually absent
course	Frequently acute, relatively transient	Frequently chronic, sub-acute, lingering

True hallucinations are often the symptoms of irritation of the cortical division of the analyzer (brain tumors, severe intoxication, traumatic brain injury, etc.). They more often than pseudohallucinations form critical attitude (especially if they occur on the background of full consciousness).

Pseudohallucinations – reflect endogenous disturbances on integrative processes in cognitive sphere.

**b) Memory disorders. Korsakoff Syndrome.**

Memory is a process of storing information and experiences. It is a main mechanism of adaptation which makes us able to hold psychological phenomena like, obtaining feelings, emotions, doing something, some actions for a long time in the brain. The work of memory is connected with the main elements of process of perception and thought, like representation and understanding. It is the basic ground of work of intellect.

Memory can be of two types:

1. Short term memory (memory of recent events up to 3 – 5 months)
2. Long term memory (memory of past events, from childhood).

● Short term memory in most of the cases is the first to be affected. As a rule, in case of memory loss patient tends to forget the most recent memories first. Like an old man can remember his acquired knowledge from university but can't remember if he ate his breakfast. In the next step, he may forget his knowledge that he obtained in university or at work, but remembers events in his childhood. In the end the childhood memory may also be lost but he knows his name, surname, street address (where he lived at his childhood). At the last stage he even can't remember his name. But in practice we seldom see this step. (even patients suffering from Alzheimer s, could tell their names). Disorder of memory in most of the cases is related to organic defect of brain. But sometimes it may also be secondary to other psychological disorder. Therefore it is important to include the attention, consciousness of the patient during grading his memory.

● Short – term memory (working memory) – for verbal and visual information, retained for 15 – 20 sec., low capacity

● Long-term memory – wide capacity and more permanent storage

● declarative (explicit) memory – episodic (for events) or semantic (for language and knowledge)

● procedural memory – for motor arts

● priming – unconscious memory

● conditioning – classic or emotional

Disorders of memory:

● Amnesia – inability to recall past events

● Jamais vu, déjà vu

- Confabulation, amnesic disorientation, Korsakoff syndrome
- Hypomnesia
- Hypermnesia

### **Disorders of memory:**

Disorder of memory conditionally may be divided in dismnnesia and paramnesia.

- Dismnesia contains hypermnesia, hypomnesia, and different types of amnesia.
- Hypermnesia is a nonproductive, some unfairly actualization of past experiences. A flood of memory about accidentally occurred situations which had negligible effect on life, doesn't improve productivity of thinking, but merely distracts the patient and disturbs him to obtain new information. Hypermnesia is seen in mania episodes or sometimes is seen in disorders of consciousness. It is also observed in case of intake of psychotropic drugs (marijuana, LSD, opioids, amphetamines etc.), or accompanied by epileptic paroxysm.

- Hypomnesia is general weakening of memory. In this case the patient remembers new names, dates, with difficulty and forgets details about events. The patients have to write the important information to remember them, without these notes they cannot remember. During reading a book, he has to return at previous pages to remember and connect what he is reading now. Hypomnesia is often accompanied by a symptom: anecphoria (greek word) i.e. when the patient cannot remember names, words, unless he is given a clue or hint. Hypomnesia is related to broad spectrum organic diseases of brain (basically vascular), sometimes it is seen in functional disorders of psychology e.g. in condition of fatigue (asthenic syndrome).

- Amnesia is a row of diseases characterized by loss of part of memory.

- Retrograde amnesia is loss of memory till the beginning of the disease (in most of the cases it is connected with acute brain catastrophe with loss of consciousness). A part of memory of the past is lost in this case.

**Case example:** patient, age 42, was born at place A, well settled, married at place B and lived there for 15 years with his wife and 2 sons. After divorce went back to place and worked as driver. Married again and had a son from the 2nd marriage. One evening the patient did not return home from work. He was found senseless under the bridge on the next morning. He was sent to ICU for 10 days. When he was conscious again, couldn't remember anything about the trauma. After that it was found that he couldn't remember last few years, he didn't remember that he divorced his 1st wife, came back to place A to live, didn't know anything about his 2nd marriage. But clearly remember about his last life at place A, told about place A and how he lived there. While his 2nd wife came to visit him, he recognized her, but called her by the name of his 1st wife.



In case of brain trauma, the patient practically does not forget his name, age, memories of childhood etc. The loss of basic information about the personality of the patient is related to psychogenic sources, which is called hysterical amnesia. Hypnosis can cure this symptom.

Anterograde amnesia is loss of memory after the onset of disease (after restoring consciousness). In this case, the patient is available for contact, he answers the questions but cannot remember the fragments of the events occurred just beforehand. The cause of anterograde amnesia is obscured consciousness and twilight state. In this case the ability to fix the events in memory may be restored with times. But in case of Korsakoff Syndrome (you will read about it later) anterograde amnesia comes as a complete loss of ability to fix events in memory.

Fixation amnesia is sudden decrease or total loss of ability to store something for some-times in memory. These patients cannot remember anything that they just heard, or saw, or read (minute memory). As they remember events till the onset of disease, they may lead professional lives. Ability to intellectual actions is also preserved. Along with these the disorder of memory leads to very rough disorientation of patient in any new situation, that the patient himself cannot continue any labor function. It is seen in chronic vascular disorder of brain (atherosclerotic dementia) and in sudden brain catastrophes (intoxication, trauma, asphyxia, stroke).

In case of progressive amnesia the loss of memory is due to progressive organic brain disorders. As a rule, here, at first the ability to memorizing is lost (hypomnesia). Then patient tends to forget recent events. Then the long term memory is affected. This includes organized (learned and abstract) memory. At last emotional experiences and practical acquired habits are lost from memory. Patient may have some fragments of childhood memory left. Progressive amnesia is seen in case of diseases like atherosclerosis of vessels in brain (in absence of stroke), Alzheimer s disease, Pick s disease, and senile dementia.

Paramnesia is distortion or perversion of contents of memory. It includes mainly pseudoreminiszenz and confabulation.

Pseudoreminiszenz: this is filling of gap of memory by real experience but of other time period from his past life.

**Case example:** patient, hospitalized for last 2 months, during conversation with a medical student says that on the previous day she made dinner, helped her grandson to do homework.

Confabulation is unintentionally or unconsciously filling of gap of memory by imagined or untrue experiences that patient believes but has no base in fact.

**Case example:** patient, age 55, hospitalized for last 6 months, during conversation with a health care provider says that she on the previous Sunday, presi-

dent Putin declared war against U.S. she is afraid because she saw the war in 1944 and it was a horrible experience for her.

### **Korsakoff Syndrome:**

Before we learn about Korsakoff Syndrome (also called Korsakoff psychosis), we have to learn some basic things about how memory functions. There are 3 steps. The 1st step is called registration or fixation (give your patient 10 simple words and immediately ask them to see how he remembers). The 2nd step is retention. This is storage of information. The 3rd step is reproduction which is output of the information. Observing the level of reproduction a doctor can understand the severity of damage of retention and fixation.

This syndrome was written as a manifestation of specific alcoholic psychosis. But the clinical picture can be observed in different types of organic diseases of brain.

One of the main symptoms of Korsakoff syndrome is fixation amnesia. The severity of fixation disorder disturbs patient in not only to remember the contents of consultation with the doctor but also the fact that there was a consultation. Even staying many days in hospital, patient cannot remember his doctor and the neighbor patients. Even if he writes the notes, he cannot remember, he even cannot remember that he wrote.

In this case, the patient cannot remember anything write from the onset of the disease, i.e. he has anterograde amnesia (2nd symptom). Korsakoff syndrome can be seen often after acute brain catastrophes, so retrograde amnesia (2nd symptom) can also be present along with anterograde amnesia. Together, this symptom is known as retroanterodrade amnesia. The patient mixes up the organization in memory blank gap. This is paramnesia (pseudoreminizsenz and confabulation as 3rd symptom).

The sudden disorder of memory leads to disturbance of orientation. This is called amnesic disorientation (the 4th symptom). Amnesic disorientation is different from obscured consciousness as here the patient has no difficulty with getting information from the surroundings, his intellect is preserved and the previous experiences helps him to interpret the situation correctly. The patient doesn't feel disoriented in familiar places. But in the hospital he is helpless. He cannot find his ward, his bed and toilet.

**Case example:** patient, age 49, with a positive history of alcoholism, after having a delirium tremens suffered from severe memory disorder. He could remember nothing from the onset of the disease, and even many other facts like he was divorced for last 1 year. At the ward, he often became very aggressive and complained that his wife rarely visited him. When the ex-wife asked about some stuffs that she brought in her last visit, the patient told that they were brought by his colleagues. At first he couldn't orient himself in the hospital. Often he entered

in other wards. After 2 months he adapted to the rules of the ward, memorized the name of his doctor, and even went for walks with other patients. Once he decided to walk alone. But he was lost on the street. For 3 hrs., he loitered on the streets and tried to find the way back to hospital. He asked the passers-by and then he understood that he was merely loitering in the hospital campus.

**c) Psychopathology of emotions. The symptoms of emotional disorders. Depressive and manic syndromes.**

### **Emotion definition and differentiation**

- Aristotle: People are THINKING ANIMAL. What makes people special is they can overcome their brutish emotions.

- Rousseau: Emotions are what makes people special and gives us reason for living.

- Hippocrates: Brain is the site of emotion

- The word emotion is derived from the latin word *emovere* which means to stir up to get agitated. English word “emotion” dates back to 1579, when it was adapted from the French word *émouvoir*, which means “to stir up”.

Emotion it is one of the most important mechanisms of psychological actions.

It is characterized by productive subjective feelings after input of signals, prosperity of internal condition of human beings and reaction at external situations. Emotions can be negative and positive. Positive emotions are happiness, enjoyment, relaxation, love, comfort etc. negative emotions are sadness, sorrow, fear, anxiety, hatred, discomfort, anger etc. In this case the quantitative characteristic of emotion should also be positive and negative, mild and severe. Affect is external expression of emotion, i.e. mimic, gesticulation, intonation, vegetative reaction. So in psychiatry emotional and affective, these two terms are used as synonyms. Emotion is also characterized by some dynamic signs. Prolonged emotional condition corresponds to mood.

### **There are three basic functions of emotions:**

1. Signaling. Signaling allows grading a situation faster than the logical analyzing.

2. Communicativeness. It helps us to interact with other people and do an action accordingly. Collective action of people suggests emotions like sympathy, empathy, cruelty etc.

3. Formation of behavior. Emotion basically allows grading noticeable demands of human beings and pushes for its realization. Like feeling of hunger ends in search of food

Emotion usually conceptualized as a complex feeling state with psychic, somatic, autonomic and behavioral components. Emotions have been described as

consistent responses to internal or external events which have a particular significance for the organism.

Emotions are brief in duration and consist of a coordinated set of responses, which may include

- verbal,
- physiological,
- behavioral,
- and neural mechanisms.

**Emotion has two components:**

- Mental
- Physical

**Mental component:**

- Cognition – Awareness of sensation and its cause.
- Affect – The feeling itself.
- Conation – urge to take action.

**Physical components:**

- Changes in viscera and skeletal muscle
- Coordinated activity of autonomic and somatic nervous system
- Example: tachycardia, tachypnoea, cutaneous vasoconstriction etc. in fear

Description of emotions often accompanied by terms:

**Feelings** are best understood as a subjective representation of emotions, private to the individual experiencing them.

**Moods** are diffuse affective states that generally last for much longer durations than emotions and are also usually less intense than emotions.

**Affect** is an encompassing term, used to describe the topics of emotion, feelings, and moods together, even though it is commonly used interchangeably with emotion.

Five components of emotion according Scherer's processing model of emotion.

- Cognitive appraisal: provides an evaluation of events and objects
- Bodily symptoms: the physiological component of emotional experience
- Action tendencies: a motivational component for the preparation and direction of motor responses.
- Expression: facial and vocal expression almost always accompanies an emotional state to communicate reaction and intention of actions
- Feelings: the subjective experience of emotional state once it has occurred

**Current theory**

- No single neural system produces emotions

- Different emotions may depend on different neural circuits, but many of these circuits converge in the same parts of the brain
- The limbic system may be involved in some emotional experiences, but it is not the sole neural system underlying emotion
- Feelings (emotion) result from the interplay between: The amygdala, hypothalamus, brain stem & autonomic nervous system.
- Current overall approach to emotion is human emotions are biological, psychological and sociological in nature. Past historical accounts of emotions have been discourteous and avoidant. The contemporary neuroscience believes that emotions are not trivial indulgences or invaders that interfere with logical thinking, but they are prime organizing methods where awareness, understanding, and memory are established.
- If the message people sense in a situation fails to evoke an emotional reaction, it will also fail to be regarded as significant and will have little likelihood of being selected into long-term memory.
- Investigations are also confirming that for someone to learn new ways of adapting they must possess a desire about what they are attempting to learn.
- Appraisals that lead to emotions, attitudes toward emotions, emotion labels, emotion concepts, and emotion expressions vary across cultures.
- As early as infancy, individuals begin to develop a characteristic style of expressing emotions, and the frequency of expression of various discrete emotions tends to remain stable over time.

#### **Additional research on emotions and psychopathology**

The functions of emotions in persons with psychopathology remain comparable to these functions in normal individuals Davidson, 1992; Davidson & Tomarken, 1989; Gray, 1979, 1982, 1995. Disturbance in any one of these components (“perception, experience, intensity, or display”) can impair a person’s “ability to achieve one or more emotion functions in an adaptive fashion”. Kring and Bachorowski (1999) discussed the relations of depression, anxiety disorders, psychopathy, and Schizophrenia to various disturbances in emotion processing and, in particular, to the hypothesized behavior activation and behavioral inhibition systems (Gray, 1978, 1995) and the conceptually similar approach and withdrawal motivation systems (Davidson, 1994).

Depression relates to deficits in the approach motivation system (Depue, Krauss, & Spont, 1987), anxiety disorders to disturbances in the withdrawal motivation system (Barlow, 1988; Gray, 1978), psychopathy to dysfunction in both the approach/behavioral activation system (strong) and withdrawal/inhibition system (weak), and schizophrenia to problems in both the activation/approach systems and the inhibition/withdrawal systems (Fowles, 1994).

Kring and Bachorowski note that a number of pathological conditions cannot be explained in terms of dysfunction in a single motivation system, a single dimension of emotionality, or a single component of emotion processes. For example, depression may include a combination of high negative affect and low positive affect, schizophrenia a diminished emotion expression and possibly diminished emotion experience as well (Earnst & Kring, 1999), and psychopathy a discordance between emotion experience and its verbal articulation (Cleckley, 1941) as well as disjunction between components of the emotion process. These reviews document the complex and highly significant role of emotions in psychopathology.

### **Emotions, Culture, and Socialization Processes**

Appraisals that lead to emotions, attitudes toward emotions, emotion labels, emotion concepts, and emotion expressions vary across cultures (Izard, 1971; Markus & Kitayama, 1991; Matsumoto, 1990), and these differences may cause variations in the relations between emotions and psychopathology.

### **Expressing Emotion and psychopathology**

As early as infancy, individuals begin to develop a characteristic style of expressing emotions, and the frequency of expression of various discrete emotions tends to remain stable over time (Hyson & Izard, 1985; Izard, Hembree, & Huebner, 1987). In later development, some aspects of emotion expression relate to many forms of psychopathology for example, attenuated or discordant expression in people at risk for Schizophrenia (Simons et al., 1993), prolonged expression of negative emotions (particularly sadness and anger) in depression (Blumberg & Izard, 1986), dampened or developmentally delayed expression in Down Syndrome Disorder (Cicchetti & Sroufe, 1976; Emde, Katz, & Thorpe, 1978), inappropriate or incongruous expression in Autism (Sigman & Capps, 1997), and deceptive expression in psychopathy (Cleckley, 1941; Patrick, 1994). Furthermore, expressions of particular emotions in certain conditions characterize aggressive rejected children (Hubbard, 2001) and delinquent youth (Keltner, Moffitt, & Stouthamer-Loeber, 1995) and may reveal the type of abuse that leads to a Posttraumatic Stress Disorder (Bonanno et al., 2002).

### **Physiological and biochemical aspects of emotion**



## **The Biological Regulators of Emotions**

The immune and endocrine systems aid in processing emotions, two integral brain systems share in the regulating duty (Edelman, 2001).

1. The cerebral cortex governs higher functions and manages communications with the outside world.
2. The brain stem which is located at the base of the brain plus the limbic system formations encompassing it directs people internally, focusing on the emotional, nurturing and survival needs. The brain stem also monitors spontaneous activity, such as heart rate.

### **The Cerebral Cortex**

While investigations are not precise on the roles the hemispheres play in emotion, a few common patterns are obvious (Corballis, 1991). The right hemisphere appears to represent processing the emotional content of gestures, faces, speech intonation and volume associated with how something is communicated, while the left hemisphere processes the actual content of language or what is spoken. The right hemisphere also processes information that point to withdrawal reactions, for instance, fear and revulsion whereas the left hemisphere processes the aspects of emotion that point to advancing reactions like laughter and joy. Tomasi and Dardo (2011) have implied that the average male brain seems to follow a left design of hemisphere specialization; however, the average female brain may disperse more emotional processing across the two hemispheres. If accurate, these organizational variations may serve to clarify regularly seen gender discrepancies.

### **The Limbic System and Brain Stem**

Consist of limbic lobe and related subcortical nuclei.

- The limbic system and brain stem react slower, from seconds to months as it governs fundamental body functions, cycles, and defenses that broadly connect to organs and systems. The reticular formation at the tip of the brain stem integrates the volume, and kind of incoming sensory data into a common level of awareness.

- The limbic system is formed from many small interconnected networks and is the brain's primary manager of emotion that plays a significant role in processing memory. This system may reveal why emotion is a significant element in memory formation as it is strong enough to reverse both rational thinking and innate brain stem reply patterns, meaning people tend to follow their emotions (Rolls, 2013).

### **Amygdala**

**The limbic systems structures that process memory and emotion are**

- the amygdala
- the hippocampus

- the thalamus
- the hypothalamus

**The amygdala is the key limbic system structure implicated in processing the emotional content of memory and behavior.** It is composed of two little almond-shaped structures that link the sensory-motor systems and autonomic nervous system, which governs survival faculties such as breathing and heart rate. The amygdala also communicates with nearly all other brain regions. Its primary responsibility is to refine and translate advanced incoming sensory data in connection with survival and emotional demands, and then assists in launching relevant actions.

### **Physiology of special emotion**

#### **Fear**

- **Site:** The hypothalamus and amygdaloid nuclei
- **Effects of lesion:** After destruction of amygdala the fear reaction and its autonomic and endocrine manifestation are absent. eg monkeys are normally terrified of snakes but after bilateral lobectomy they approach snake pick them n eat them
  - In humans amygdala damage causes deficient fear response to visual and auditory stimulus

#### **The classical and curious case of Phineus Gage.**

Phineas P. Gage (July 9, 1823 – May 21, 1860) was an American railroad construction foreman now remembered for his improbable survival of an accident in which a large iron rod was driven completely through his head, destroying much of his brain's left frontal lobe. The damage to Gage's frontal cortex had resulted in a **complete loss of social inhibitions, which often led to inappropriate behaviour.**

**Anxiety:** It is normal emotion in appropriate situation but excessive anxiety & anxiety in inappropriate situation is disabling.

- **Site:** associated with bilateral increase in blood flow in discreet portion of anterior end of each temporal lobe.
- **Facts:** Anxiety is relieved by benzodiazepine which binds to GABA receptors and increase conductance of these ion channels.

#### **Rage and placidity:**

- Rage – extreme form of anger
- Placidity – Calm and peaceful
- **Site: Neocortex, ventromedial hypothalamic nuclei**

Facts: Human maintains a balance between rage and placidity. Major irritation makes normal individual loose temper but minor stimuli are ignored

#### **Physiology of addiction**

- A kind of dependence which manifests as:



- compulsive non-medical use of a substance
- loss of control over its use despite negative consequences
- Despite many differences, virtually all substances with the potential for addiction affect dopamine levels in the pleasure / reward pathway of the brain.

### **Syndromes of depression and mania:**

Before we start syndromes of depression and mania which are the main syndromes in emotional and will disorder, we must discuss some symptoms related to them.

### **Symptoms of emotional disorders are as follows:**

**Hypothymia:** It is sickly decrease of one's mood. It includes sadness, sorrow, and suppression. The difference between hypothymia and actual feeling of sorrow is that in hypothymia, the person is not only feeling sorrow but cannot experience happiness even in the presence of stimulation. So a patient with hypothymia will not praise his son if he gets a job, if he becomes a grandfather. Depending on the severity of the disease hypothymia can be from a mild feeling of bores, pessimism till profound physical (vital) feelings worrying as spiritual pain, discomfort or shyness in heart. These types of feeling called vital sorrow and are accompanied by the senses of catastrophe, hopelessness and failure.

**Hypothymia** is considered as positive symptom. It is not a specific symptom and can be seen in many mental diseases as well as somatic diseases (brain tumor). It is one of the main symptoms of depression.

**Hyperthymia:** it is sickly increase in one's mood. It is connected with bright positive emotions, i.e. happiness, joy, enthusiasm etc. from situational happiness, hyperthymia differs by its duration. For a week or even a month the person can keep extra optimism, happiness, enthusiasm in case of hyperthymia. These patients are very energetic, show initiative everywhere, and have interest in everything. Even some sad events cannot change their good mood. Hyperthymia is the main characteristic of mania. Most acute case of hyperthymia is expressed as oneiroid. One of the basic variants of hyperthymia is euphoria. It not only expression of happiness, joy but and also of kindness and light-heartedness. The patient doesn't show initiative but talks continuously and the content of his speech is empty. Euphoria is seen in exogenous and somatic diseases (intoxication, hypoxia, brain tumor, liver and renal failure, MI and etc.) and can be accompanied by grandiose delusions (in paraphrenic syndrome in patient with progressive paralysis). The term *moriam* refers to foolish, carelessness odd behaviour. Laugh unproductive excitation in profound dementia.

**Dysphoria:** It is characterized by sudden occurrence of anger, spite, irritation and discontent surroundings. In this condition the patient is able to be aggressive, cruel. He can end up with antisocial action, rude sarcasm, mockery and cyni-

cal insult. paroxysmal character of the symptom says epileptic characteristic of the disorder. In epilepsy it is seen as a separate fit or as aura and obscured and twilight state. Dysphoria is one of the main symptoms of psychoorganic syndrome. It is also seen in explosive psychopathy, alcohol intoxication and drug addiction during their abstinent phase.

**Anxiety:** It is an important emotion of human beings which is connected with the demand in unsafe situations and is expressed as the feeling of undefined threat, internal worry. Anxiety is sthenic emotion and is accompanied by throwing, restlessness, involuntary movement of muscles. As an important signal anxiety can occur in the initial period of any mental disease.

**Ambivalence:** It is characterized by simultaneously working of two completely opposite emotions for the same object or subject or situation (like love and hatred for mother). In psychiatry ambivalence plays an important role which makes the patient suffers, disorganizes his behaviour, and is accompanied by contradictory speeches and actions. It is basically not a specific symptom and can be seen in schizophrenia, introvert psychopathy and in older people.

**Apathy:** It is absence or severely decreased expression of emotion. The patient is indifferent to everything. He also has no interest in anything. Their speech is monotonous, and boring. They keep silent in any conversation. They don't show love to parents. Cannot answer simple question like, which is your favorite food?

Apathy is a negative symptom. As a rule it is seen in the last stage of schizophrenia. Other causes of apathy are brain injury, brain tumor, atrophic paralysis. It is important to differentiate apathy from "anaesthesia psychica dorosa". The later one is characterized by an indifferent feeling for others but himself. It is an egoistic worrying which is most of the time containing delusion of guilt. Patient complains of: "I have become like tree, I don't have a heart, it is just an empty can, I don't feel anxiety for my teenage daughter". Anaesthesia psychica dorosa is a typical symptom of depression.

**Emotional lability:** It is a condition when the patient cannot hold his emotion and fluctuate to another emotion easily e.g. from cry to laugh, from severe anxiety to complete relaxation. It is one of the important characteristics in hysteric neurosis and hysteric psychopathy. Similar condition can also be seen in twilight states. One of the variants of emotional lability is emotional fatigue which is characterized not only by fast changing of emotions but also by disability to control the external presentation of emotions. Emotional lability is a typical symptom for vascular diseases of brain (cerebral atherosclerosis), but it can be present in a person as personality specialty.

**Emotional rigidity:** Patient is very rigid, emotionally strong and doesn't show emotions and holds the same emotion for a long time. Generally he remem-

bers only the sad memories. The speech shows circumstantiality. The patient cannot go to other topic and continue talking about how he suffered. Emotional rigidity is often seen in patients suffering from epilepsy.

### **Depressive syndrome:**

The experience of depression has plagued humans since the earliest documentation of human experience. Ancient Greek descriptions of depression referred to a syndrome of melancholia, which translated from the Greek means black bile. In humoral theory, black bile was considered an etiologic factor in melancholia. This Greek tradition referred to melancholic temperament which is comparable to our understanding of early onset dysthymic conditions or depressive personality. During the late 19th and early 20th centuries, phenomenologists increasingly used the term depression or mental depression to refer to the clinical syndrome of melancholia. Emil Kraepelin distinguished mood which was dejected, gloomy, and hopeless in the depressive phase in manic-depressive insanity from the mood which was withdrawn and irritable in paranoia. In addition, Kraepelin distinguished depression which represented one pole of manic-depressive insanity from melancholia, which involves depression associated with fear, agitation, self-accusation and hypochondriacal symptoms.

### **Clinical significance**

Some difficulty in continuing with ordinary work and social activities, but will probably not cease to function completely in mild depressive episode; considerable difficulty in continuing with social, work or domestic activities in moderate depressive episode; considerable distress or agitation, and unlikely to continue with social, work, or domestic activities, except to a very limited extent in severe depressive episode.

### **Duration of symptoms**

At least 2 weeks required for diagnosis for depressive episodes of all three grades of severity.

### **Severity**

Depressed mood, loss of interest and enjoyment, and reduced energy leading to increased fatigability and diminished activity in typical depressive episodes; other common symptoms are: (1) Reduced concentration and attention (2) Reduced self-esteem and self-confidence (3) ideas of guilt and unworthiness (even in mild type of episode) (4) Bleak and pessimistic views of the future (5) Ideas or acts of self-harm or suicide (6) Disturbed sleep (7) Diminished appetite Typical examples of “somatic” symptoms are: loss of interest or pleasure in activities that are normally enjoyable; lack of emotional reactivity to normally pleasurable surroundings and events; waking in the morning 2 h or more before the usual time; depression worse in the morning; objective evidence of definite psychomotor retardation or

agitation; marked loss of appetite; weight loss; marked loss of libido. For mild depressive episode, two of most typical symptoms of depression and two of the other symptoms are required. If four or more of the somatic symptoms are present, the episode is diagnosed: With somatic symptoms. For moderate depressive episode, two of three of most typical symptoms of depression and at least three of the other symptoms are required. If four or more of the somatic symptoms are present, the episode is diagnosed: With somatic symptoms. For severe depressive episode, all three of the typical symptoms noted for mild and moderate depressive episodes are present and at least four other symptoms of severe intensity are required.

### **Maniacal syndrome**

Mania is the mood of an abnormally elevated arousal energy level, or “a state of” heightened overall activation with enhanced affective expression together with lability of affect. Although it is often thought of as a “mirror image” to depression, the heightened mood can be either euphoric or irritable and, indeed, as the mania progresses, irritability becomes more prominent and can eventuate in violence. Although bipolar disorder is by far the most common cause of mania, it is a key component of other psychiatric conditions (e.g., schizoaffective disorder, bipolar type; cyclothymia) and may happen secondary to neurologic or general medical conditions, or as a result of substance abuse.

The nosology of the various stages of a manic episode has changed over the decades. The word derives from the Greek *μανία* (mania), “madness, frenzy” and the verb *μαίνομαι*, “to be mad, to rage, to be furious”. In current DSM-5 nomenclature, hypomanic episodes are separated from the more severe full manic ones, which, in turn, are characterized as either mild, moderate, or severe (with or without psychotic features). However, the “staging” of a manic episode – hypomania, or stage I; acute mania, or stage II; and delirious mania, or stage III – remains very useful from a descriptive and differential diagnostic point of view, in particular allowing for a more thorough consideration of the more pronounced manic states, wherein the fundamental signs become increasingly obscured by other symptoms, such as delusions.

The cardinal symptoms of mania are the following: heightened mood (either euphoric or irritable); flight of ideas and pressure of speech; and increased energy, decreased need for sleep; and hyperactivity. These cardinal symptoms are often accompanied by the likes of distractibility, disinhibited behaviour, and poor judgment, and, as the mania progresses, become less and less apparent, often obscured by symptoms of psychosis and an overall picture of disorganized and fragmented behaviour.

Mania may be caused by drug intoxication (notably stimulants, such as cocaine and methamphetamine), medication side effects (notably SSRIs), and malig-

nancy (the worsening of a condition), to name but a few. Mania, however, is most commonly associated with bipolar disorder, a serious mental illness in which episodes of mania may alternate unpredictably with episodes of depression or periods of euthymia. Gelder, Mayou, and Geddes (2005) suggest that it is vital that mania be predicted in the early stages because otherwise the patient becomes reluctant to comply with the treatment. Those who never experience depression also experience cyclical changes in mood. These cycles are often affected by changes in sleep cycle (too much or too little), diurnal rhythms, and environmental stressors.

Mania varies in intensity, from mild mania (hypomania) to delirious mania, marked by such symptoms as a dreamlike clouding of consciousness, florid psychotic disorganization, and incoherent speech.

### Topic № 3

**a) Psychiatric propedeutics: examination of patients with mental and behavioral disorders (aphronia, intellectual disabilities, impaired attention). Curation of patients. General description, appearance of the patient, attitude to the doctor, behavior, psychomotor activity, and speech. Thought process (speed, productivity, thought content: obsessive, overvalued, and delusional ideas). Types of delusions: paranoid, paraphenic, induced, and residual. Kandinsky-Clérambault syndrome.**

**b) Intelligence: the official educational level of the patient, the general level of knowledge. Intellectual disability and acquired dementia, degrees of the dementia.**

**a) Psychiatric propedeutics: examination of patients with mental and behavioral disorders (aphronia, intellectual disabilities, impaired attention). Curation of patients. General description, appearance of the patient, attitude to the doctor, behavior, psychomotor activity, and speech. Thought process (speed, productivity, thought content: obsessive, overvalued, and delusional ideas). Types of delusions: paranoid, paraphenic, induced, and residual. Kandinsky-Clérambault syndrome.**

#### **Disorders of Thinking**

Thinking is the basic and specific to the human cognitive process in which dialectically established internal (semantic) connection, describing the structure of objects of reality, their relationship to each other and to the subject of cognitive activity.

Thinking – Goal-directed flow of ideas and associations initiated by a problem and leading toward a reality-oriented conclusion. Thinking is a very complex and complicated psychic function. It is closely associated with speech.

We also can say: Thinking is mental behavior wherein ideas, pictures, cognitive symbolizations, or other hypothetical components of thought are experienced or manipulated. In this sense, thinking is inclusive of imagining, recalling, solving problems, free association, daydreaming, concept formation, and a variety of other procedures.

In other words, thinking is a mental process knowledge associated with the opening of a new subjective knowledge to solving problems with the creative transformation of reality.

**Normal human thinking has three characteristics:**

- Content: what is being thought about – this would include delusions and obsessional thoughts
- Form: in what manner, or shape, is the thought about; abnormalities of the way thoughts are linked together
- Stream or flow: how it is being thought about – the amount and speed of thinking

**Disorders of thinking that could have:**

- Quantitative (or form) and
- Qualitative (content) qualities

Thought disorder: any disturbance of thinking that affects language, communication, or thought content the hallmark feature of schizophrenia manifestations range from simple blocking and mild circumstantiality to profound loosening of associations, incoherence, and delusions

**Quantitative disturbances:**

1. disturbances of speed of thinking
  - a) slowed thoughts:
    - slowing of the flow of associations, slowed and diminished verbal production (bradypsychism)
    - blocking of thoughts – cessation of the flow of associations ( patient stops the verbal production without any recognizable impulse from surroundings)

Occurrence: depression, schizophrenia
  - b) flight of thoughts:
    - excessive rapidity of thinking manifested as extreme rapidity in speech (logorrhoea)

Occurrence: mania

**Quantitative disturbances:**

2. disturbance of structure of thinking
  - perseverative thinking: involuntary persistence of response to some question or topic, verbigeration – a meaningless repetition of specific word or phrase

- circumstantiality: indirect speech that is delayed in reaching the point, characterized by an overinclusion of details
- tangentiality: patient never gets from desired point to desired goal  
Occurrence: fatigue, organic mental disorders
- illogical thinking: thinking containing erroneous conclusions or internal contradiction
- neologism: new word created by the patient often by combining syllables or other words
- incoherent thinking: thought that is not understandable
- word salad: incoherent mixture of words and phrases
- autistic thinking: preoccupation with inner, private world
- poverty of content: thought that gives little information because of vagueness, empty repetitions, or obscure phrases
- symbolic and magical thinking: real objects have other, symbolic meaning, in magical thinking words, situations, action have special power and meaning.

### **Qualitative disturbances: disturbances of content of thoughts**

Three main pathologies of thoughts: delusions, obsessions and overvalued ideas

#### **Delusions:**

The English word “delude” comes from Latin and implies playing or mocking, defrauding or cheating. Since time immemorial, delusion has been taken as the basic characteristic of madness. To be mad was to be deluded. What is delusion is indeed one of the basic questions of psychopathology. It would be a superficial and wrong answer to this question just to call a delusion a false belief which is held with incorrigible certainty. We may not hope to resolve this issue quickly with a definition. Delusion is a basic phenomenon. It is the primary task to get this into view. The subjective dimension within which delusion exists is to experience and think our reality (Jaspers, 1973). Whether we like it or not, this is the unavoidable field of tension in which research on delusions is situated: A tight, objectivity-oriented conceptualization on the one hand and the basic anthropological dimensions of subjectivity and interpersonally (i.e. human interdependence or “universal fraternity”) on the other hand. Even if one is skeptical about these “basic” aspects, Jaspers’ central idea should be kept in mind: Delusion is never a mere object which can be objectively detected and described, because it evolves and exists within subjective and interpersonal dimensions only, however “pathological” these dimensions may be. A person with a delusion will hold firmly to the belief regardless of evidence to the contrary. Delusions can be difficult to distinguish from overvalued ideas, which are unreasonable ideas that a person holds, but the affected person has at least some level of doubt as to its truthfulness. A person with a delusion is abso-

lutely convinced that the delusion is real. Delusions are a symptom of either a medical, neurological, or mental disorder.

A delusion, unlike an overvalued idea, “is not understandable” in terms of the patient’s cultural and educational background although the secondary delusion (or delusion-like idea) is understandable with the addition of some other psychopathological event such as hallucination or abnormal mood.

Delusions may be present in any of the following mental disorders:

1) Psychotic disorders, or disorders in which the affected person has a diminished or distorted sense of reality and cannot distinguish the real from the unreal, including schizophrenia, schizoaffective disorder, delusional disorder, schizophreniform disorder, shared psychotic disorder, brief psychotic disorder, and substance-induced psychotic disorder,

2) Bipolar disorder,

3) Major depressive disorder with psychotic features

4) Delirium,

5) Dementia.

Delusions are false beliefs based on incorrect inference about external reality, not consistent with patient’s intelligence and cultural background that cannot be corrected by reasoning

**Characteristics:**

- a. belief firmly held on inadequate grounds may influence on behavior
- b. not affected by rational arguments/ not corrected by reasoning
- c. not a conventional belief/ bizarre content

**Division of delusions:**

according to onset

- a) primary (delusion mood, perception)
- b) secondary (systematized)
- c) shared (folie a deux)

Stages of delusional formation

Roberts G. (1992) reviewed all concepts and gave the following general model of delusion formation.

Delusions – classification according to the content

**Melancholic delusions:**

- delusion of self-accusation (false interpretation of real past event resulting in feeling of guilt)
- hypochondriac delusion (false belief of having a fatal physical illness)
- nihilistic delusions (false feeling that self, others or the world is non-existent or ending)
- delusions of failure (false belief that one is unable to do anything useful)



- delusion of property (false belief that one lost all property)

#### **Delusions of grandeur:**

- delusion of importance (exaggerated conception of one's importance)
- delusion of power, (exaggerated conception of one's abilities/possibilities)
- delusion of identity (false belief of being the offspring of member of an important family)

**Paranoid delusions:** are based on ideas of reference (false ideas that behavior of others refers to a patient):

- delusion of persecution (false belief that one is being persecuted)
- delusion of infidelity (false belief that one's lover is unfaithful)
- erotomanic delusion (false belief, that someone is deeply in love with them)

Delusion of control (false feeling that one's will, thoughts or feelings are being controlled):

- thought withdrawal (false belief that one's thoughts are being removed from one's mind by other people or forces)
- thought insertion (false belief that thoughts are being implanted in one's mind by other people or force)
- thought broadcasting (false belief that one's thoughts can be heard by others)
- thought control (false belief that one's thoughts are being controlled by other people or forces)

#### **Delusional syndromes**

**Paranoiac syndrome.** His clinical exhausted systematic delusions of interpretation of different content (delirium invention, persecution, jealousy, hypochondriac, etc.). This delusional syndrome characteristic of the slow development of the plot with the gradual expansion of delirium, and in time formed the complicated system of reasoning, until the development in some cases delusional worldview. Note the content of delusions resistance, the complexity of the evidence with delusional interpretation of the smallest details. Hallucinations and phenomena psychic automatism are missing. Behavior of patients with the development of the syndrome are more and more determined by delusions (delusional activity), it is different at thoroughness, lost flexibility of thinking dominates delusional content, all the facts and events that are contrary raving concept, rejected or, in turn, are delusional interpretation. Relationships with others also become distorted, delusional character (friends — enemies). Emotional reactions are concentrated around the delusional system with their strong delineated from all other events. Mood patients often slightly elevated, but periodically become maliciously tense, and then patients can make socially dangerous acts. The slow development of a paranoid syndrome, mainly interpretative nature of delirium with its detailed logical develop-

ment, the use of the sick in their argument of real facts often make it difficult early diagnosis. However, in the very logical constructs a patient can reveal signs of “logic of the curve”.

Paranoiac syndrome almost fully expresses clinical paranoia, often observed with paranoid schizophrenia as a stage of development (paranoid schizophrenia), is less common in other psychiatric disorders (organic brain damage, alcohol delusion of jealousy, etc.). This syndrome may manifest as dysmorphophobia (delusions physical disability), when patients believe in ugliness of the body, its parts, or in violation of its duties.

Paranoid syndrome characterized by secondary delusions. There are several paranoid syndromes such as hallucination-delusion syndrome, depressive-delusional, catatonic-delusional and other.

Kandinsky-Clérambault syndrome is occurring frequent for schizophrenia. Kandinsky-Clérambault syndrome consist of 2 symptoms: pseudohallucination (hallucination like), mental automatism, delusions of influenced by external forces. In a monograph published posthumously in 1890, Kandinsky described a condition which involved being alienated from one’s personal mental processes, combined with delusions of being physically and mentally influenced by external forces. The syndrome he described is now known as Kandinsky-Clérambault syndrome, named along with French psychiatrist Gaëtan Gatian de Clérambault.

Described independently by 2 psychiatrists – Victor Kandinsky and de Clérambault, is less known and is used mainly by French and Russian psychiatrists.

Kandinsky’s classic German-language book on pseudohallucinations was published in 1885. In a monograph written in Russian and published posthumously in 1890, he described a syndrome of mental automatism that, as mentioned above, was largely based on his self-observation. The syndrome involved alienation from or loss of one’s own mental processes (cognitive, sensory and motor), which are attributed to somebody else, combined with delusions of physical or mental influences, such as stealing or insertion of thoughts.

Gaëtan Gatian de Clerambault (1872–1934) was born at la Bourges, not far from Paris. After finishing high school in 1888, he studied at the School for Decorative Art. After that, at his father’s request and in accordance with family tradition, he studied law, and only after graduation did he begin to study medicine. He dedicated his doctorate to pilot health after aircraft accidents (4). Starting in 1898, he worked as an internist. From 1905 until his death in 1934, de Clerambault worked in different fields of medicine.

#### **Mental automatism types:**

1. ideatoric (associative/cognitive) automatism – feelings if alien interference in person stream of thoughts, thoughts are being “put it”, or thoughts being

taking away, sensation that person thoughts are opened to others ощущение, “echo of thoughts”, forced inner speech, verbal pseudohallucinations.

2. sensory automatism. Feelings of uncomfortable sensation, burning sensation, sexual excitement all of the above forced by external forces

3. motor automatism, feelings of forced some of the acts, behaviors which are forced on person by external forces.

Paraphrenic syndrome is combination of fantastic and grandeur ideas with expansive affect, possible mental automatisms, delusions of influence and pseudohallucinations. Paranoid schizophrenia has paraphrenic syndrome as a concluding stage of psychotic course.

The **Cotard delusion** (also Cotard’s syndrome and walking corpse syndrome) is a rare condition, in which an afflicted person holds the delusion that they are dead, either figuratively or literally; yet said delusion of negation is not a symptom essential to the syndrome proper. Statistical analysis of a hundred-patient cohort indicates that the denial of self-existence is a symptom present in 69 percent of the cases of Cotard's syndrome; yet, paradoxically, 55 percent of the patients might present delusion of immortality.

In 1880, the neurologist Jules Cotard described the condition as *Le délire des négations* (“The Delirium of Negation”), a psychiatric syndrome of varied severity. A mild case is characterized by despair and self-loathing, and a severe case is characterized by intense delusions of negation and chronic psychiatric depression. The case of Mademoiselle X describes a woman who denied the existence of parts of her body and of her need to eat, and said that she was condemned to eternal damnation and therefore could not die a natural death. In the course of suffering “The Delirium of Negation”, Mademoiselle X died of starvation.

As a mental illness, Cotard's syndrome also includes the patient's delusion that they do not exist as a person and has lost blood, internal organs, or both. In the tenth edition of the (ICD-10), of the WHO, code F22 identifies the Cotard delusion as a disease of human health.

### **Obsessions as pathology of thinking**

In these definitions, the idea that obsessive thoughts are unrealistic is implicit, and they are tacitly compared with delusions, from which they are distinguished by epistemological criteria (e.g. insight into their origin in one's own mind, etc.)

Obsessions are persistent ideas, thoughts, impulses, or images that are experienced as intrusive and inappropriate and that cause marked anxiety or distress. They are “ego-dystonic”. This refers to the individual's sense that the content of the obsession is alien, not within his or her own control, and not the kind of thought that he or she would expect to have. However, the individual is able to recognize

that the obsessions are the product of his or her own mind and are not imposed from without (as in thought insertion).

The most common obsessions are repeated thoughts about contamination (e.g., becoming contaminated by shaking hands), repeated doubts (e.g. wondering whether one has performed some act such as having hurt someone in a traffic accident...), a need to have things in a particular order (e.g. intense distress when objects are disordered or asymmetrical), aggressive or horrific impulses (e.g. to hurt one's child...), and sexual imagery (e.g. a recurrent pornographic image). Obsessions are not simply excessive worries about real-life problems and are unlikely to be related to a real-life problem.

The individual with obsessions usually attempts to ignore or suppress... or neutralize them with some other thought or action (i.e., a compulsion). For example, an individual plagued by doubts about having turned off the stove attempts to neutralize them by repeatedly checking to ensure that it is off DSM-IV.

Obsessional thoughts are ideas, images, or impulses that enter the patient's mind again and again in a stereotyped form. They are almost invariably distressing and the patient often tries, unsuccessfully, to resist them. They are, however, recognized as his or her own thoughts, even though they are involuntary and often repugnant. (ICD-10 International Statistical Classification of Diseases and Related Health Problems.)

### **Obsessive-Compulsive Disorder (OCD)**

Everyone double checks things sometimes. For example, you might double check to make sure the stove or iron is turned off before leaving the house. But people with obsessive-compulsive disorder (OCD) feel the need to check things repeatedly, or have certain thoughts or perform routines and rituals over and over. The thoughts and rituals associated with OCD cause distress and get in the way of daily life.

The frequent repetitive thoughts are called obsessions. To try to control them, a person will feel an overwhelming urge to repeat certain rituals or behaviors called compulsions. People with OCD can't control these obsessions and compulsions. The rituals end up controlling them most of the time.

**For example**, if people are obsessed with germs or dirt, they may develop a compulsion to wash their hands over and over again. If they develop an obsession with intruders, they may lock and relock their doors many times before going to bed. Being afraid of social embarrassment may prompt people with OCD to comb their hair compulsively in front of a mirror-sometimes they get "caught" in the mirror and can't move away from it. Performing such rituals is not pleasurable. At best, it produces temporary relief from the anxiety created by obsessive thoughts.

Other common rituals are a need to repeatedly check things, touch things (especially in a particular sequence), or count things. Some common obsessions include having frequent thoughts of violence and harming loved ones, persistently thinking about performing sexual acts the person dislikes, or having thoughts that are prohibited by religious beliefs. People with OCD may also be preoccupied with order and symmetry, have difficulty throwing things out (so they accumulate), or hoard unneeded items.

Healthy people also have rituals, such as checking to see if the stove is off several times before leaving the house. The difference is that people with OCD perform their rituals even though doing so interferes with daily life and they find the repetition distressing. Although most adults with OCD recognize that what they are doing is senseless, some adults and most children may not realize that their behavior is out of the ordinary.

### **Signs & Symptoms**

People with OCD generally:

- Have repeated thoughts or images about many different things, such as fear of germs, dirt, or intruders; acts of violence; hurting loved ones; sexual acts; conflicts with religious beliefs; or being overly tidy
- Do the same rituals over and over such as washing hands, locking and unlocking doors, counting, keeping unneeded items, or repeating the same steps again and again
- Can't control the unwanted thoughts and behaviors
- Don't get pleasure when performing the behaviors or rituals, but get brief relief from the anxiety the thoughts cause
- Spend at least 1 hour a day on the thoughts and rituals, which cause distress and get in the way of daily life.

### **Overvalued idea**

The overvalued idea, first described by Wernicke, refers to a solitary, abnormal belief that is neither delusional nor obsessional in nature, but which is pre-occupying to the extent of dominating the sufferer's life. Disorders conforming to his definition are well documented, though their recognition as such has been variable, and they may not be as rare as is often thought. As well as sharing a distinctive phenomenology, the conditions develop in similar settings and carry a uniformly poor prognosis. Their pathogenesis is obscure and difficult to account for in conventional terms.

An unreasonable and sustained belief that is maintained with less than delusional intensity (i.e., the person is able to acknowledge the possibility that the belief may not be true). The belief is not one that is ordinarily accepted by other members of the person's culture or subculture."

Examples of thinking impairments: delusions of grandeur, derailment, paralogic conclusions etc.:

Dr. Pavlov: Hi, Jake. How are you?

Jake: I'm fine.

Dr. Pavlov: Good. My name is, Dr. Pavlov.

Jake: Okay, my name is, Jake and you can call me, Jake.

Dr. Pavlov: Nice to meet you.

Dr. Pavlov: So I'm going to be asking you some questions about how you've been feeling, different experiences you might have had in the past week.

Jake: Okay.

Dr. Pavlov: Okay. So why don't we start out with you telling me a little bit about yourself and your background?

Jake: Okay, I'm an actor, a musician/singer.

Dr. Pavlov: An actor, a musician, and a singer, okay. And how long have you been doing that?

Jake: For a while.

Dr. Pavlov: For a while. And how is it going for you?

Jake: It's great.

Dr. Pavlov: It's great? Tell me a little bit more about that.

Jake: Well it's good; I like music, I like singing, I like performing.

Dr. Pavlov: Okay, where do you perform?

Jake: In movie theaters... not movie theaters, in movies.

Dr. Pavlov: In movies, okay. And are you in movies that people would know about and recognize?

Jake: Yes.

Dr. Pavlov: Okay, can you give me an example?

Jake: Most likely one movie I played. I'm training to become an actor.

Dr. Pavlov: You're what?

Jake: One of the Fantastic Four movies.

Dr. Pavlov: I'm sorry I couldn't hear you.

Jake: It's like I'm training to become an actor.

Dr. Pavlov: You're training to become an actor, I see. Anything else about yourself background that you'd like to share with me?

Jake: I'm Spanish, I'm Puerto Rican and Cuban.

Dr. Pavlov: You're Spanish, Puerto Rican, and Cuban.

Jake: Yeah.

Dr. Pavlov: Okay, where are you from?

Jake: I'm from... I'm from America, but my family is Puerto Rican and Cuban.

Dr. Pavlov: You're American but your family is...

Jake: Yeah, born in New York City.

Dr. Pavlov: Okay. Has anything been bothering you lately?

Jake: No.

Dr. Pavlov: No, okay. Could you tell me a little something about your thoughts on life and its purpose?

Jake: I want to go to New Zealand.

Dr. Pavlov: What's that?

Jake: I want to go to New Zealand.

Dr. Pavlov: To New Zealand, and why is that?

Jake: It's a very beautiful place. It's where the Lord of the Rings movies were at, were made.

Dr. Pavlov: You're right. You like that movie?

Jake: Yeah.

Dr. Pavlov: So you want to go to New Zealand. Anything else about life and its purpose?

Jake: I'm going to New Zealand to live there.

Dr. Pavlov: You want to live there?

Jake: I have families that are in New Zealand too.

Dr. Pavlov: You have family in New Zealand?

Jake: Members who are in New Zealand too. That's where they live.

Dr. Pavlov: Okay. Are you in touch with them?

Jake: (yes). I have a brother... I have some brothers who were born in New Zealand too.

Dr. Pavlov: You have brothers?

Jake: They were born in New Zealand too.

Dr. Pavlov: He was born in New Zealand.

Jake: (yes).

Dr. Pavlov: And what were the circumstances that he was born in New Zealand?

Jake: He was born in New Zealand because my parents were traveling and they had a baby there.

Dr. Pavlov: And where are your parents now?

Jake: My father is... my father is in Manhattan. My aunt... I live with my aunt, and my father lives in Manhattan. My grandmother lives in Manhattan. And what do you call it? What do you call it? And my mother... my mother is... my mother most likely lives with my father, so I live with my aunt, they live in Manhattan.

Dr. Pavlov: So your family is in the New York area too?

Jake: (yes).

Dr. Pavlov: Okay. Jake, do you follow any particular religion or philosophy?

Jake: I'm Christian Pentecost.

Dr. Pavlov: Christian Pentecostal, okay.

Jake: Oh, I'm performing plays.

Dr. Pavlov: I'm sorry?

Jake: I'm performing plays.

Dr. Pavlov: You do.

Jake: Yeah.

Dr. Pavlov: What plays are you performing?

Jake: I played an Ed Tierney at the center.

Dr. Pavlov: How did that go?

Jake: It's good, but I'm trying to get to the movies so.

Dr. Pavlov: Right, okay. So you said that you're Christian Pentecostal. Sometimes people tell me that they think there's a devil, what do you think about that?

Jake: I'm religious, but it's like, I'm not saying, well, I think so, yeah. There's one or two other God's, so...

Dr. Pavlov: So there's a devil also.

Jake: (yes).

Dr. Pavlov: Have you ever had any personal experiences that involved the devil in any way?

Jake: Any personal... oh, no. My brothers in New Zealand are also Puerto Rican, Cuban, and New Zealand too.

Dr. Pavlov: Also Puerto Rican...

Jake: Cuban and New Zealand too. That makes me a Puerto Rican, Cuban, and New Zealand too.

Dr. Pavlov: Okay. Jake, can you read other people's minds?

Jake: No, I can't read other people's minds.

Dr. Pavlov: Can others read your mind?

Jake: No, others can't read my mind.

Dr. Pavlov: Okay. Who controls your thoughts?

Jake: Me, I control my thoughts.

Dr. Pavlov: How have you been spending your time?

Jake: In the past week?

Dr. Pavlov: (yes).

Jake: Great. Most likely, I did play in the one I did. I'm a singer, I danced, and I already played in a movie. I played in a movie and it was in New Zealand. It



was in New Zealand it was a movie I played, and it's showing in the movie theater and I played in the Fantastic Four.

Dr. Pavlov: In New Zealand?

Jake: Yeah, I'm the Human Torch.

Dr. Pavlov: Okay, so this past week, what kinds of things have you been doing with your time?

Jake: Most like relaxing.

Dr. Pavlov: Do you prefer to be alone?

Jake: No.

Dr. Pavlov: Do you like to be with others?

Jake: Yeah, some of my brothers, it's a lot, I have a lot of brothers. Some of them I wish to see them all, but I can't see them all, like all the time that's how many brothers I have.

Dr. Pavlov: How many brothers do you have?

Jake: I have, I have a lot of brothers, I have no sisters. I have around 17 brothers.

Dr. Pavlov: 17 brothers, that's a lot.

Jake: Yeah, it's a lot. Wait a minute, excuse me it was 19 brothers.

Dr. Pavlov: 19 brothers, okay. This past week, Jake, have you joined in activities with other people?

Jake: What's that?

Dr. Pavlov: Have you done things with others – the other patients here?

Jake: I went to Yankee Stadium.

Dr. Pavlov: You went to Yankee Stadium, really? How was that?

Jake: Good.

Dr. Pavlov: Yeah.

Jake: I like going out. I like telling my other lovers... I told my other love, I said, "I'll take you out to eat." Saying, "We could go together, we could go together, and the movies are not all we can see, and we could go together. We'll go out to anything you want, we'll go to this, and we'll go to wonderful places." I was like, "Okay, okay – I love you, I love you." Stuff like that.

Dr. Pavlov: Okay, when you're here at the hospital, do you interact with the other patients or do you keep to yourself?

Jake: I talk to other people.

Dr. Pavlov: You talk to other people, okay. Do you have many friends?

Jake: Yes. Matters of fact, my brothers are my friends too.

Dr. Pavlov: And your brothers also, okay. How about here in the hospital, do you have any friends?

Jake: No.

Dr. Pavlov: No, why not?

Jake: I don't have no friends... how do you find friends in a hospital?

Dr. Pavlov: Okay, so you have friends outside of the hospital.

Jake: (yes).

Dr. Pavlov: Okay, do you have any friends that you consider close friends?

Jake: The only close friends I have is brothers, because I have many parents. And they have... I have many parents, I don't have just one parent. I have many parents.

Dr. Pavlov: Tell me about that. How do you have many parents?

Jake: I have many parents because my father... my father did not fall in love with one woman, he fell in love with many people. If not my father would be a lesbian, would be a lesbian. He's a male. He's had male lovers and female lovers and he had children and had children and he had children. So my parents aren't together, so I have many parents. So I have more than just 19 brothers too.

Dr. Pavlov: Okay. Do you have any close friends?

Jake: Close friends would be my lovers. I have male lovers that I have. I have male lovers because my male lovers, male lovers are my male... I have male lovers are my close friends.

Dr. Pavlov: Okay, do you have many lovers?

Jake: Yes, a lot.

Dr. Pavlov: Okay.

Jake: That would make me a lesbian too.

Dr. Pavlov: Okay. Jake, do you feel that you can trust most people?

Jake: I trust most people.

Dr. Pavlov: Are there any people in particular that you don't trust?

Jake: Enemies.

Dr. Pavlov: Enemies, okay. Who are your enemies?

Jake: People who hate me, and hate my family.

Dr. Pavlov: Who hates you?

Jake: Have you ever run across someone who is very bad? Someone like that.

Dr. Pavlov: And is there anybody that hates your family?

Jake: (yes). People, who don't like me, you know, drug addicts and who do drugs, like that.

Dr. Pavlov: Okay. Other than people who are your enemies, is there anybody else that you don't trust?

Jake: Strangers.

Dr. Pavlov: Strangers, okay. And why don't you trust strangers?

Jake: Because strangers do a lot of terrible things to you.

Dr. Pavlov: Okay. Do you have a good reason not to trust strangers?

Jake: Huh?

Dr. Pavlov: Do you have a good reason not to trust strangers?

Jake: Because strangers go in your house and steal things.

Dr. Pavlov: Okay. Jake, do you get along well with others?

Jake: Yes.

Dr. Pavlov: Do you have a quick temper?

Jake: Yes. Oh, no I'm sorry, I'm sorry, I'm sorry. I meant no on that. I was thinking about something, that I forgot about.

Dr. Pavlov: I'm sorry, do you have a quick temper?

Jake: No I don't have a quick...I do have my mother's temper, sorry.

Dr. Pavlov: Okay, this past week have you had any arguments or fights with anyone?

Jake: No, I didn't.

Dr. Pavlov: Are there may be some people who don't like you?

Jake: No.

Dr. Pavlov: No, okay. Do other people talk about you behind your back?

Jake: No.

Dr. Pavlov: You just nodded as if to say yes though.

Jake: I know I was nodding, I'm sorry, no. Some people talk behind my back, I take that back. Some people talk... talk behind my back.

Dr. Pavlov: Okay, I'm just trying to make sure. What do you... what do you think they're saying about you?

Jake: "I hate you" and stuff like that.

Dr. Pavlov: Why do you think they'd be saying those things?

Dr. Pavlov: Alright. Okay, Jake, if you were to compare yourself to the average person, how would you come out? A little better, a little worse, or about the same?

Jake: Okay.

Dr. Pavlov: Okay? Are you special in some ways?

Jake: (no).

Dr. Pavlov: Would you consider yourself gifted?

Jake: (yes).

Dr. Pavlov: How are you gifted? What are your gifts?

Jake: I'm just talented in my things. Like being a musician... singing.

Dr. Pavlov: In the music.

Jake: Singing.

Dr. Pavlov: And singing, okay. And you said that you are training to be an actor right now, is that correct?

Jake: Yeah, I just played in one movie. It was Fantastic Four.

Dr. Pavlov: You were in Fantastic Four?

Jake: Yeah, it was shown in New Zealand.

Dr. Pavlov: Okay, and how about your music and your singing? Tell me about that.

Jake: It's good, it's good.

Dr. Pavlov: And how are you pursuing that?

Jake: Huh?

Dr. Pavlov: When you say that you are a musician and a singer, do you perform to people?

Jake: I'm performing to people.

Dr. Pavlov: Okay. Do you have... are you just practicing now or do you have a record deal, or...?

Jake: It takes time for performing doing things like in performing arts – especially musician/singing.

Dr. Pavlov: What kind of music do you like?

Jake: I like, one of my favorite singers is Celine Dion, and with my other song I sing is Mariah Carrey, the one that she sung, "Hero" song. That's her song. I think that my favorite song is "My Heart Will Go On."

Dr. Pavlov: You like those two songs?

Jake: (yes).

Dr. Pavlov: Okay. Other than your acting and musical talents, do you have any other talents that most people don't have?

Jake: No, just acting and singing.

Dr. Pavlov: Okay. Do you have any special powers?

Jake: No.

Dr. Pavlov: Do you have ESP – Extra Sensory Perception?

Jake: What does that mean?

Dr. Pavlov: Like reading other people's minds or being able to see what's going to happen in the future?

Jake: No.

Dr. Pavlov: No, okay. Are you very wealthy?

Jake: Yes.

Dr. Pavlov: Yes. Tell me about your wealth. How did you become wealthy?

Jake: Because I work hard.

Dr. Pavlov: Because you work hard. How long have you been wealthy?

Jake: A long time, my aunt too for a long time.

Dr. Pavlov: From your art?

Jake: My aunt.

Dr. Pavlov: Oh, from your aunt, sorry. And how did your aunt become wealthy?

Jake: Because she works hard.

Dr. Pavlov: What kind of work does she do?

Jake: She takes care of people and works at daycare centers.

Dr. Pavlov: Daycare centers?

Jake: Daycare centers.

Dr. Pavlov: Okay, can you be considered to be very bright or intelligent?

Jake: Yeah.

Dr. Pavlov: More so than other people?

Jake: Yeah.

Dr. Pavlov: And why would you say so?

Jake: What'd you say... say what you said again?

Dr. Pavlov: Would you consider yourself to be more intelligent than other people?

Jake: Yes, because other people don't know a lot of stuff.

Dr. Pavlov: And why would you say you're more intelligent?

Jake: Because some people don't know a lot of things. I may not know a lot of things. 19:05

Dr. Pavlov: And what kinds of things do you know?

Jake: I know art, acting, music and singing.

Dr. Pavlov: Okay, would you describe yourself as famous?

Jake: Yes.

Dr. Pavlov: And would some people recognize you from being on TV, the radio, movies, newspaper?

Jake: (yes).

Dr. Pavlov: Okay, so you mentioned earlier that you were in the movie Fantastic Four.

Jake: (yes).

Dr. Pavlov: Is there any other way that people would recognize you?

Jake: If they go to New Zealand.

Dr. Pavlov: And why would they recognize you in New Zealand?

Jake: Because they show, I'm playing in movies there.

Dr. Pavlov: What's that?

Jake: I play in the movies there.

Dr. Pavlov: You're playing in movies?

Jake: I played movies in there.

Dr. Pavlov: Okay. Alright, Jake, I'm going to say a pair of words and I'm going to ask you to tell me how these two things are similar or alike, okay? So, for an example, apple and banana – how are those two things alike?

Jake: They're fruits.

Dr. Pavlov: They're fruits, exactly. How about a pencil and a pen?

Jake: Materials to write with.

Dr. Pavlov: A hat and a shirt.

Jake: Something to wear.

Dr. Pavlov: An arm and a leg.

Jake: They're part of the human body.

Dr. Pavlov: Okay, and how about a painting and a poem?

Jake: Performing arts.

Dr. Pavlov: Okay. Now I'm going to tell you some expressions that people use sometimes, and ask you to tell me what you think they mean. Okay?

Jake: Okay.

Dr. Pavlov: So, for example, if somebody says, "Carrying a chip on your shoulder" – what do you think they mean when they say that?

Jake: Carry a chip on the shoulder... is strong?

Dr. Pavlov: What's that?

Jake: Be strong.

Dr. Pavlov: Be strong, okay. How about, "Two heads are better than one."

Jake: Work together with someone.

Dr. Pavlov: Okay. "All that glitters is not gold."

Jake: Don't do wrong.

Dr. Pavlov: Don't do wrong, okay. How about, "Don't keep all your eggs in one basket?"

Jake: Share with people.

Dr. Pavlov: Okay. And, how about, "The acorn never falls far from the tree?"

Jake: Okay, be strong.

Dr. Pavlov: Be strong. Let me try just one more. "People who live in glass houses shouldn't throw stones at others."

Jake: Don't lie.

Dr. Pavlov: Don't lie, okay.

Dr. Pavlov: Do you think that you have a psychiatric or mental health problem right now?

Jake: Most likely I do not have problem. I just realized that I have fathers, my fathers had reproduction system, and they have children. So my fathers, I don't have no mother, I have fathers. Fathers have reproduction system. It's something

where men... men can have babies and they have reproduction system and they have children. I don't have any mothers.

Dr. Pavlov: No mothers.

Jake: No mothers, just fathers.

Jake: Most likely it is medicine to be better. I also dropped out of high school in a love relationship.

Dr. Pavlov: A love relationship, okay. How serious are the problems that you've had, would you say – that brought you to the hospital.

Jake: Well, I fell in love in high school. I've had a relation... I had sex freshman year and out of school my senior year. And so the teacher that's found that, there was something those parents have to know.

Dr. Pavlov: what are your future plans?

Jake: My future plans – keep acting, musician, singing too...performing too.

Dr. Pavlov: Okay, and any other longer range goals?

Jake: I already appeared in a Broadway play.

Dr. Pavlov: A Broadway play?

Jake: I was on the cover.

Dr. Pavlov: You were already in a couple Broadway plays?

Jake: I was the cover. I was in the cover.

Dr. Pavlov: On the cover of like the Broadway program.

Jake: Yeah, program.

Dr. Pavlov: So where do you see yourself in 10 years from now?

Jake: Singing and performing and acting.

Dr. Pavlov: Okay, any other long range goals that you have for yourself?

Jake: No, to go to New Zealand. Back to New Zealand, back to New Zealand.

Dr. Pavlov: That's right.

Jake: I wasn't born in America I was born in... I was born in New Zealand. I wasn't born in America; I was born in New Zealand. Sorry for saying that, but I was born in New Zealand.

Dr. Pavlov: Okay, but you've been in New York for a while?

Jake: I've been in New York for a while. I was born in New Zealand though.

**b) Intelligence: the official educational level of the patient, the general level of knowledge. Intellectual disability and acquired dementia, degrees of the dementia.**

**Syndromes of intellect disorders:**

On the other hand understanding intellect is very complex to define and to examine. It is suggested that it is the understanding that signifies the total potential

of a person, complex of his ability and the way of its realization for adaptation in life.

**Intellect can be categorized in 3 types.**

Vision – action thinking: a baby till his ability to deliver a speech looks at the surrounding and copies the action of adults, which leads to formation of action out of own interest like take the toys, eating with spoon without the help of adults.

After formation of speech, from the experience, the baby starts selection of things. This basic representation of thinking is called concrete form of thinking.

When the baby starts going school, he gradually meets the world with abstract understanding and symbols, which sometimes is not representable, like mathematical actions, laws of physics etc. Brain-operations of these understandings are called abstract thinking. So the intellect of an adult suggests coexistence and interaction of practical experiences, concrete-situational representation and ability to abstract thinking.

Level of intellect may be characterized quantitatively with the help of I.Q. i.e.  $\text{mental age} \times 100 / \text{chronical age}$ . Mental age of a person develops till the age of 16. After that it's all experiences, that is developed and make a person mature.

Abstract thinking cannot be given grades by simply asking questions, which patients already solved many times in their lives. Even a patient with severe disorder of intellect may answer the months of the year one by one.

Intelligence includes:

- abstract
- practical
- social

Disorders of intellect:

- Intellectual disability
- dementia

Disorder of intellect can be represented by syndrome of intellectual disability, i.e. oligophrenia and decreased intellect, i.e. dementia.

Oligophrenia: it is undeveloped psychological functions due to several reasons which were present till the birth or during the 1st year of life. Oligophrenia is expressed when the formation of most of the nerve functions develops considerably slow. In this case many important functions for adaptation do not form at all. Traditionally oligophrenia is divided according to its severity of study in 3 groups.

a) Idiocy: I.Q level is below 20. This is the most profound study of oligophrenia. Patient is completely helpless in this situation. He cannot form a speech consciously. Emotional reactions are rather primitive (cry, shout). He consciously doesn't know his parents. Motor functions are not developed. Some patients even cannot walk. There may be some stereotypic movements that the patient copies



from the surrounding. Patient cannot take care of himself. He is always kept under strong supervision. Psychological defect in oligophrenia is often combined with multiple defects of internal organs and anomalies. Often any kind of infectious disease or somatic disease may cause death of the patient. An oligiphrenic hardly becomes an adult.

b) Imbecility: I.Q. level is 20 to 49. This is severe to moderate disorder of intellect where the patient has no abstract thinking.

This is due to late activity. Patients show bad articulation, stammering, very poor vocabulary, and most of the times monosyllabic answers (rarely a phrase formation). Patient says the name of the object, knows the function of it, but cannot explain why it has such function or why this function is needed, and cannot use it in any condition. Patient knows alphabets and numbers but cannot spell and cannot do any sum. Most of the imbeciles are emotionally attached to relatives, tender and obedient. Though his coordination is disturbed, but he can be taught how to take care of himself. He can also be taught simple operations of life, but without proper supervision he is distracted fast and doesn't carry out the work.

c) Debility (moronic): I.Q. level is 50 to 69. This is mild disorder inclined at concrete situational thinking along with sudden decreased abstract thinking. Patient is practical, well oriented with the situations. He can be given education in special school where he learns to read, write letters, simple but necessary operations to lead a normal life.

**Dementia:** this is loss of intelligence after a period of its normal development. This is more or less psychological defect with a disorder of intellectual function. The signs of dementia are loss of ability and knowledge, general decrease of productivity of psychological action and change in personality. Dementia can be seen in brain tumor, atrophic diseases of brain, and vascular diseases of brain.

Clinical picture of dementia differs in different types of psychological diseases. According to clinical pictures, dementia can be due to organic disorders, epilepsy and schizophrenia.

Organic dementia occurs due to disturbed structure of brain and massive death of neurons in the brain. Clinical picture shows the severe disorder of memory and decreased ability to abstract thinking. Organic dementia can be of two types.

1) **Lacunar dementia:** this is also called atherosclerotic dementia. Clinical picture shows primary marked disorder of memory, slight deficiency in understanding, mild personality changes (expression of personality traits), and a good insight about the disease, i.e. patient understands his disease and seeks for help and feels sad about his condition. Causes of this dementia are atherosclerosis of arteries of brain, hypertension, diabetic microangiopathy, disorder.

2) **Total dementia:** this is characterized by primary loss of understanding, severe disorder of memory, poor or very formal insight about the disease, and severe changes in personality. Causes of total dementia are atrophic diseases of brain. This may be a diffuse process like degenerative diseases, (e.g. Alzheimer's disease, Pick's disease), meningoencephalitis (e.g. syphilitic meningoencephalitis, progressive paralysis), severe brain injury. Division of organic dementia is not pathoanatomical, but syndromal.

**Epileptic dementia:** this is one of the variations of organic dementia which occurs at the final stage of epilepsy with the manifestation of loss of memory and ability to understanding ' action (cognition). This is also characterized by disorder of thought (circumstantiality, oligophasia). Personality of the person also changes severely. Patient becomes egocentric. Disorder of memory in the patient has a special character. He can remember about anything that is related to him (name of doctor, name and number and doses of each medicine, amount of his pension, and date of pension) but he cannot remember that his wife is ill or the name of the president of the country.

**Schizophrenic dementia** (nowadays called apathico-abulia syndrome): this is not an organic disease. In case of schizophrenia, memory is not lost. There is also no loss of ability of abstract thinking. There becomes a disorder of the structure of abstract thinking and its aim. There develops a passivity and indifference. Disorder of thought (schizophasia) is seen. The patient cannot fulfill any work. This leads to more indifference. Patient may lie on his bed the whole day. He ' may not like to read, watch television, doesn't do any household work. In any question asked to him, his answer will be "I don't know". He stops taking care of himself, doesn't change clothes, stops taking shower and brushing teeth.

#### Topic № 4

a) **Psychopathology willpower: stages of the act of will. Symptoms of the willpower disorder.**

b) **Psychopathology consciousness: clear and impaired consciousness criteria. The notion of paroxysmal and non-paroxysmal consciousness disorders: delirium, oneirism, amentia, twilight state.**

c) **Catatonic syndrome.**

a) **Psychopathology willpower: stages of the act of will. Symptoms of the willpower disorder.**

**Symptoms of will and inclinations are as follow:**

**Hyperbulia:** It is characterized by increase in will and drive (inclination). It can be presented as increased appetite, hyper sexuality, talkativeness, etc. The patient can steal food from other patients in the ward due to increase appetite. Hyper

sexuality can be expressed by talking to opposite or same sex more, giving them more attention, frequently using of bright cosmetics to draw the attention, buying presents, inviting for dates. To be remembered that simultaneously increase of will and inclination doesn't lead to danger for the patient and surroundings. Only the patient disturbs other people by his behaviour (he can call 20 times a day to a girl and asks for date). Hyperbulia is characteristic symptom for mania.

**Hypobulia:** This is just the opposite of hyperbulia. The patient doesn't show any will and inclination including physiological drives. Like, he may refuse to eat for days or eating minimal quantity saying that he doesn't have appetite, he may complain of sleeplessness. Opposite sex or same sex will not attract him. The patient will not like to talk with anybody even with his doctor. He won't understand the necessities of conversation, and wants to be left alone. Suppression of self defense mechanism leads to attempt of suicide. This is characteristic for the feeling shame for his disability and helplessness. Hyperbulia is characteristic symptom for depression.

**Abulia:** This disorder is characterized by abrupt decrease of will. Lying whole day on the bed and doing nothing is the normal conduct to abulia but this patient shows the need of food, sex, and other things but not socially accepted way. So the patient instead of going to grocery when he feels hungry, calls his neighbour and asks to feed him. Sexual drive is fulfilled by continuous masturbation. The patient loses the higher social demands, he doesn't need contacts, and can sit at home for days. He doesn't show interest in the events in family or in the world. In the ward, he doesn't talk with other patients, doesn't know their names, names of doctors and nurses.

**Abulia** is a negative symptom, sometimes with apathy they present apathia-abulia syndrome, characteristic for schizophrenia. In prodromal disease presentation of abulia varies from mild laziness, to disability with severe passivity.

**Ambivalence:** It is characterized by simultaneously working of two completely opposite emotions for the same object or subject or situation (like love and hatred for mother). In psychiatry ambivalence plays an important role which makes the patient suffers, disorganizes his behaviour, and is accompanied by contradictory speeches and actions. It is basically not a specific symptom and can be seen in schizophrenia, introvert psychopathy and in older people.

**b) Psychopathology consciousness: clear and impaired consciousness criteria. The notion of paroxysmal and non-paroxysmal consciousness disorders: delirium, oneirism, amentia, twilight state.**

Consciousness – the highest integrative mental process, the highest form of reflection of objective reality, peculiar to the person. It provides a cognitive reflection of the world and of itself promotes the adaptation of the individual in a social

environment and allows you to modify it to suit your needs. Consciousness – is not an independent process, and the human psyche as a whole – a product of a gradual development of the individual.

The man has the ability to be conscious of the world and aware of your body, thoughts, actions, feelings, interests and position in society.

### **Disorders of consciousness**

Understanding of consciousness greatly differs by its multiple meanings, and is used in different ways in psychology, physiology and philosophy. In most of the cases this term indicates the ability to perceive oneself and outside world in all safe events. Consciousness intends at first possibility of objects, or senses, cognition and understanding the links amongst phenomena (abstract cognition). From the above mentioned definition it is understood that practically any psychiatric disorder (hallucination, delusion, dementia etc.) is accompanied by disturbance of consciousness.

For definition of upset consciousness condition we use some criteria:

- a) Distraction from real outside world. This is expressed by the fact that the patient fragmentarily, unclearly, perceives reality.
- b) Disturbance of orientation of time, place, situation, and rarely of own personality.
- c) Disturbance of framing thought right up to incoherence.
- d) Amnesia disorder of ability to store the ongoing events in memory during the disturbance of consciousness.

One should remember that all the above mentioned criteria must be present to diagnose disturbance of consciousness. It is also very important for the diagnosis of upset consciousness condition to monitor specific dynamics of same criteria. This condition is acute transient disorder.

Jaspers divided disturbances of consciousness in 3 groups.

- 1) Condition of clouding of consciousness or deterioration consciousness;
- 2) Obscured consciousness;
- 3) Condition of changed consciousness.

But actual understanding of these terms differs from author to author.

We may define them as following:

Deterioration of consciousness doesn't contain psychological process. It is an unlimited row between clear consciousness and completely absence of consciousness (coma). It is devoid of any positive symptom.

Obscured consciousness presents own rows of acute psychosis with bright positive symptoms: hallucination, delusion, psychomotor excitement etc. In this condition the patient doesn't perceive the reality in the 1st order, because he is filled with fantasy and fictions. Fantastic events may coincide with reality and it is

transformed in accordance with imagination and fantasy. In obscured consciousness the patient is active and can commit dangerous acts.

Changed consciousness condition is seen in healthy people. It actually demonstrates the connection of consciousness with function of attention. Concentration or attention in any subject or object makes a person distract from the surrounding world. Thus he cannot get information from the outside world but gets information from the object he is concentrating at the moment. The abnormal conditions categorized as disorders of consciousness are those in which the perception of external objects and spatial and temporal orientation are disrupted, thinking is disordered, events are not fixed in the memory, and alienation from the real world sets in (K.Jaspers). Each of these symptoms is observed in various psychic disorders, but in combination they are characteristic of clouded consciousness. As a result, disorders of consciousness are characterized by the disruption of abstract, logical and visual, sensory and cognition.

In clinical practice, stupor is the most frequently encountered disorder of consciousness, manifested in retardation, somnolence, impoverished psychic life, and elevated threshold for external irritants. Cases range from mild (clouding of consciousness) to extremely severe, characterized by sopor and coma. Delirious clouding of consciousness, or delirium, is characterized by illusions, hallucinations, affective disorders, acute delirium, and motor excitation, in combination with symptoms common to all forms of disruption of consciousness.

Characteristic of oneiric (dreamlike) clouding of consciousness are fantastic, sensual, day dream-like experiences, acute affective and motor disorders and disruption of self-consciousness. The dominant symptom in amnesia is gross disorder of the flow of associative processes (incoherent, fragmentary thinking), accompanied by motor excitation, incoherent talkativeness, and continual changes of mood.

Unlike the above mentioned syndromes, the twilight state develops suddenly, is generally brief (minutes or hours), and has a distinct onset and termination. The patient' outward behaviour often seems purposeful and logical, but malicious depressed affect, acute delirium, and vivid hallucinations may bring on outbursts of furious excitation, with senseless aggression.

Disorders of consciousness:

- qualitative
- quantitative

short-term

long-term

Hypnosis – artificially incited change of consciousness

Syncope – short-term unconsciousness

- Quantitative changes of consciousness mean reduced alertness:

somnolence

sopor

coma

Qualitative changes of consciousness mean disturbed perception, thinking, affectivity and memory:

- **Delirium (confusional state)** – characterized by disorientation, distorted perception, enhanced suggestibility, misinterpretations and mood disorders. Delirium (delirium syndrome) is characterized by impaired orientation in place and time at safety orientation in the self, the influx frightening visual and less auditory hallucinations fear. Hallucinations tend to zoopsychic (animals, especially reptiles often devils). The patient's behavior is determined by the content of hallucinatory images. After exiting delirium amnesia is absent. Occurs when organic disorders and intoxications, is considered exogenous syndrome.

- Patient K., aged 68, after the interruption of alcoholic binge began to see the wall crumbling castles, he was surrounded by people with terrible faces and tried to strangle to him. At the same time saw the series flying UFO. He ran away from home, hid in the woods. During hospitalization insisted that is his friend, who died a few years ago, incorrectly called the year and time of year, was confused in dates. On his face was an expression of horror.

- **Oneiric (oneiroid syndrome)** is a disorder of consciousness with complete disorientation, the influx of cosmic or apocalyptic visual hallucinations, exit oneiroid without amnesia. Characteristic of catatonic schizophrenia that sometimes occurs during substance intoxication and epilepsy. Considered mainly endogenous syndrome.

Patient K., 42 years old was delivered to the clinic by rescuers; he was discovered in a clearing in the mountain forest, sitting by alone. For questions not answered, instructions performed passively. The state of lethargy and passivity with indifference continued for another week. Then the Patient K was leaving state reported that was kidnapped from the forest by the aliens and about 30 years, traveled with them “in the light beam” in the past. Saw how they build the pyramids, canals in Mexico and canals on Mars. After discharge Patient K published in the esoteric newspaper article about the types of aliens and gave examples of their language, consisting of stretching, the letter “a”.

- **Amentia (amental syndrome)** is characterized by complete disorientation, incoherence of the speech (thinking), fleeing movements and partial or complete amnesia after the release of amentia. When the delirium in amentia one of the first symptoms is mumbling and fleeing of motion (mussitant delirium). Occurs when organic disorders and intoxications, also refers to exogenous syndromes.

Patient L., 34 years upon admission to the clinic called correctly your passport details, but was disoriented in time and place. He was hanging outside the window, felt fear. For two nights did not sleep. By the end of the day lies within the bed, stereotyped movements tightens on his blanket. It is quiet, muttering, repeating single syllables, sometimes shouting “go, go”, looks around, biting his lip.

**Twilight’s disorder** is characterized by a narrowing of consciousness with the influx of visual hallucinations, it often painted in yellow and red colors (erythropoie) after the release of twilight disorder patient has partial or complete amnesia. Epilepsy is more common in.

Patient D. 30 years has a history of epilepsy. She with her husband was waiting for her flight during the two days was at the airport, which was constantly postponed. Suddenly she disappeared. She was found 10 kilometers from the airport, broke a window in kindergarten and fell asleep on the floor. She could not to call a date though called month and year, and believed that “the husband moved out, and they have already arrived home.”

**Ambulatory automatism** characterized by the shutdown of consciousness with automatic actions and amnesia. If such actions are accompanied by agitation, but continue to a few seconds (Jogging, cotton door), talk about the Fugue, if a long time (several days), talk about the TRANS. Occur in epilepsy.

Patient L., 24 years, two years ago he suffered a traumatic brain injury. Periodically he had get headaches with nausea. One day he rode a bicycle to the store and disappeared. Patient L was discovered by police in the city, at a distance of nearly 40 miles through a week. He could not call his name and accurately to determine the date, didn't know how to come in the city. Neurological examination show horizontal nystagmus. Confused, trying to recall the events of the past week. Relatives established that he passed through the neighboring settlements, which was seen acquaintances, but their questions did not react, “looked ahead”. This man lived a few days in an abandoned house, collecting the leftovers. After the therapy recovered memory only current events, but for the period of the trance remained amnesia. Dual orientation characteristic of delusions, such as delusions of grandeur, when the patient calls himself simultaneously a significant person and his name, or with delusions of staging argues that, although located in this place, still believes it is not true, staged.

Patient Zh., 30 years, political leader of one of the parties. He was delivered with a rally of his party in psychomotor agitation. Patient Zh. is in the correct orientation in place and time, but insists that at the time of the meeting concurrently with the speeches of speakers behind the stage there were executions, since he heard the shots. Understands that is in office, but believes that all men are recruited by the opponents. Although he knows the date of hospitalization, believes that us-

ing drugs others “alienate from the date of the election, replacing the calendars”. Calls it correctly, but believes that at the same time is “devoted to higher ideas.” Special States of consciousness include psychosensory disorders such as derealization, depersonalization on the background narrowing of consciousness.

Exceptional States of consciousness include pathological intoxication pathological affect. Pathological intoxication – narrowed state of consciousness that arises in the use of minimal doses of alcohol, aggression or other unwarranted actions with subsequent amnesia.

Patient N., 19, was taken out of the pool, where he participated in competitions in diving. At the time of the swim was under water trying to strangle his opponent. When extracted from water behaved inadequately, rushed to his comrades, stripped his trunks, inarticulate scream. The condition is amnesia. At clarification of circumstances it turned out that previously, the inner surface of the mask when you swim only dry rubbed, but in this day coach has recommended to wipe it with alcohol. Previously N. never hard liquor was taken and only once tried beer.

**Pathological affect** – Inadequate a strong reaction to the insult, humiliation, loss with narrowing of consciousness, aggression, auto-aggression. Special ethnic changes of consciousness (amok, low) also refer to pathological affect. According to the description of ethnographers Indian custom of self-immolation of widows after the death of a spouse often has been associated with affective narrowing of consciousness.

Patient S., 35 years, was in inpatient treatment for alcohol addiction, was getting ready to be discharged. He was expected his wife and two sons will arrive by car. They got in a car accident and died. After the message about this event he turned around and ran away, hit a passerby and tore at his clothes, causing considerable damage.

Funds are also multiple consciousness, which is characterized by the transition of a person into a different person with other habits, behavior, name, and amnesia of the previous personality.

### **Diagnosis of disorders of consciousness:**

Psychiatristalgebraic model of consciousness is quite simple – It equates consciousness to the orientation in itself, time and space. Orientation in itself includes awareness of mind and body and interpersonal communications, orientation in time is purely calendar character, and orientation in space – formally territorial. Person should tell you who was talking to whom, he should name the current date and the place. If he does not talk about this it means restriction of consciousness. If unable to name correctly – doctor talks about disorientation. To determine the characteristics of attention, it is important to establish the degree of passivity of attention, the presence of a definition of perception (the patient looks or listens,



asks), the degree of attenuation of remembering and memory, impaired thinking, decreased ability to judgements and conclusions. In a speech at minimal disorders of consciousness can detect replays (perseverative), repetitions of the question (echolalia), increasing distances between words, the increase in the number of words such as “Yes”, “here”, “well”, not the end of words.

### **c) Catatonic syndrome.**

The concept of catatonia was first described by Kahlbaum (1874). Catatonic stupor is one of the most dramatic psychiatric conditions, but is becoming increasingly rare in the Western world. However, it has been suggested that catatonia is under-recognized and under-diagnosed (Van der Heijden et al, 2005). Although the introduction of antipsychotics has reduced the incidence of catatonia, it is still not uncommon (Stompe et al, 2002) and its detection rate can be significantly improved by using a standardized rating scale (Van der Heijden et al).

### **Mechanism of catatonia**

The exact cause of catatonia has not been elucidated, but a number of hypotheses have been offered. According to Northoff (2002), a “top-down modulation” of basal ganglia due to deficiency of cortical gamma-aminobutyric acid (GABA), the primary inhibitory neurotransmitter of the brain, may explain the motor symptoms of catatonia. This explanation might account for the dramatic therapeutic effect of benzodiazepines, which cause an increase in GABA activity. Similarly, hyperactivity of glutamate, the primary excitatory neurotransmitter, has also been suggested as an underlying neurochemical dysfunction (Northoff et al, 1997). Osman & Khurasani (1994) have suggested that catatonia is caused by a sudden and massive blockade of dopamine. This may explain why dopamine-blocking antipsychotics are not generally beneficial in catatonia. Indeed, by exacerbating dopamine deficiency, antipsychotics may cause worse of the condition. Clozapine-withdrawal catatonia is postulated to be due to cholinergic and serotonergic rebound hyperactivity (Yeh et al, 2004). In chronic catatonia with prominent speech abnormalities, positron emission tomography (PET) has identified abnormalities in metabolism bilaterally in the thalamus and frontal lobes (Lauer et al, 2001). A very interesting hypothesis proposed by Moskowitz (2004) suggests that catatonia may be understood as an evolutionary fear response, originating in ancestral encounters with carnivores whose predatory instincts were triggered by movement. This response, of remaining still, is now expressed in a range of major psychiatric or medical conditions, where catatonic stupor may represent a common “end-state” response to feelings of imminent doom.

### **Clinical features of catatonia**

Catatonia is a syndrome that encompasses more than two dozen signs, some of which are relatively nonspecific.

**Stupor** is the classic and most striking catatonic sign. It is a combination of immobility and mutism, although the two can also occur independently.

**Posturing:** The patient is able to maintain the same posture for long periods. A classic example is the “crucifix”. An extreme version of posturing is catalepsy.

**Waxy flexibility:** The examiner is able to position the patient in what would be highly uncomfortable postures, which are maintained for a considerable period of time.

**Negativism:** The patient resists the attempts of the examiner to move parts of their body and, according to the original definition; the resistance offered is exactly equal to the strength applied.

**Automatic obedience:** The patient demonstrates exaggerated cooperation, automatically obeying every instruction of the examiner. *Mitmachen* and *Mitgehen* are forms of automatic obedience. In *Mitmachen* the body of the patient can be put into any posture, even if the patient is given instructions to resist. *Mitgehen* is an extreme form of automatic obedience in which the examiner is able to move the patient’s body with the slightest touch, but the body part immediately returns to the original position (unlike in waxy flexibility).

**Ambitendency:** The patient alternates between resistance to and cooperation with the examiner’s instructions; for example, when asked to shake hands, the patient repeatedly extends and withdraws the hand.

**Psychological pillow:** The patient assumes a reclining posture, with their head a few inches above the bed surface, and is able to maintain this position for prolonged periods.

**Forced grasping:** The patient forcibly and repeatedly grasps the examiner’s hand when offered.

**Obstruction:** The patient stops suddenly in the course of a movement and is generally unable to give a reason. This appears to be the motor counterpart of thought block.

**Echopraxia:** The patient imitates the actions of the interviewer. Aversion  
The patient turns away from the examiner when addressed.

**Mannerisms:** These are repetitive, goal-directed movements (e.g. saluting).

**Stereotypies:** These are repetitive, regular movements that are not goal-directed (e.g. rocking).

**Motor perseveration:** The patient persists with a particular movement that has lost its initial relevance.

**Excitement:** The patient displays excessive, purposeless motor activity that is not influenced by external stimuli.

**Speech abnormalities:** *Echolalia*, *logorrhoea* and *verbigeration* are the main speech abnormalities in catatonia. *Echolalia* refers to the repetition of the examiner’s

words. Logorrhoea is characterized by incessant, incoherent and usually monotonous speech. Verbigeration is a form of verbal perseveration in which the patient repeats certain syllables (logoclonia), words (palilalia), phrases or sentences.

### **Differential diagnoses of catatonia**

Although traditionally link to schizophrenia, catatonia is more commonly associated with mood disorders (Pommepuy & Januel, 2002). For example, Abrams & Taylor (1976) recorded that, in a sample of 55 people with catatonia, only four had **schizophrenia** and more than two-thirds had **affective disorders**, especially **mania**. Similarly, Barnes et al (1986) reported only one person with schizophrenia in their sample of 25, but nine with affective disorders. Increasing age may be a significant risk factor for catatonia in **depression** (Starkstein et al, 1996). Catatonia may also occur as a feature of **post-partum psychiatric disorders** (Lai & Huang, 2004). **Temporal lobe epilepsy** is a recognized cause of catatonia (Kirubakaran et al, 1987). Catatonia is a potential risk of abrupt **discontinuation of clozapine**, and is reversible by reinstatement of the drug (Yeh et al, 2004). Immobility seen in advanced dementia might reflect a catatonic state seen in other serious organic disorders, and may respond to lorazepam (Alisky, 2004). There have been case reports suggesting that patients with **thrombotic thrombocytopenic purpura** may be at higher risk of developing catatonia (Yacoub et al, 2004). Catatonia **induced by cocaine** (Gingrich et al, 1998) and **ecstasy** (Masi et al, 2002) have been reported. Prescribed medication such as **ciprofloxacin** (Akhtar & Ahmad, 1993) can also cause catatonia. **Metabolic abnormalities** such as hyponatraemia may cause catatonia (Lee & Schwartz, 1997), and people with rare metabolic disorders such as **Wilson's disease** (Davis & Borde, 1993) and **Tay Sachs** disease (Rosebush et al, 1995) may also present with the condition. Prior brain injury and physical illness at onset of psychosis are more common in patients who subsequently develop catatonia than in those who do not (Wilcox & Nasrallah, 1986). A history of severe infectious disease in childhood, including rheumatic fever, is associated with an increased risk of catatonia in adult life (Wilcox, 1986). **Hysteria** has also been traditionally mentioned as a cause of catatonia. In a significant minority, no cause is identified (Barnes et al, 1986). Benegal et al (1993) reported a high prevalence of idiopathic catatonia, and found it to be more common in females.

### **Catatonia in ICD–10 and DSM–IV**

#### **ICD–10**

The ICD–10 diagnosis of catatonic schizophrenia (category F20.2) requires that the patient prominently exhibits at least one of the following catatonic features, for at least 2 weeks: stupor, excitement, posturing, negativism, rigidity, waxy flexibility and command automatism (automatic obedience). If a patient with severe depression is in a stupor, a diagnosis of “severe depressive episode with psy-

chotic symptoms” (F32.3) is made, even if there are no delusions or hallucinations. Similarly, a patient with manic stupor will be diagnosed as having ‘mania with psychotic symptoms’ (F30.2). Thus, for depression or mania, only stupor, which is the most extreme of catatonic signs, seems to have diagnostic implications, whereas for schizophrenia a broader range of signs are considered relevant. Catatonia due to physical causes is diagnosed as “organic catatonic disorder” (F06.1).

#### **DSM–IV**

In DSM–IV a diagnosis of “schizophrenia, catatonic type” (code 295.20) is made if the clinical picture is dominated by at least two of the following: motor immobility, excessive motor activity, extreme negativism, peculiarities of voluntary movements, and echolalia/echopraxia. If a physical cause is identified the diagnosis is “catatonic disorder due to a medical condition” (code 293.89). As in ICD–10, there is no separate diagnostic category for catatonia due to either depression or mania, but catatonia can be added as a specifier in mood disorders.

**Types of catatonia:** Taylor & Fink (2003) believe that catatonia should be classified as an independent syndrome with the following subtypes: non-malignant, delirious and malignant. The nonmalignant type refers to the classic features first described by Kahlbaum, the delirious type includes delirious mania, and the malignant type includes lethal catatonia, neuroleptic malignant syndrome and serotonin syndrome. Van Den Eede & Sabbe (2004) have proposed an alternative classificatory system. They divide catatonia broadly into non-malignant and malignant types, with each further divided into retarded and excited subtypes. In their system, classic catatonia (Kahlbaum syndrome), delirious mania, neuroleptic malignant syndrome and lethal catatonia would respectively be examples of the non-malignant retarded, non-malignant excited, malignant retarded and malignant excited subtypes. A further classification used by the Wernicke–Kleist–Leonhard school of psychiatry, which has proponents especially in Germany, identifies two main types of catatonia – systematic and periodic. These appear to have significant differences in symptomatology, treatment and prognosis (Pfuhlmann & Stober, 2001). The systematic type is less genetically determined, has a higher prevalence and earlier age at onset in males (Stober et al, 1998), and is associated with mid-gestational infections (Stober, 2001). Periodic catatonia has no differences in either age at onset or prevalence between males and females (Stober et al, 1998). Periodic catatonia, according to Stober et al (2002), is the first subtype of schizophrenia with confirmed genetic linkage. Leonhard (1979) differentiated chronic catatonia, on the basis of the speech abnormalities present, into speech-prompt and speech-sluggish (speechinactive) types. A specific category of autistic catatonia has been suggested for catatonia occurring in people with developmental disorders (Hare & Malone, 2004). Similarities between autism and catatonia include abnormal GABA

function, small cerebellar structures and susceptibility genes on the long arm of chromosome 15 (Dhossche, 2004). Ictal catatonia, in which the seizure manifests itself as catatonia, is postulated to be due to involvement of the limbic system. Ictal catatonia is considered a manifestation of non-convulsive status epilepticus.

### **Rating scales for catatonia**

Using a rating scale helps to identify people who have catatonia that might otherwise not have been diagnosed (Van der Heijden et al, 2005). The Bush–Francis Catatonia Rating Scale (BF CRS) appears to be the most widely used instrument for catatonia. The BF CRS has 23 items, and there is also a shorter, 14-item screening version. The reliability and validity of the BF CRS has been established (Bush et al, 1996). Ungvari et al (2005) reported that using the BF CRS, 32% of 225 patients with chronic schizophrenia met the criteria for catatonia. Their study adds strength to the view that catatonia is still not uncommon and that its incidence is grossly underestimated.

## **3. Mental disorders**

### **Topic № 1**

#### **Mental and behavioral disorders due to psychoactive substances abuse. Clinical manifestations, differential diagnosis, fundamentals of treatment.**

##### **Definition**

The Tenth Revision of the International Classification of Diseases and Health Problems (ICD-10) defines the dependence syndrome as being a cluster of physiological, behavioral, and cognitive phenomena in which the use of a substance or a class of substances takes on a much higher priority for a given individual than other behaviors that once had greater value. A central descriptive characteristic of the dependence syndrome is the desire (often strong, sometimes overpowering) to take the psychoactive drugs (which may or not have been medically prescribed), alcohol, or tobacco. There may be evidence that return to substance use after a period of abstinence leads to a more rapid reappearance of other features of the syndrome than occurs with nondependent individuals.

##### **Types of the dependence syndrome**

- Mental and behavioral disorders due to use of alcohol
- Mental and behavioral disorders due to use of opioids
- Mental and behavioral disorders due to use of cannabinoids
- Mental and behavioral disorders due to use of sedatives or hypnotics
- Mental and behavioral disorders due to use of cocaine
- Mental and behavioral disorders due to use of other stimulants, including caffeine
- Mental and behavioral disorders due to use of hallucinogens
- Mental and behavioral disorders due to use of tobacco

- Mental and behavioral disorders due to use of volatile solvents
- Mental and behavioral disorders due to multiple drug use and use of other psychoactive substances

### **ICD-10 Clinical description**

A cluster of physiological, behavioral, and cognitive phenomena in which the use of a substance or a class of substances takes on a much higher priority for a given individual than other behaviors that once had greater value. A central descriptive characteristic of the dependence syndrome is the desire (often strong, sometimes overpowering) to take psychoactive drugs (which may or may not have been medically prescribed), alcohol, or tobacco. There may be evidence that return to substance use after a period of abstinence leads to a more rapid reappearance of other features of the syndrome than occurs with nondependent individuals

### **ICD-10 Diagnostic guidelines**

A definite diagnosis of dependence should usually be made only if three or more of the following have been present together at some time during the previous year:

1. A strong desire or sense of compulsion to take the substance;
2. Difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use;
3. A physiological withdrawal state when substance use has ceased or have been reduced, as evidenced by: the characteristic withdrawal syndrome for the substance; or use of the same (or closely related) substance with the intention of relieving or avoiding withdrawal symptoms;
4. Evidence of tolerance, such that increased doses of the psychoactive substance are required in order to achieve effects originally produced by lower doses (clear examples of this are found in alcohol- and opiate-dependent individuals who may take daily doses sufficient to incapacitate or kill nontolerant users);
5. Progressive neglect of alternative pleasures or interests because of psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects;
6. Persisting with substance use despite clear evidence of overtly harmful consequences, such as harm to the liver through excessive drinking, depressive mood states consequent to periods of heavy substance use, or drug-related impairment of cognitive functioning; efforts should be made to determine that the user was actually, or could be expected to be, aware of the nature and extent of the harm

Diagnosis of the dependence syndrome may be further specified by the following:

- Currently abstinent but in a protected environment (e.g., in a hospital, in a therapeutic community, in prison, etc.)

ronment (e.g., in a hospital, in a therapeutic community, in prison, etc.)

- Currently on a clinically supervised maintenance or replacement regime (controlled dependence) (e.g., with methadone; nicotine gum or nicotine patch)

- Currently abstinent, but receiving treatment with aversive or blocking drugs (e.g., naltrexone or disulfiram)

- Currently using the substance (active dependence)

Without physical features

With physical features

The course of the dependence may be further specified, if desired, as follows:

- Continuous use
- Episodic use (dipsomania)
- Early remission
- Partial remission
- Full remission

Symptoms withdrawal, including: (general withdrawal symptoms)

A. The general criteria for withdrawal state must be met.

B. Any three of the following signs must be present:

- (1) tremor of the tongue, eyelids, or outstretched hands
- (2) sweating
- (3) nausea, retching, or vomiting
- (4) tachycardia or hypertension
- (5) psychomotor agitation
- (6) headache
- (7) insomnia
- (8) malaise or weakness
- (9) transient visual, tactile, or auditory hallucinations or illusions
- (10) grand mal convulsions

Comment

If delirium is present, the diagnosis should be alcohol withdrawal state with delirium (delirium tremens).

#### **Alcohol withdrawal state**

A. The general criteria for withdrawal state must be met. (Note that an opioid withdrawal state may also be induced by administration of an opioid antagonist after a brief period of opioid use.)

B. Any three of the following signs must be present:

- (1) craving for an opioid drug
- (2) rhinorrhea or sneezing
- (3) lacrimation

- (4) muscle aches or cramps
- (5) abdominal cramps
- (6) nausea or vomiting
- (7) diarrhea
- (8) pupillary dilatation
- (9) piloerection, or recurrent chills
- (10) tachycardia or hypertension
- (11) yawning
- (12) restless sleep

#### Cannabinoid withdrawal state

Note. This is an ill-defined syndrome for which definitive diagnostic criteria cannot be established at the present time. It occurs following cessation of prolonged high-dose use of cannabis. It has been reported variously as lasting from several hours to up to 7 days.

Symptoms and signs include anxiety, irritability, tremor of the outstretched hands, sweating, and muscle aches.

#### **Sedative or hypnotic withdrawal state**

- A. The general criteria for withdrawal state must be met.
- B. Any three of the following signs must be present:
  - (1) tremor of the tongue, eyelids, or outstretched hands
  - (2) nausea or vomiting
  - (3) tachycardia
  - (4) postural hypotension
  - (5) psychomotor agitation
  - (6) headache
  - (7) insomnia
  - (8) malaise or weakness
  - (9) transient visual, tactile, or auditory hallucinations or illusions
  - (10) paranoid ideation
  - (11) grand mal convulsions

#### **Comment**

If delirium is present, the diagnosis should be sedative or hypnotic withdrawal state with delirium.

#### **Cocaine withdrawal state**

- A. The general criteria for withdrawal state must be met.
- B. There is dysphoric mood (e.g., sadness or anhedonia).
- C. Any two of the following signs must be present:
  - (1) lethargy and fatigue
  - (2) psychomotor retardation or agitation



- (3) craving for cocaine
- (4) increased appetite
- (5) insomnia or hypersomnia
- (6) bizarre or unpleasant dreams

Withdrawal state from other stimulants, including caffeine

- A. The general criteria for withdrawal state must be met.
- B. There is dysphoric mood (e.g., sadness or anhedonia).
- C. Any two of the following signs must be present:
  - (1) lethargy and fatigue
  - (2) psychomotor retardation or agitation
  - (3) craving for stimulant drugs
  - (4) increased appetite
  - (5) insomnia or hypersomnia
  - (6) bizarre or unpleasant dreams

**Tobacco withdrawal state**

- A. The general criteria for withdrawal state must be met.
- B. Any two of the following signs must be present:
  - (1) craving for tobacco (or other nicotine-containing products)
  - (2) malaise or weakness
  - (3) anxiety
  - (4) dysphoric mood
  - (5) irritability or restlessness
  - (6) insomnia
  - (7) increased appetite
  - (8) increased cough
  - (9) mouth ulceration
  - (10) difficulty in concentrating

**Withdrawal state with delirium**

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) diagnostic criteria for delirium is as follows:

- Disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and awareness.
- Change in cognition (e.g., memory deficit, disorientation, language disturbance, perceptual disturbance) that is not better accounted for by a preexisting, established, or evolving dementia.
- The disturbance develops over a short period (usually hours to days) and tends to fluctuate during the course of the day.
- There is evidence from the history, physical examination, or laboratory findings that the disturbance is caused by a direct physiologic consequence of a

general medical condition, an intoxicating substance, medication use, or more than one cause.

Delirium tremens is a severe form of alcohol withdrawal that involves sudden and severe mental or nervous system changes.

### Causes

Delirium tremens can occur when person you stops drinking alcohol after a period of heavy drinking, especially if person do not eat enough food. Delirium tremens may also be caused by head injury, infection, or illness in people with a history of heavy alcohol use. It is most common in people who have a history of alcohol withdrawal. It is especially common in those who drink 4-5 pints of wine or 7-8 pints of beer (or 1 pint of «hard» alcohol) every day for several months. Delirium tremens also commonly affects people who have had an alcohol habit or alcoholism for more than 10 years.

### Symptoms of Delirium tremens

Symptoms most often occur within 48 – 96 hours after the last drink. However, they may occur up to 7 – 10 days after the last drink.

Symptoms may get worse quickly, and can include:

- Body tremors
  - Changes in mental function
  - Agitation, irritability
  - Confusion, disorientation
  - Decreased attention span
  - Deep sleep that lasts for a day or longer
  - Delirium
  - Excitement
  - Fear
  - Hallucinations (seeing or feeling things that are not really there)
  - Increased activity
  - Quick mood changes
  - Restlessness, excitement
  - Sensitivity to light, sound, touch
  - Stupor, sleepiness, fatigue
  - Seizures (may occur without other symptoms of DTs)
  - Most common in people with past complications from alcohol withdrawal.
- Usually generalized tonic-clonic seizures

### Withdrawal state with Psychotic disorder

A. Onset of psychotic symptoms must occur during or within 2 weeks of substance use.

B. The psychotic symptoms must persist for more than 48 hours.

C. Duration of the disorder must not exceed 6 months.

The diagnosis of psychotic disorder may be further specified by using the following:

- Schizophrenia-like
- Predominantly delusional
- Predominantly hallucinatory
- Predominantly polymorphic
- Predominantly depressive symptoms
- Predominantly manic symptoms
- Mixed

For research purposes it is recommended that change of the disorder from a nonpsychotic to a clearly psychotic state be further specified as either abrupt (onset within 48 hours) or acute (onset in more than 48 hours but less than 2 weeks).

#### **Withdrawal state with Amnesic syndrome**

A. Memory impairment is manifest in both:

(1) a defect of recent memory (impaired learning of new material) to a degree sufficient to interfere with daily living

(2) a reduced ability to recall past experiences

B. All of the following are absent (or relatively absent):

(1) defect in immediate recall (as tested, for example, by the digit span)

(2) clouding of consciousness and disturbance of attention, as defined in delirium, not induced by alcohol and other psychoactive substances, Criterion A

(3) global intellectual decline (dementia)

C. There is no objective evidence from physical and neurological examination, laboratory tests, or history of a disorder or disease of the brain (especially involving bilaterally the diencephalic and medial temporal structures), other than that related to substance use, that can reasonably be presumed to be responsible for the clinical manifestations described under Criterion A.

#### **Residual and late-onset psychotic disorder**

A. Conditions and disorders meeting the criteria for the individual syndromes listed below should be clearly related to substance use. Where onset of the condition or disorder occurs subsequent to use of psychoactive substances, strong evidence should be provided to demonstrate a link.

#### **Comments**

In view of the considerable variation in this category, the characteristics of such residual states or conditions should be clearly documented in terms of their type, severity, and duration. For research purposes full descriptive details should be specified.

If required, use as follows:

- Flashbacks
- Personality or behavior disorder

B. The general criteria for personality and behavioral disorder due to brain disease, damage and dysfunction must be met.

**Residual affective disorder**

B. The criteria for organic mood (affective) disorder must be met.

Dementia

A.B. The general criteria for dementia must be met.

Other persisting cognitive impairment

B. The criteria for mild cognitive disorder must be met, except for the exclusion of psychoactive substance use in Criterion D.

Late-onset psychotic disorder

A.B. The general criteria for psychotic disorder must be met, except with regard to the onset of the disorder, which is more than 2 weeks but not more than 6 weeks after substance use.

**Other mental and behavioral disorders**

Unspecified mental and behavioral disorder

Treatment Delirium and other Psychotic disorder

1. Sedatives such as diazepam or lorazepam

The patient may need to be put into a sedated state for a week or more until withdrawal and DTs are finished. Also it helps treat seizures, anxiety, and tremors.

2. Antipsychotic medications such as haloperidol may sometimes be needed for persons with severe psychotic symptoms, especially if they have an underlying problem such as schizophrenia. However, these drugs should be avoided if possible because they may contribute to seizures.

**Treatment dependence syndrome: Models and Approaches**

The three historical orientations that still underlie different treatment models are:

A medical model emphasizes biological and genetic causes of addiction that require treatment. A physician uses pharmacotherapy to relieve symptoms or change behavior (e.g., disulfiram, methadone, and medical management of withdrawal). A psychological model is focusing on an individual's maladaptive motivational learning or emotional dysfunction assuming it as the primary cause of substance abuse. This approach includes psychotherapy or behavioral therapy directed by a mental health professional.

A sociocultural model, stressing deficiencies in the social and cultural milieu or socialization process that can be ameliorated by changing the physical and social environment, particularly through involvement in self-help fellowships or spiritual activities and supportive social networks. Treatment authority is often vested

in persons who are in recovery themselves and whose experiential knowledge is valued.

These three models have been woven into a biopsychosocial approach in most contemporary programs. The four major treatment approaches now prevalent in public and private programs are

#### Example of biopsychosocial approach to dependency treatment

The Minnesota model of residential chemical dependency treatment incorporates a biopsychosocial disease model of addiction that focuses on abstinence as the primary treatment goal and uses the AA 12-Step program as a major tool for recovery and relapse prevention. Initially required 28 to 30 days of inpatient treatment followed by extensive community-based aftercare, more recent models have shortened inpatient stays considerably and substituted intensive outpatient treatment followed by less intensive continuing care. The new hybrid has used extensively by public and private sector programs, blends 12-Step concepts with professional medical practices.

Skilled chemical dependency counselors, often people in recovery as well as mental health and social work professionals, use a variety of behavioral and reality-oriented approaches. Psychosocial evaluations and psychological testing are conducted; medical and psychiatric support is provided for identified conditions; and the inpatient program utilizes therapeutic community concepts. Although a disease model of etiology is stressed, the individual patient has ultimate responsibility for making behavioral changes.

Pharmacological interventions may be used, particularly for detoxification; extensive education about chemical dependency is provided through lectures, reading, and writing; and individual and group therapy are stressed, as is the involvement of the family in treatment planning and aftercare

Drug-free outpatient treatment uses a variety of counseling and therapeutic techniques, skills training, and educational supports and little or no pharmacotherapy to address the specific needs of individuals moving from active substance abuse to abstinence. This is the least standardized treatment approach and varies considerably in both intensity, duration of care, and staffing patterns. Most of these programs see patients only once or twice weekly and use some combination of counseling strategies, social work, and 12-Step or self-help meetings. Some programs now offer prescribed medications to ameliorate prolonged withdrawal symptoms; others stress case management and referral of patients to available community resources for medical, mental health, or family treatment; educational, vocational, or financial counseling; and legal or social services. Optimally, a comprehensive continuum of direct and supportive services is offered through a combination of onsite and referral services. High rates of attrition are often a problem for drug-

free outpatient programs; legal, family, or employer pressure may be used to encourage patients to remain in treatment.

Methadone maintenance – or opioid substitution – treatment specifically targets chronic heroin or opioid addicts who have not benefited from other treatment approaches. Such treatment includes replacement of licit or illicit morphine derivatives with longer-acting, medically safe, stabilizing substitutes of known potency and purity that are ingested orally on a regular basis. The methadone or other long-acting opioid, when administered in adequate doses, reduces drug craving, blocks euphoric effects from continued use of heroin or other illegal opioids, and eliminates the rapid mood swings associated with short-acting and usually injected heroin. The approach, which allows patients to function normally, does not focus on abstinence as a goal, but rather on rehabilitation and the development of a productive lifestyle.

A major emphasis in recent years has been on reducing HIV infection transmission rates among patients who remain in treatment and stop injection drug use. Individual and group counseling in addition to pharmacotherapy and urine testing are the mainstay of most programs, but more comprehensive and successful programs also offer psychological and medical services, social work assistance, family therapy, and vocational training. Methadone maintenance treatment, which is more controversial and extensively evaluated than any other treatment approach, has consistently been found to be effective in reducing the use of illicit opioids and criminal activity as well as in improving health, social functioning, and employment.

Therapeutic community residential treatment is best suited to patients with a substance dependence diagnosis who also have serious psychosocial adjustment problems and require resocialization in a highly structured setting. Treatment generally focuses on negative patterns of thinking and behavior that can be changed through reality-oriented individual and group therapy, intensive encounter sessions with peers, and participation in a therapeutic milieu with hierarchical roles, privileges, and responsibilities. Strict and explicit behavioral norms are emphasized and reinforced with specified rewards and punishments directed toward developing self-control and social responsibility. Tutorials, remedial and formal education, and daily work assignments in the communal setting or conventional jobs (for residents in the final stages before graduation) are usually required. Enrollment is relatively long-term and intensive, entailing a minimum of 3 to 9 months of residential living and gradual reentry into the community setting. While patients who stay in therapeutic communities for at least a third to half the planned course of treatment usually have markedly improved functioning in terms of reduced criminal activity and drug consumption and improved rates of employment or schooling (and graduates

do even better), the biggest drawback to therapeutic communities is the large percentage of enrollees (75 percent or more) who never complete treatment)

Counseling and Support groups (such as Alcoholics Anonymous).

## **Topic № 2**

**Schizophrenia and related disorders. Schizophrenia, its forms and course types. Acute psychotic disorders. Delusional disorders. Clinical manifestations, differential diagnosis, and treatment.**

Schizophrenia, schizotypal and delusional disorders

Definition of Schizophrenia

Schizophrenia is a chronic and severe mental disorder that is characterized by a disintegration of the process of thinking, of emotional responsiveness, and of contact with reality. The term schizophrenia itself means “fragmented mind”, referring to the schisms between thought, emotion, and behavior that characterize the disease. It is not the same as “split personality”, which is an altogether different illness now known as dissociative identity disorder. People with schizophrenia do not alternate between “good” and “bad” personalities.

### **SCHIZOPHRENIA.**

**INTRODUCTION:** In this chapter we will discuss the disease that comes in class F2 in ICD classification. Though symptoms of the disease show extreme diversity, but the main manifestation presents delusion and closely related psychopathological phenomena. In all similar symptoms that are noticed in the disease differs from each other by tendency, duration, exit, remission, and quality of social disadaptation of the patient. The fundamental disease in class F2 is schizophrenia. Schizophrenia (F20) is a chronic, psychic, endogenous, progressive and functional (though sometimes organic involvements are found with the development of the disease) disease that occurs as a rule at young age. Positive symptoms in schizophrenia are very diverse. Altogether they are called schisis (internal conflict, disturbances of psychological processes). Negative symptoms express as disturbances in thought process and progressive personality change with loss of interest and enthusiasm and emotional indifference. In case of poor prognosis, at the final stage of the disease, there forms profound apathy- abulia defect (schizophrenic dementia). Kraepelin in 1896 was the first who studied and wrote about the disease under the name of DEMENTIA PRAECOX which means early dementia. In 1911 the term schizophrenia was coined by Bleuler which means confused spirit or soul. The question of spreading of schizophrenia among population is vital in scientific studies and as well as in practical practices. The statistical data and epidemiological surveys show that about 2 – 3% of total world population is suffering from schizophrenia. Men and women affected by the disease in almost similar ratio but the on-

set of the disease in men are early. Similarly the symptoms differ depending on the sex of the patient. In women the symptoms are more acute, clinical manifestations are more frequent and are expressed as different affective pathologies. Early malignant variant of the disease is frequently observed in male teenagers.

### **F20 Schizophrenia**

The schizophrenic disorders are characterized in general by fundamental and characteristic distortions of thinking and perception, and affects that are inappropriate or blunted. Clear consciousness and intellectual capacity are usually maintained although certain cognitive deficits may evolve in the course of time. The most important psychopathological phenomena include thought echo; thought insertion or withdrawal; thought broadcasting; delusional perception and delusions of control; influence or passivity; hallucinatory voices commenting or discussing the patient in the third person; thought disorders and negative symptoms. Most people who develop schizophrenia have their first episode of illness in adolescence or early adulthood. It is very rare before age 11, and it is not usually diagnosed before age 18 (when it is called early onset schizophrenia) or after age 50. As a general rule, males will develop symptoms about three to four years earlier than females, with the peak ages of onset for the disease occurring between 15 and 25 for males and between 25 and 35 for females. In addition, over half of all males with schizophrenia are admitted to a psychiatric hospital before age 25, compared to only one-third of female patients. On the whole, females with the disease are more likely than males to have better social functioning and a better outcome with less negative symptoms and improved quality of life. Notwithstanding all the gender-related differences in how and when schizophrenia develops, it occurs equally across the sexes. Some researchers have speculated on a theory of “season of birth effect,” holding that people with schizophrenia are more likely to have been born in winter and early spring, and less likely to have been born in late spring or summer. Others have suggested just the opposite effect: In recent research conducted across six countries, a striking number of young people with a severe form of the disease were found to have been born in the summer months of June and July. Schizophrenia is distributed unevenly throughout the world – some geographic areas report more cases than others – and because of this, some researchers have hypothesized that the disease may have a viral cause. Other researchers, however, have speculated that schizophrenia is precipitated by social stress, based on the fact that it is more common in large cities than anywhere else.

#### **Positive Symptoms**

The first episode of schizophrenia is usually defined as the first episode of psychosis, or break with reality, which is sometimes called a psychotic break. This episode generally marks the first time the person shows positive symptoms of



schizophrenia, or symptoms produced by the disorder itself. The classic examples of positive symptoms of schizophrenia are delusions and hallucinations. Both types of symptoms can only be experienced subjectively, by the person who has them – they cannot be observed or shared directly. Because of this, we are dependent on reports from people who have experienced them for descriptions of what they are like.

#### Delusions

A delusion is generally an irrational belief – for example, that one is all-powerful or persecuted or under the control of others – and is maintained by the believer in the face of overwhelming contradictory evidence. The ideas that make up delusions often seem wildly farfetched to others yet are taken for granted by the people who hold them.

#### Hallucinations

The other classic positive symptom of schizophrenia is the hallucination, or false perception of one or another of the five senses – sight, hearing, taste, touch, or smell. When hallucinating, a person experiences a sensory phenomenon as real even though it is not actually happening. Common auditory hallucinations include hearing a voice or voices commenting on one's actions, two voices arguing, or voices that speak one's thoughts out loud.

#### Negative Symptoms

Schizophrenia is also associated with negative symptoms, which are called that because they represent personality traits or characteristic behaviors that are taken away by the disease. The hallmark of the negative symptoms is a gradual withdrawal from the world, including from one's family and even from one's own self. Other common negative symptoms include loss of interest in things, poor grooming, and noticeable reductions in speech, emotion, and motivation. The speech reductions are of two sorts: poverty of speech, in which the person speaks little or not at all; and poverty of content of speech, in which the person does talk but conveys little meaning in what he or she says. The emotional reductions manifest themselves as the absence or blunting of the ability to express emotion verbally or physically. And the loss of motivation – or avolition – appears as the lack of will to act, as in the act of maintaining one's personal hygiene. Negative symptoms are nowhere near as dramatic or as memorable as the positive ones, although they may be the ones to appear first. They are rarely described by the people who develop them, although the following passage is an exception to that rule – a vivid description of the negative aspects of schizophrenia by an 18-year-old English boy who had had the disorder for about a year.

The course of schizophrenic disorders can be either continuous, or episodic with progressive or stable deficit, or there can be one or more episodes with complete or incomplete remission. The diagnosis of schizophrenia should not be made

in the presence of extensive depressive or manic symptoms unless it is clear that schizophrenic symptoms antedate the affective disturbance. Nor should schizophrenia be diagnosed in the presence of overt brain disease or during states of drug intoxication or withdrawal. Similar disorders developing in the presence of epilepsy or other brain disease should be classified under F06.2, and those induced by psychoactive substances under F10-F19 with common fourth character .5.

Excl.:

schizophrenia:

- acute (undifferentiated) (F23.2)
- cyclic (F25.2)

schizophrenic reaction (F23.2)

schizotypal disorder (F21)

### **F20.0 Paranoid schizophrenia**

Paranoid schizophrenia is dominated by relatively stable, often paranoid delusions, usually accompanied by hallucinations, particularly of the auditory variety, and perceptual disturbances. Disturbances of affect, volition and speech, and catatonic symptoms, are either absent or relatively inconspicuous.

Paranoid schizophrenia: Patients with this disorder are characteristically preoccupied with delusions and/or hallucinations that suggest they are being persecuted by others. Those with this subtype tend to develop it somewhat later in life than do those with other subtypes, and they are higher functioning. An example that received international attention is that of John Nash, a Nobel laureate, who had grandiose delusions, such as that he was on the cover of Time, disguised as the Pope. Another example is Jane, a college student with a part-time job who became suspicious that her coworkers were taking special notice of her. She thought that they exchanged glances when she entered the office and that during lunchtime they talked about her. She initially confronted them, and when they denied her allegations, she became more suspicious and isolated. She could no longer perform her duties, as she was more preoccupied with the “signals at work.” She believed that her phone was tapped and that she was unsafe. When she sought professional help, she did so from a public phone, whispering so that those who “followed” her would not hear. She disconnected frequently and only after several calls was she persuaded that it was safe to be seen by a doctor.

Excl.: involuntal paranoid state (F22.8) paranoia (F22.0)

### **F20.1 Hebephrenic schizophrenia (Disorganized schizophrenia)**

A form of schizophrenia in which affective changes are prominent, delusions and hallucinations fleeting and fragmentary, behaviour irresponsible and unpredictable, and mannerisms common. The mood is shallow and inappropriate, thought is disorganized, and speech is incoherent. There is a tendency to social iso-

lation. Usually the prognosis is poor because of the rapid development of “negative” symptoms, particularly flattening of affect and loss of volition. Hebephrenia should normally be diagnosed only in adolescents or young adults. Disorganized schizophrenia: People with this type of schizophrenia (formerly called hebephrenic schizophrenia) tend to be bizarre and inappropriate in their behavior. They may choose to wear peculiar clothes, laugh inappropriately, grimace weirdly for no apparent reason, or talk about nonsensical ideas. One example is Josh, a young man in his late twenties who still lived at home with his parents. He had been ill for ten years, had never held a job, and had no friends. On visits to his doctor, he sat in the waiting area next to his mother, glancing around the room with a puzzled look and occasionally grimacing or bursting into laughter. He dressed sloppily, with shoes untied, and he invariably carried a bag full of papers. When he got to the doctor’s office, he seemed remote and deep into his inner world, responding to the doctor’s questions only intermittently. His thoughts were disorganized and difficult to follow, and the papers he brought out of his bag to show the doctor had nothing whatsoever to do with what he was saying. At the end of each visit, he had a hard time organizing his papers, and someone always had to help him put them back in the bag.

### **F20.2 Catatonic schizophrenia**

Catatonic schizophrenia is dominated by prominent psychomotor disturbances that may alternate between extremes such as hyperkinesia and stupor, or automatic obedience and negativism. Constrained attitudes and postures may be maintained for long periods. Episodes of violent excitement may be a striking feature of the condition. The catatonic phenomena may be combined with a dream-like (oneiroid) state with vivid scenic hallucinations. This rare type of schizophrenia involves a disturbance of motor function: Patients may be in a stupor, mute, and physically rigid for hours, often in peculiar postures. Sometimes people with this type of schizophrenia will alternate between periods of stupor and wild agitation. Jeff, for example, was a young man in his late teens whose parents sought help for him because, according to them, “he stopped functioning,” which turned out to mean that he lay immobile in bed with his eyes open, staring at the ceiling. During the examination, he was initially mute, answering none of the doctor’s questions, but later in the interview, he repeated the last word of the examiner’s questions over and over (echolalia). When the examiner moved Jeff’s arms, they stayed in the same position until they were returned to his side. Because Jeff had stopped eating or drinking, he had to be hospitalized and ultimately required intensive care.

Catatonic stupor

Schizophrenic:

- catalepsy
- catatonia

- *flexibilitas cerea*

### F20.3 Undifferentiated schizophrenia

Psychotic conditions meeting the general diagnostic criteria for schizophrenia but not conforming to any of the subtypes in F20.0-F20.2, or exhibiting the features of more than one of them without a clear predominance of a particular set of diagnostic characteristics.

Atypical schizophrenia

Excl.:

acute schizophrenia-like psychotic disorder (F23.2)

chronic undifferentiated schizophrenia (F20.5)

post-schizophrenic depression (F20.4)

### **F20.4 Post-schizophrenic depression**

A depressive episode, which may be prolonged, arises in the aftermath of a schizophrenic illness. Some schizophrenic symptoms, either “positive” or “negative”, must still be present but they no longer dominate the clinical picture. These depressive states are associated with an increased risk of suicide. If the patient no longer has any schizophrenic symptoms, a depressive episode should be diagnosed (F32.-). If schizophrenic symptoms are still florid and prominent, the diagnosis should remain that of the appropriate schizophrenic subtype (F20.0-F20.3).

### **F20.5 Residual schizophrenia**

A chronic stage in the development of a schizophrenic illness in which there has been a clear progression from an early stage to a later stage characterized by long-term, though not necessarily irreversible, “negative” symptoms, e.g. psychomotor slowing; underactivity; blunting of affect; passivity and lack of initiative; poverty of quantity or content of speech; poor nonverbal communication by facial expression, eye contact, voice modulation and posture; poor self-care and social performance.

Chronic undifferentiated schizophrenia

Restzustand (schizophrenic)

Schizophrenic residual state

### **F20.6 Simple schizophrenia**

A disorder in which there is an insidious but progressive development of oddities of conduct, inability to meet the demands of society, and decline in total performance. The characteristic negative features of residual schizophrenia (e.g. blunting of affect and loss of volition) develop without being preceded by any overt psychotic symptoms.

### **F21 Schizotypal disorder**

A disorder characterized by eccentric behaviour and anomalies of thinking and affect which resemble those seen in schizophrenia, though no definite and

characteristic schizophrenic anomalies occur at any stage. The symptoms may include a cold or inappropriate affect; anhedonia; odd or eccentric behaviour; a tendency to social withdrawal; paranoid or bizarre ideas not amounting to true delusions; obsessive ruminations; thought disorder and perceptual disturbances; occasional transient quasi-psychotic episodes with intense illusions, auditory or other hallucinations, and delusion-like ideas, usually occurring without external provocation. There is no definite onset and evolution and course are usually those of a personality disorder.

Incl.:

Latent schizophrenic reaction

Schizophrenia:

- borderline
- latent
- prepsychotic
- prodromal
- pseudoneurotic
- pseudopsychopathic

Schizotypal personality disorder

Excl.:

Asperger's syndrome (F84.5)

schizoid personality disorder (F60.1)

## **F22 Persistent delusional disorders**

Includes a variety of disorders in which long-standing delusions constitute the only, or the most conspicuous, clinical characteristic and which cannot be classified as organic, schizophrenic or affective. Delusional disorders that have lasted for less than a few months should be classified, at least temporarily, under F23.

### **F22.0 Delusional disorder**

A disorder characterized by the development either of a single delusion or of a set of related delusions that are usually persistent and sometimes lifelong. The content of the delusion or delusions is very variable. Clear and persistent auditory hallucinations (voices), schizophrenic symptoms such as delusions of control and marked blunting of affect, and definite evidence of brain disease are all incompatible with this diagnosis. However, the presence of occasional or transitory auditory hallucinations, particularly in elderly patients, does not rule out this diagnosis, provided that they are not typically schizophrenic and form only a small part of the overall clinical picture.

Paranoia

Paranoid:

- psychosis

- state

Paraphrenia (late)

Sensitiver Beziehungswahn

Excl.:

paranoid:

- personality disorder (F60.0)
- psychosis, psychogenic (F23.3)
- reaction (F23.3)
- schizophrenia (F20.0)

F22.8 Other persistent delusional disorders

This Disorders include delusions are accompanied by persistent hallucinatory voices or by schizophrenic symptoms that do not justify a diagnosis of schizophrenia (F20.-).

Delusional dysmorphophobia

Involuntal paranoid state

Paranoia querulans

### **F23 Acute and transient psychotic disorders**

Recognizing the Symptoms and What to Do About Them?

One of the challenges of schizophrenia is its often subtle early development prior to the first psychotic break. Early symptoms of the disease – known as “prodromal” or preliminary symptoms that may begin two to six years before the first psychotic episode – may include

- Reduced concentration and attention
- Decreased motivation and energy
- Mood changes, such as depression and anxiety
- Sleep difficulties
- Social withdrawal
- Suspiciousness
- Irritability
- Neglected physical appearance
- Decline in academic performance and abandonment of previous interests

The problem with these symptoms is their vagueness: They can be easy to confuse, for example, with aspects of “normal” adolescence or with the effects of drug use. Moreover, not every one of these symptoms appears in every individual who develops schizophrenia, nor does every teenager who experiences some or even all of these signs go on to develop the disease. Thus, for many parents, the first major inkling that their child is ill is the appearance of more overt signs of the disease, which may include

- Seeing things or hearing voices that are not seen or heard by others

- Exhibiting odd or eccentric behavior and/or speech
- Having unusual or bizarre thoughts
- Confusing TV or dreams with real life
- Communicating confused thinking that is hard to follow
- Behaving like a much younger child
- Showing severe anxiety and fearfulness

A heterogeneous group of disorders have characterized by the acute onset of psychotic symptoms such as delusions, hallucinations, and perceptual disturbances, and by the severe disruption of ordinary behaviour. Acute onset is defined as a crescendo development of a clearly abnormal clinical picture in about two weeks or less. For these disorders there is no evidence of organic causation. Perplexity and puzzlement are often present but disorientation for time, place and person is not persistent or severe enough to justify a diagnosis of organically caused delirium (F05.-). Complete recovery usually occurs within a few months, often within a few weeks or even days. If the disorder persists, a change in classification will be necessary. The disorder may or may not be associated with acute stress, defined as usually stressful events preceding the onset by one to two weeks.

### **F23.0 Acute polymorphic psychotic disorder without symptoms of schizophrenia**

An acute psychotic disorder in which hallucinations, delusions or perceptual disturbances are obvious but variable, this symptoms are changing from day to day or even from hour to hour. Emotional turmoil with intense transient feelings of happiness or ecstasy, or anxiety and irritability, is also frequently present. The polymorphism and instability are characteristic for the overall clinical picture and the psychotic features do not justify a diagnosis of schizophrenia (F20.-). These disorders often have an abrupt onset, developing rapidly within a few days, and they frequently show a rapid resolution of symptoms with no recurrence. If the symptoms persist the diagnosis should be changed to persistent delusional disorder (F22.-).

Bouffée délirante without symptoms of schizophrenia or unspecified

Cycloid psychosis without symptoms of schizophrenia or unspecified

#### **F23.1 Acute polymorphic psychotic disorder with symptoms of schizophrenia**

An acute psychotic disorder in which the polymorphic and unstable clinical picture is present, as described in F23.0; despite this instability, however, some symptoms typical of schizophrenia are also in evidence for the majority of the time. If the schizophrenic symptoms persist the diagnosis should be changed to schizophrenia (F20.-).

Bouffée délirante with symptoms of schizophrenia

Cycloid psychosis with symptoms of schizophrenia

#### **F23.2 Acute schizophrenia-like psychotic disorder**

An acute psychotic disorder in which the psychotic symptoms are comparatively stable and justify a diagnosis of schizophrenia, but have lasted for less than about one month; the polymorphic unstable features, as described in F23.0, are absent. If the schizophrenic symptoms persist the diagnosis should be changed to schizophrenia (F20.-).

Acute (undifferentiated) schizophrenia

Brief schizophreniform:

- disorder
- psychosis

Oneirophrenia

Schizophrenic reaction

Excl.:

organic delusional [schizophrenia-like] disorder (F06.2)

schizophreniform disorders NOS (F20.8)

F23.3 Other acute predominantly delusional psychotic disorders

Acute psychotic disorders in which comparatively stable delusions or hallucinations are the main clinical features, but do not justify a diagnosis of schizophrenia (F20.-). If the delusions persist the diagnosis should be changed to persistent delusional disorder (F22.-).

Paranoid reaction

Psychogenic paranoid psychosis

#### **F24 Induced delusional disorder**

A delusional disorder shared by two or more people with close emotional links. Only one of the people suffers from a genuine psychotic disorder; the delusions are induced in the other(s) and usually disappear when the people are separated.

Incl.: Folie à deux

Induced:

- paranoid disorder
- psychotic disorder

#### **F25 Schizoaffective disorders**

Episodic disorders in which both affective and schizophrenic symptoms are prominent but which do not justify a diagnosis of either schizophrenia or depressive or manic episodes. Other conditions in which affective symptoms are superimposed on a pre-existing schizophrenic illness, or co-exist or alternate with persistent delusional disorders of other kinds, are classified under F20-F29. Mood-incongruent psychotic symptoms in affective disorders do not justify a diagnosis of schizoaffective disorder.



**F25.0 Schizoaffective disorder, manic type**

A disorder in which both schizophrenic and manic symptoms are prominent so that the episode of illness does not justify a diagnosis of either schizophrenia or a manic episode. This category should be used for both a single episode and a recurrent disorder in which the majority of episodes are schizoaffective, manic type.

Schizoaffective psychosis, manic type

Schizophreniform psychosis, manic type

**F25.1 Schizoaffective disorder, depressive type**

A disorder in which both schizophrenic and depressive symptoms are prominent so that the episode of illness does not justify a diagnosis of either schizophrenia or a depressive episode. This category should be used for both a single episode and a recurrent disorder in which the majority of episodes are schizoaffective, depressive type.

Schizoaffective psychosis, depressive type

Schizophreniform psychosis, depressive type

**F25.2 Schizoaffective disorder, mixed type**

Cyclic schizophrenia

Mixed schizophrenic and affective psychosis

**F25.8 Other schizoaffective disorders****F25.9 Schizoaffective disorder, unspecified**

Schizoaffective psychosis NOS

**F28 Other nonorganic psychotic disorders**

Delusional or hallucinatory disorders that do not justify a diagnosis of schizophrenia (F20.-), persistent delusional disorders (F22.-), acute and transient psychotic disorders (F23.-), psychotic types of manic episode (F30.2), or severe depressive episode (F32.3).

Incl.: Chronic hallucinatory psychosis

**What Causes Schizophrenia?**

In spite of over a century of research, scientists acknowledge that they know relatively little about the cause of schizophrenia. Among the many possible causes that have been explored, three areas in particular stand out as key targets of current research: brain abnormalities, genetics, and environmental factors. Because certain abnormalities have been discovered in the brains of people with schizophrenia, compared to the brains of those without the disease, the most accepted hypothesis among today's researchers is that schizophrenia is a brain disease. For example, brain-imaging technology, including such methods as positron emission tomography (PET) scans, have identified reductions in metabolic activity in the frontal cortex of people who have been diagnosed with schizophrenia. These findings are most notable when the patient has performed a mental task during the scan, which

is taken to suggest that the affected brain cannot react to what is going on around it in the world as efficiently as can a normal brain. In addition, neuropsychological studies of higher-level thought processes such as abstraction and concept formation indicate that people with schizophrenia, who perform poorly in both tasks, probably have reduced activity in the frontal cortex. So far, researchers have not yet been able to formulate definitively just what it is that causes people with schizophrenia to have reduced frontal cortex activity, among other physical findings they have identified and associated with schizophrenia. Progress in finding the cause of brain dysfunction in schizophrenia has been slow, but this is explained by the extreme complexity of the illness. Many theories, however, involve the neurotransmitter systems of the brain.

### **How neurotransmitters work?**

The central nervous system is made up of thousands of cells called neurons, some of which collect information acquired through the senses of taste, touch, sight, smell, and hearing, which they send to other neurons for processing. To relay messages, the nervous system relies on neurotransmitters to carry information, in chemical form, across a tiny gap between neurons called a synapse. When a nerve impulse reaches a synapse, it causes the release of a chemical neurotransmitter, which diffuses across the gap and triggers an electrical impulse in the next neuron. The neurotransmitter does this by reaching a receptor site on the target neuron, a site designed to permit the neurotransmitter to bind to the host neuron. When a significant number of receptors are occupied, an electric impulse – a tiny electrical charge – is created and is sent across the host neuron. This is how the neurons in the brain and the rest of the nervous system communicate with each other, thereby regulating all functions of mind and body. Diseases and injuries can disrupt the process by which neurons send messages to one another in various ways. The neuron where the message originates may produce too much neurotransmitter, or not enough, or the wrong kind; the host neuron may not have enough receptors, or too many; and receptors themselves can be the wrong shape, preventing neurotransmitters from binding to them. Many psychiatric disorders are known to involve inadequate quantities of a neurotransmitter in the brain – depression, for example, is treated by increasing the amount of serotonin in the brain – and many researchers have hoped to find such a link between schizophrenia and neurotransmitters.

### **The dopamine hypothesis**

The neurotransmitter dopamine has long been studied for its role in schizophrenia, largely because some antipsychotic medications, such as chlorpromazine (Thorazine), seem to work by blocking dopamine receptors, thereby preventing dopamine from carrying messages across the relevant neurons. One version of the dopamine hypothesis assumes that the dopamine circuits in the brain are overload-

ed, causing people with schizophrenia to think they hear voices when they don't (hallucinations) and to act on false beliefs (delusions). Another version of the dopamine hypothesis suggests that while excessive dopamine activity causes these positive symptoms of schizophrenia, the negative symptoms are caused by the breakdown of dopamine into other chemicals over time. However, in one study only about two-thirds of schizophrenia patients have been found to have increased numbers of dopamine receptors, which suggests that dopamine overload is not the sole cause of the disease. With the failure of the dopamine hypothesis to explain schizophrenia once and for all, research attention has turned to other neurotransmitters in the brain in the hope of finding additional explanations for the disease. Although so far, no definitive explanation has been forthcoming, researchers have found a number of anomalies among neurotransmitters in the brains of people with schizophrenia:

- Tyrosine hydroxylase, a chemical related to dopamine, has
  - been found in large quantities in the brains of people with
  - schizophrenia, and researchers have speculated that an
  - excess of tyrosine might create an excess of dopamine.
  - Abnormally high levels of norepinephrine have been found
  - in the brains of patients with schizophrenia.
  - Because the antipsychotic drug clozapine (Clozaril) is able
  - to treat the symptoms of schizophrenia by balancing the
- activity of both dopamine and serotonin, some researchers suspect that an excess of serotonin may be present in the brains of people with schizophrenia – unlike those with depression, who have inadequate amounts of serotonin.

### **Genetics**

The question genetic researchers start with is: Does schizophrenia run in families? The question is answered by finding whether a close relative of a person with the disorder is at increased risk for developing it, compared with a similar individual chosen at random from the population at large. Since 1980, 11 major family studies have been reported in which the risk of schizophrenia was higher in first-degree (immediate family) relatives of schizophrenia patients than matched controls from the general population. On average, the studies determined that parents, siblings, and children of people with schizophrenia were twelve times more likely to develop the disease than the general population – 5.9% risk versus 0.5%. The goal of genetic studies of schizophrenia is to identify a genetic abnormality responsible for the disease. Once found, such an abnormal function would presumably shed sufficient light on what goes wrong in schizophrenia so that successful treatments could be developed and the abnormal function corrected at its source. So far, although a great deal of effort has gone into such studies, the results have

been disappointing, most likely because there is no clear biological marker for schizophrenia. Because certain abnormalities of brain structure are present at the time of the first episode of disease, many researchers believe that schizophrenia is a neurological disorder beginning very early in life that for some reason does not lead to symptoms until late in childhood or early adulthood. Ideally, the next step would be to examine the developing nervous systems of people before they develop schizophrenia, but it is obviously very difficult to know in advance who should be studied. The good news is that research goes on all the time, and some results are indeed promising. Some progress has been made in identifying genes associated with schizophrenia, which will in turn lead to opportunities to discover new targets for prevention and treatment of the disease. At the same time, researchers are investigating the role of genetic abnormalities in the brains of people with the disease, while others seek to understand how such genes influence perception, attention, and memory in schizophrenia.

### **Environment**

If only because we know that not everyone at genetic risk for schizophrenia will develop the disease, it is assumed that environmental factors also play a role in its occurrence. For example, many observers assume that adverse environmental factors, such as maternal illness or trauma, that happen during fetal development will play a role in causing some cases of schizophrenia, while others have noted that poor socioeconomic conditions can affect the course of the disease in other cases. Untangling the role of such factors in the development of schizophrenia will always be a challenge, if only because it is difficult to decide which is cause and which is effect. If a person with schizophrenia who is employed performs poorly because of hearing voices while on the job and gets fired, the result is almost certain to be an increase in symptoms of schizophrenia. Symptoms of the disease may have caused the person to get fired, but the effect of being fired can exacerbate those same symptoms. Suffice it to say that schizophrenia may be a biological disease, but the people who have the disease must live in the real world, which will have its own impact on their behavior. An example of this cause and effect relationship can be seen in the case of the 18-year-old boy who insisted he had to walk in the desert for 40 days and 40 nights: His father reports that even after the boy had been medicated, his “need” to walk in the desert tended to reemerge in times of great personal stress, as when someone close to him died. The presence of a number of specific environmental factors has been studied in people with schizophrenia. The role of stress in precipitating episodes of schizophrenia has been explored with mixed results, in part because of the difficulty in determining cause and effect – Is the person’s life stressful because he or she has schizophrenia, or does the person’s illness arise because of stress? Other possible environmental fac-

tors that have been studied, albeit also with mixed results, include the previously mentioned “season of birth effect” and the role of city living in schizophrenia. Regarding the latter, the rate at which people develop schizophrenia is known to be consistently higher in cities, and cases are concentrated in the poorest areas of the city. But even though people have been studying this phenomenon since 1939, no one has yet been able to figure out which precise aspects of city life are responsible, in part because so many confounding factors are involved – cities are complex social settings, and the people who live in them must cope with multiple stressors all the time, any of which could challenge the coping mechanisms of those who are vulnerable to stress. Among the hypotheses that have been proposed to explain the situation is the possibility that social isolation within cities predisposes vulnerable individuals to develop the disease.

### Topic № 3

**a) Affective (mood) disorders. Bipolar affective disorder, course types, and treatment. Recurrent depression.**

**b) Eating disorders Anorexia nervosa, bulimia. Differential diagnosis and treatment.**

**c) Sleep disorders**

**a) Affective (mood) disorders. Bipolar affective disorder, course types, and treatment. Recurrent depression.**

**Depressive syndrome:** The experience of depression has plagued humans since the earliest documentation of human experience. Ancient Greek descriptions of depression referred to a syndrome of melancholia, which translated from the Greek means black bile. In humoral theory, black bile was considered an etiologic factor in melancholia. This Greek tradition referred to melancholic temperament which is comparable to our understanding of early onset dysthymic conditions or depressive personality. During the late 19th and early 20th centuries, phenomenologists increasingly used the term depression or mental depression to refer to the clinical syndrome of melancholia. Emil Kraepelin distinguished mood which was dejected, gloomy, and hopeless in the depressive phase in manic-depressive insanity from the mood which was withdrawn and irritable in paranoia. In addition, Kraepelin distinguished depression which represented one pole of manic-depressive insanity from melancholia, which involves depression associated with fear, agitation, self-accusation and hypochondriacal symptoms.

**Clinical significance:** Some difficulty in continuing with ordinary work and social activities, but will probably not cease to function completely in mild depressive episode; considerable difficulty in continuing with social, work or domestic

activities in moderate depressive episode; considerable distress or agitation, and unlikely to continue with social, work, or domestic activities, except to a very limited extent in severe depressive episode.

**Duration of symptoms:** Duration of at least 2 weeks is usually required for diagnosis for depressive episodes of all three grades of severity.

### **Severity**

Depressed mood, loss of interest and enjoyment, and reduced energy leading to increased fatigability and diminished activity in typical depressive episodes; other common symptoms are: (1) Reduced concentration and attention (2) Reduced self-esteem and self-confidence (3) ideas of guilt and unworthiness (even in mild type of episode) (4) Bleak and pessimistic views of the future (5) Ideas or acts of self-harm or suicide (6) Disturbed sleep (7) Diminished appetite Typical examples of “somatic” symptoms are: loss of interest or pleasure in activities that are normally enjoyable; lack of emotional reactivity to normally pleasurable surroundings and events; waking in the morning 2 h or more before the usual time; depression worse in the morning; objective evidence of definite psychomotor retardation or agitation; marked loss of appetite; weight loss; marked loss of libido. For mild depressive episode, two of most typical symptoms of depression and two of the other symptoms are required. If four or more of the somatic symptoms are present, the episode is diagnosed: With somatic symptoms. For moderate depressive episode, two of three of most typical symptoms of depression and at least three of the other symptoms are required. If four or more of the somatic symptoms are present, the episode is diagnosed: With somatic symptoms. For severe depressive episode, all three of the typical symptoms noted for mild and moderate depressive episodes are present and at least four other symptoms of severe intensity are required.

### **Depressive episode**

In typical mild, moderate, or severe depressive episodes, the patient suffers from lowering of mood, reduction of energy, and decrease in activity. Capacity for enjoyment, interest, and concentration is reduced, and marked tiredness after even minimum effort is common. Sleep is usually disturbed and appetite diminished. Self-esteem and self-confidence are almost always reduced and, even in the mild form, some ideas of guilt or worthlessness are often present. The lowered mood varies little from day to day, is unresponsive to circumstances and may be accompanied by so-called “somatic” symptoms, such as loss of interest and pleasurable feelings, waking in the morning several hours before the usual time, depression worst in the morning, marked psychomotor retardation, agitation, loss of appetite, weight loss, and loss of libido. Depending upon the number and severity of the symptoms, a depressive episode may be specified as mild, moderate or severe.

Includes: single episodes of:

- depressive reaction
- psychogenic depression
- reactive depression

Excludes: adjustment disorder (F43.2), recurrent depressive disorder (F33.-), when associated with conduct disorders in F91. – (F92.0), F31.3 Bipolar affective disorder, current episode mild or moderate depression

The patient is currently depressed, as in a depressive episode of either mild or moderate severity (F32.0 or F32.1), and has had at least one authenticated hypomanic, manic, or mixed affective episode in the past.

**F32.0 Mild depressive episode:** Two or three of the above symptoms are usually present. The patient is usually distressed by these but will probably be able to continue with most activities.

**F32.1 Moderate depressive episode:** Four or more of the above symptoms are usually present and the patient is likely to have great difficulty in continuing with ordinary activities.

**F32.2 Severe depressive episode without psychotic symptoms:** An episode of depression in which several of the above symptoms are marked and distressing, typically loss of self-esteem and ideas of worthlessness or guilt. Suicidal thoughts and acts are common and a number of “somatic” symptoms are usually present.

- Agitated depression
- Major depression
- Vital depression (single episode without psychotic symptoms)

**F32.3 Severe depressive episode with psychotic symptoms:** An episode of depression as described in F32.2, but with the presence of hallucinations, delusions, psychomotor retardation, or stupor so severe that ordinary social activities are impossible; there may be danger to life from suicide, dehydration, or starvation. The hallucinations and delusions may or may not be mood-congruent. Single episodes of:

- major depression with psychotic symptoms
- psychogenic depressive psychosis
- psychotic depression
- reactive depressive psychosis

**F32.8 Other depressive episodes – Atypical depression.**

**F33 Recurrent depressive disorder:** A disorder characterized by repeated episodes of depression as described for depressive episode (F32.-), without any history of independent episodes of mood elevation and increased energy (mania). There may, however, be brief episodes of mild mood elevation and overactivity (hypomania) immediately after a depressive episode, sometimes precipitated by

antidepressant treatment. The more severe forms of recurrent depressive disorder (F33.2 and F33.3) have much in common with earlier concepts such as manic-depressive depression, melancholia, vital depression and endogenous depression. The first episode may occur at any age from childhood to old age, the onset may be either acute or insidious, and the duration varies from a few weeks to many months. The risk that a patient with recurrent depressive disorder will have an episode of mania never disappears completely, however many depressive episodes have been experienced. If such an episode does occur, the diagnosis should be changed to bipolar affective disorder (F31.-). Includes: recurrent episodes of:

- depressive reaction
- psychogenic depression
- reactive depression
- seasonal depressive disorder

**Excludes: recurrent brief depressive episodes (F38.1)**

**F33.0 Recurrent depressive disorder, current episode mild:** A disorder characterized by repeated episodes of depression, the current episode being mild, as in F32.0, and without any history of mania.

**F33.1 Recurrent depressive disorder, current episode moderate:** A disorder characterized by repeated episodes of depression, the current episode being of moderate severity, as in F32.1, and without any history of mania.

**F33.2 Recurrent depressive disorder, current episode severe without psychotic symptoms:** A disorder characterized by repeated episodes of depression, the current episode being severe without psychotic symptoms, as in F32.2, and without any history of mania.

Endogenous depression without psychotic symptoms:

- Major depression, recurrent without psychotic symptoms
- Manic-depressive psychosis, depressed type without psychotic symptoms
- Vital depression, recurrent without psychotic symptoms

**F33.3 Recurrent depressive disorder, current episode severe with psychotic symptoms:** A disorder characterized by repeated episodes of depression, the current episode being severe with psychotic symptoms, as in F32.3, and with no previous episodes of mania.

Endogenous depression with psychotic symptoms:

- Manic-depressive psychosis, depressed type with psychotic symptoms

Recurrent severe episodes of:

- major depression with psychotic symptoms
- psychogenic depressive psychosis
- psychotic depression
- reactive depressive psychosis



**F33.4 Recurrent depressive disorder, currently in remission:** The patient has had two or more depressive episodes as described in F33.0 – F33.3, in the past, but has been free from depressive symptoms for several months.

**F34 Persistent mood [affective] disorders:** Persistent and usually fluctuating disorders of mood in which the majority of the individual episodes are not sufficiently severe to warrant being described as hypomanic or mild depressive episodes. Because they last for many years, and sometimes for the greater part of the patient's adult life, they involve considerable distress and disability. In some instances, recurrent or single manic or depressive episodes may become superimposed on a persistent affective disorder.

**F34.0 Cyclothymia:** A persistent instability of mood involving numerous periods of depression and mild elation, none of which is sufficiently severe or prolonged to justify a diagnosis of bipolar affective disorder (F31.-) or recurrent depressive disorder (F33.-). This disorder is frequently found in the relatives of patients with bipolar affective disorder. Some patients with cyclothymia eventually develop bipolar affective disorder. Affective personality disorder:

- Cycloid personality
- Cyclothymic personality

**F34.1 Dysthymia:** A chronic depression of mood, lasting at least several years, which is not sufficiently severe, or in which individual episodes are not sufficiently prolonged, to justify a diagnosis of severe, moderate, or mild recurrent depressive disorder (F33.-).

Depressive:

- Neurosis personality disorder
- Neurotic depression
- Persistent anxiety depression

Excludes: anxiety depression (mild or not persistent) (F41.2)

### **Maniacal syndrome**

Maniacal syndrome: It is just opposite of depressive syndrome.

The triad of maniacal syndrome includes

- a) elation of mood (happiness, joy, delight etc),
- b) pressure of thought and talk, and
- c) psychomotor excitation.

Hyperthymia in this condition is expressed as continuous optimism without paying attention how difficult the task may be. The patient smiles continuously, never complains of anything. Pressure of talk is expressed as fast speech (so fast that sometimes it is slurred, sometimes no voice comes out of the mouth but the lip movements are seen, due to excessive talking saliva can be seen at angles of the lips and dry mouth is noticed.), often distraction ' from the topic and superficial as-

sociation. Activity due to excessive distraction becomes playful, nonproductive. Patient cannot sit for a long time, inclines to go out of the house, request for discharge from hospital. Patient thinks that he is able to do anything. He even think that he is genius and seductive, and continues to talk about his talents. Due to pressure of thought, he has many ideas, so he starts writing poem or novel and demonstrates his creations to surrounding people. Severe stage of mania includes grandiose delusion.

In mania all types of inclinations are increased. Abruptly increases appetite, sometimes patient tends to become alcoholic. He cannot be alone and continuously seeks conversation. During the consultation with doctor distance between patient and doctor is not maintained. The patient pays more attention on his dress which is gorgeous, he put up make up. Attraction at the opposite sex (or same sex) is increased too, which ends up in proposals, sudden marriages, presenting gifts, sudden sexual acts (may be even without protection). The patient is ready to help anybody but his family where he cannot find time. He wastes money, does unnecessary shopping. Due to over activity he cannot finish any of his works, and distracts himself to other work as every time he gets new ideas. In order to fulfill his drives, the patient can be irritative, angry (wrathful mania) and may does antisocial acts (rape, robbery, fighting, taking drugs etc).

For maniacal syndrome, is characteristic to have sudden decrease in night sleep. The patient feels that sleeping is wasting of time, so he works at night and wakes up very early in the morning.

Unlike depression during the early hours in the morning his mood is excellent, he has many ideas and starts his activity. Patient never complains of being tired. Mild sub psychotic level of ' the syndrome is called hypomania.

In hypomania, delusion is not seen.

The patient in maniacal syndrome looks healthy, young. Due to excessive psychomotor activity he loses weight in spite of showing a huge appetite. In hypomania, overweight is seen.

Maniacal syndrome is often seen in MDP and schizophrenia. They are also noticed in intoxication (phenamine, cocaine, corticosteroids, cyclosporine, sulfonamides, hallucinogens etc). Mania is the sign of acute psychosis. It is a collection of positive symptoms and can be treated with proper medication.

Like depression, maniacal syndrome has its atypical variants.

In maniacal-delusional syndrome, besides happiness, nonsystematic delusional ideas of persecution, quarrel, and megalomaniac delusion of grandiosity (acute paraphrenia) are seen.

Mania is the mood of an abnormally elevated arousal energy level, or "a state of heightened overall activation with enhanced affective expression together

with lability of affect.” Although it is often thought of as a “mirror image” to depression, the heightened mood can be either euphoric or irritable and, indeed, as the mania progresses, irritability becomes more prominent and can eventuate in violence. Although bipolar disorder is by far the most common cause of mania, it is a key component of other psychiatric conditions (e.g., schizoaffective disorder, bipolar type; cyclothymia) and may occur secondary to neurologic or general medical conditions, or as a result of substance abuse.

The nosology of the various stages of a manic episode has changed over the decades. The word derives from the Greek *μανία* (mania), “madness, frenzy” and the verb *μαίνομαι* (mainomai), “to be mad, to rage, to be furious”. In current DSM-5 nomenclature, hypomanic episodes are separated from the more severe full manic ones, which, in turn, are characterized as either mild, moderate, or severe (with or without psychotic features). However, the “staging” of a manic episode – hypomania, or stage I; acute mania, or stage II; and delirious mania, or stage III – remains very useful from a descriptive and differential diagnostic point of view, in particular allowing for a more thorough consideration of the more pronounced manic states, wherein the fundamental signs become increasingly obscured by other symptoms, such as delusions.

The cardinal symptoms of mania are the following: heightened mood (either euphoric or irritable); flight of ideas and pressure of speech; and increased energy, decreased need for sleep; and hyperactivity. These cardinal symptoms are often accompanied by the likes of distractibility, disinhibited behaviour, and poor judgement, and, as the mania progresses, become less and less apparent, often obscured by symptoms of psychosis and an overall picture of disorganized and fragmented behaviour.

Mania may be caused by drug intoxication (notably stimulants, such as cocaine and methamphetamine), medication side effects (notably SSRIs), and malignancy (the worsening of a condition), to name but a few. Mania, however, is most commonly associated with bipolar disorder, a serious mental illness in which episodes of mania may alternate unpredictably with episodes of depression or periods of euthymia. Gelder, Mayou, and Geddes (2005) suggest that it is vital that mania be predicted in the early stages because otherwise the patient becomes reluctant to comply with the treatment. Those who never experience depression also experience cyclical changes in mood. These cycles are often affected by changes in sleep cycle (too much or too little), diurnal rhythms, and environmental stressors.

Mania varies in intensity, from mild mania (hypomania) to delirious mania, marked by such symptoms as a dreamlike clouding of consciousness, florid psychotic disorganization, and incoherent speech.

**F30 Manic episode:** All the subdivisions of this category should be used only for a single episode. Hypomanic or manic episodes in individuals who have had

one or more previous affective episodes (depressive, hypomanic, manic, or mixed) should be coded as bipolar affective disorder (F31.-). **Includes:** bipolar disorder, single manic episode

**F30.0 Hypomania:** A disorder characterized by a persistent mild elevation of mood, increased energy and activity, and usually marked feelings of well-being and both physical and mental efficiency. Increased sociability, talkativeness, over-familiarity, increased sexual energy, and a decreased need for sleep are often present but not to the extent that they lead to severe disruption of work or result in social rejection. Irritability, conceit, and boorish behaviour may take the place of the more usual euphoric sociability. The disturbances of mood and behaviour are not accompanied by hallucinations or delusions.

**F30.1 Mania without psychotic symptoms:** Mood is elevated out of keeping with the patient's circumstances and may vary from carefree joviality to almost uncontrollable excitement. Elation is accompanied by increased energy, resulting in overactivity, pressure of speech, and a decreased need for sleep. Attention cannot be sustained, and there is often marked distractibility. Self-esteem is often inflated with grandiose ideas and overconfidence. Loss of normal social inhibitions may result in behaviour that is reckless, foolhardy, or inappropriate to the circumstances, and out of character.

**F30.2 Mania with psychotic symptoms:** In addition to the clinical picture described in F30.1, delusions (usually grandiose) or hallucinations (usually of voices speaking directly to the patient) are present, or the excitement, excessive motor activity, and flight of ideas are so extreme that the subject is incomprehensible or inaccessible to ordinary communication.

Mania with:

- Mood-congruent psychotic symptoms
- Mood-incongruent psychotic symptoms
- Manic stupor

**F31 Bipolar affective disorder:** A disorder characterized by two or more episodes in which the patient's mood and activity levels are significantly disturbed, this disturbance consisting on some occasions of an elevation of mood and increased energy and activity (hypomania or mania) and on others of a lowering of mood and decreased energy and activity (depression). Repeated episodes of hypomania or mania only are classified as bipolar.

Excludes: bipolar disorder, single manic episode (F30.- ) cyclothymia (F34.0)

**F31.0 Bipolar affective disorder, current episode hypomanic:** The patient is currently hypomanic, and has had at least one other affective episode (hypomanic, manic, depressive, or mixed) in the past.

**F31.1 Bipolar affective disorder, current episode manic without psychotic symptoms:** The patient is currently manic, without psychotic symptoms (as in F30.1), and has had at least one other affective episode (hypomanic, manic, depressive, or mixed) in the past.

**F31.2 Bipolar affective disorder, current episode manic with psychotic symptoms:** The patient is currently manic, with psychotic symptoms (as in F30.2), and has had at least one other affective episode (hypomanic, manic, depressive, or mixed) in the past.

**F31.3 Bipolar affective disorder, current episode mild or moderate depression:** The patient is currently depressed, as in a depressive episode of either mild or moderate severity (F32.0 or F32.1), and has had at least one authenticated hypomanic, manic, or mixed affective episode in the past.

**F31.4 Bipolar affective disorder, current episode severe depression without psychotic symptoms:** The patient is currently depressed, as in severe depressive episode without psychotic symptoms (F32.2), and has had at least one authenticated hypomanic, manic, or mixed affective episode in the past.

**F31.5 Bipolar affective disorder, current episode severe depression with psychotic symptoms:** The patient is currently depressed, as in severe depressive episode with psychotic symptoms (F32.3), and has had at least one authenticated hypomanic, manic, or mixed affective episode in the past.

**F31.6 Bipolar affective disorder, current episode mixed:** The patient has had at least one authenticated hypomanic, manic, depressive, or mixed affective episode in the past, and currently exhibits either a mixture or a rapid alteration of manic and depressive symptoms.

**Excludes:** single mixed affective episode (F38.0)

**F31.7 Bipolar affective disorder, currently in remission:** The patient has had at least one authenticated hypomanic, manic, or mixed affective episode in the past and at least one other affective episode (hypomanic, manic, depressive, or mixed) in addition, but is not currently suffering from any significant mood disturbance, and has not done so for several months. Periods of remission during prophylactic treatment should be coded here.

**b) Eating disorders Anorexia nervosa, bulimia. Differential diagnosis and treatment.**

**Anorexia Nervosa: Symptoms and ICD Diagnostic Criteria**

The formal diagnosis of anorexia nervosa is defined by this set of symptoms, which can be evaluated by psychiatrists and other mental health professionals.

The following information is reproduced verbatim from the ICD-10 Classification of Mental and Behavioral Disorders, World Health Organization, Geneva, 1992. (Since the WHO updates the overall ICD on a regular basis, individual clas-

sifications within it may or may not change from year to year; therefore, you should always check directly with the WHO to be sure of obtaining the latest revision for any particular individual classification.) Also see the related diagnostic criteria for bulimia nervosa.

### **F50.0 Anorexia Nervosa**

Anorexia nervosa is a disorder characterized by deliberate weight loss, induced and/or sustained by the patient. The disorder occurs most commonly in adolescent girls and young women, but adolescent boys and young men may be affected more rarely, as may children approaching puberty and older women up to the menopause. Anorexia nervosa constitutes an independent syndrome in the following sense: the clinical features of the syndrome are easily recognized, so that diagnosis is reliable with a high level of agreement between clinicians; follow-up studies have shown that, among patients who do not recover, a considerable number continue to show the same main features of anorexia nervosa, in a chronic form.

Although the fundamental causes of anorexia nervosa remain elusive, there is growing evidence that interacting sociocultural and biological factors contribute to its causation, as do less specific psychological mechanism and a vulnerability of personality. The disorder is associated with undernutrition of varying severity, with resulting secondary endocrine and metabolic changes and disturbances of bodily function. There remains some doubt as to whether the characteristic endocrine disorder is entirely due to the undernutrition and the direct effect of various behaviours that have brought it about (e.g. restricted dietary choice, excessive exercise and alterations in body composition, induced vomiting and purgation and the consequent electrolyte disturbances), or whether uncertain factors are also involved.

#### **Diagnostic Guidelines**

For a definite diagnosis, all the following are required:

- Body weight is maintained at least 15% below that expected (either lost or never achieved), or Quetelet's body-mass index is 17.5 or less. Prepubertal patients may show failure to make the expected weight gain during the period of growth.
- The weight loss is self-induced by avoidance of "fattening foods" and one or more of the following: self-induced vomiting; self-induced purging; excessive exercise; use of appetite suppressants and/or diuretics.
- There is body-image distortion in the form of a specific psychopathology whereby a dread of fatness persists as an intrusive, overvalued idea and the patient imposes a low weight threshold on himself or herself.
- A widespread endocrine disorder involving the hypothalamic-pituitary-gonadal axis is manifest in women as amenorrhea and in men as a loss of sexual interest and potency. (An apparent exception is the persistence of vaginal bleeds in anorexic women who are receiving replacement hormonal therapy, most common-

ly taken as a contraceptive pill.) There may also be elevated levels of growth hormone, raised levels of cortisol, changes in the peripheral metabolism of the thyroid hormone, and abnormalities of insulin secretion.

- If onset is prepubertal, the sequence of pubertal events is delayed or even arrested (growth ceases; in girls the breasts do not develop and there is a primary amenorrhea; in boys the genitals remain juvenile). With recovery, puberty is often completed normally, but the menarche is late.

### **Differential Diagnosis**

There may be associated depressive or obsessional symptoms, as well as features of a personality disorder, which may make differentiation difficult and/or require the use of more than one diagnostic code. Somatic causes of weight loss in young patients that must be distinguished include chronic debilitating diseases, brain tumors, and intestinal disorders such as Crohn's disease or a malabsorption syndrome.

### **F50.2 Bulimia Nervosa: Symptoms and ICD Diagnostic Criteria**

The formal diagnosis of bulimia nervosa is defined by this set of symptoms, which can be evaluated by psychiatrists and other mental health professionals.

Bulimia nervosa is a syndrome characterized by repeated bouts of overeating and an excessive preoccupation with the control of body weight, leading the patient to adopt extreme measures so as to mitigate the "fattening" effects of ingested food. The term should be restricted to the form of the disorder that is related to anorexia nervosa by virtue of sharing the same psychopathology. The age and sex distribution is similar to that of anorexia nervosa, but the age of presentation tends to be slightly later. The disorder may be viewed as a sequel to persistent anorexia nervosa (although the reverse sequence may also occur). A previously anorexic patient may first appear to improve as a result of weight gain and possibly a return of menstruation, but a pernicious pattern of overeating and vomiting then becomes established. Repeated vomiting is likely to give rise to disturbances of body electrolytes, physical complications (tetany, epileptic seizures, cardiac arrhythmias, muscular weakness), and further severe loss of weight.

For a definite diagnosis, all the following are required:

- There is a persistent preoccupation with eating, and an irresistible craving for food; the patient succumbs to episodes of overeating in which large amounts of food are consumed in short periods of time.

- The patient attempts to counteract the "fattening" effects of food by one or more of the following: self-induced vomiting; purgative abuse, alternating periods of starvation; use of drugs such as appetite suppressants, thyroid preparations or diuretics. When bulimia occurs in diabetic patients they may choose to neglect their insulin treatment.

- The psychopathology consists of a morbid dread of fatness and the patient sets herself or himself a sharply defined weight threshold, well below the premorbid weight that constitutes the optimum or healthy weight in the opinion of the physician. There is often, but not always, a history of an earlier episode of anorexia nervosa, the interval between the two disorders ranging from a few months to several years. This earlier episode may have been fully expressed, or may have assumed a minor cryptic form with a moderate loss of weight and/or a transient phase of amenorrhea.

Includes: anorexia nervosa

### **Differential Diagnosis**

- upper gastrointestinal disorders leading to repeated vomiting (the characteristic psychopathology is absent);
- a more general abnormality of personality (the eating disorder may coexist with alcohol dependence and petty offences such as shoplifting);
- depressive disorder (bulimic patients often experience depressive symptoms).

### **Treatment Eating disorders.**

Anorexia nervosa is difficult to treat because of the shame, denial, and lack of insight concomitant with the disorder. Medical management is directed toward correcting and preventing the disease's complications. Reestablishing normal eating patterns is crucial to restoring the patient's health.

Hospital admission may be indicated for patients who are extremely ill, have cardiac dysrhythmias, or have severe metabolic abnormalities. Most patients will be admitted to medical facilities for refeeding, referred to psychiatric facilities and counseling if medically stable, or be managed on an outpatient basis.

Outpatient treatment should be undertaken only with very close monitoring, such as weekly weight measurement with the patient wearing only a gown.

As with all psychiatric and behavioral emergencies, care must be taken to prove and document competency upon discharge. Many patients with anorexia nervosa may have additional psychopathology, which may leave them incapacitated during an anorexic crisis. If doubt remains, the patient must be admitted for more thorough psychiatric and physiologic monitoring or be discharged in the care of a competent caretaker.

Transfer to an inpatient psychiatric facility may be the disposition for patients who are medically safe for discharge but who require aggressive inpatient psychiatric treatment of their disorder.

### **Psychological therapy**

Various psychological therapies have proven helpful in treating patients with anorexia nervosa, including the following:



- Individual therapy (insight-oriented)
- Cognitive analytic therapy
- Cognitive behavioral therapy (CBT)
- Interpersonal therapy (IPT)
- Motivational enhancement therapy
- Dynamically informed therapies
- Group therapy
- Family therapy
- Conjoint family therapy
- Separated family therapy
- Multifamily groups
- Relatives and caregiver support groups

Individuals with anorexia nervosa may respond best to family based treatment, also known as the Maudsley method, an established therapeutic modality for achieving and maintaining remission from anorexia nervosa.

#### Psychopharmacologic therapy

Evidence regarding the efficacy of medication treatment for eating disorders has tended to be weak or moderate. However, fluoxetine has been found to be generally helpful in patients with anorexia nervosa who have been stabilized with weight restoration. Psychotherapy with adjunctive low-dose olanzapine may be useful for anorexia nervosa during inpatient treatment, especially in the context of anxiety, obsessive eating-related ruminations, and treatment resistance due to failure to engage.

The use of medication in individuals with anorexia nervosa is limited to the treatment of medical complications. To treat osteopenia and to prevent further bone loss, daily dietary intake of calcium 1000-1500 mg and vitamin D 400 IU are recommended. Estrogen replacement (i.e., oral contraceptives) has also been recommended for the treatment of osteopenia, although the benefits and minimal effective dose is being explored. Bisphosphonate therapy can be effective, but the patient should be closely monitored for osteonecrosis of the mandible.

Evidence regarding the efficacy of medication treatment for eating disorders has tended to be weak or moderate, especially as side effects tend to limit long-term compliance compared with the time devoted to psychotherapeutic treatments. However, randomized, controlled trials have shown benefits from the use of medication in combination with cognitive behavioral therapy (CBT).

Fluoxetine was found to be generally helpful in patients with anorexia nervosa who had been stabilized with weight restoration. Psychotherapy with adjunctive low-dose olanzapine may be useful for anorexia nervosa during inpatient treatment, especially in the context of anxiety, obsessive eating-related rumina-

tions, and treatment resistance due to failure to engage. Higher-dose fluoxetine and/or topiramate may be helpful in bulimia nervosa. At this time, however, medication for weight loss in bulimia nervosa is not recommended, due to significant adverse effects such as pulmonary hypertension and heart failure.

Antidepressive and neuroleptic agents, although not reported to be effective, have a limited use in patients who have adequate nutrition and mood changes associated with anorexia nervosa. Prolongation of the QT interval is a contraindication to tricyclic antidepressants because a prolonged QT may increase the risk of ventricular tachycardia and death.

#### SSRIs and SNRIs

Selective serotonin reuptake inhibitors (SSRIs) have been shown to be beneficial in patients with bulimia nervosa but not anorexia. However, since many patients with anorexia have concurrent mood disorders, medication may be of benefit.

In patients with anorexia nervosa who have attained 85% of their expected weight, the SSRI fluoxetine has been used to stabilize recovery. Zinc and cyproheptadine have not been useful. SSRIs and serotonin norepinephrine reuptake inhibitors (SNRIs) may be more helpful for addressing concurrent obsessive-compulsive issues and, owing to their relative neutral effect on weight, may be more easily accepted by the patient.

SSRIs are greatly preferred over the other classes of antidepressants. Because the adverse effect profile of SSRIs is less prominent, improved compliance is promoted. SSRIs do not have the cardiac arrhythmia risk associated with tricyclic antidepressants. Arrhythmia risk is especially pertinent in overdose, and suicide risk must always be considered in the treatment of a child or adolescent with a mood disorder.

In a meta-analysis of 8 studies involving 221 patients with anorexia nervosa, antipsychotics failed to show efficacy for body weight or other anorexia-related outcomes. Pharmacotherapy should not be the only line of treatment and should be used with caution in suspected bipolar disorder, but it may be helpful for depression. Most patients who recover from anorexia nervosa will have been treated with a multidisciplinary approach that includes medication, psychotherapy, nutritional counseling, and frequent medical evaluations.

#### c) **Sleep disorders**

##### **F51 Nonorganic sleep disorders**

Scientists investigated comprehensive epidemiological studies using a sample representative of the general population; they found that a current complaint of insomnia was reported by 32.2% of the respondents. In addition, 7.1% of the respondents suffered from excessive sleep, either current or past, 11.2% had a problem with nightmares and 2.5% reported having sleepwalking, either current or past.

Concurrent psychiatric diagnoses are common in individuals with sleep disturbances. In their 1989 study, Ford and Kamerow demonstrated that 40% of respondents with insomnia and 46.5% of respondents with hypersomnia had a psychiatric disorder, compared with 16.4% of individuals with no sleep complaints. Anxiety disorders were found to be the most common mental disorders, in both insomnia and hypersomnia (23.9% and 27.6%, respectively). The prevalence of major depression, alcohol abuse or other substance abuse was also increased.

The DSM-IV sleep disorders section (7) consists of:

Primary sleep disorders, subdivided into dyssomnias and parasomnias.

Sleep disorders related to another mental disorder.

Other sleep disorders, e.g., sleep disorder due to a general medical condition and substance-induced sleep disorder.

In ICD-10, non-organic sleep disorders are listed with mental and behavioral disorders. The section of non-organic sleep disorders is divided into: a) dyssomnia, i.e. predominant disturbance in the amount, quality, or timing of sleep due to emotional causes (non-organic insomnia, non-organic hypersomnia, non-organic disorder of sleep-wake schedule), and b) parasomnias, i.e. abnormal episodic events occurring during sleep (sleepwalking, sleep terrors, nightmares). Non-psychogenic sleep disorders, such as narcolepsy or sleep apnoea, are placed in chapter 6 of ICD-10.

### **Insomnia and psychopathology**

It is virtually axiomatic that a disturbance of the mind can manifest itself in the sleeping state as well as in the waking state. A wealth of data on sleep in mental disorders has been accumulated to date. Clinical manifestations of chronic insomnia were thoroughly documented in a series of journal articles published in the 1970s and 1980s by Kales's group, which were integrated into a monograph on the evaluation and treatment of insomnia.

Insomnia is a condition of heterogeneous origin. Multiple diagnoses are the rule, not the exception. Stressful life events or stressors of everyday life are triggering factors, and maladaptive habits contribute to the development and persistence of insomnia. However, some predisposing factors, such as female gender and family history of sleep disturbances, increase the vulnerability to insomnia. McCarren et al, using the Vietnam Era Registry, demonstrated that genetic effects were stronger predictors of self-reported insomnia than combat exposure.

For many years, insomnia has been viewed as a disorder of minor importance, although it was clear that insomniacs have poorer physical and mental health, and attempt suicide four times more often than controls. Prospective epidemiological studies consistently report that insomniacs are at greater risk for developing a depressive disorder. Ford and Kamerow were the first to demonstrate that individuals who complained of insomnia at baseline and one year later had a

greater risk of developing new depression over the intervening year. Eaton et al found that sleep problems identify 47% of the new cases of major depression occurring in the next year, and sleep problems are a better predictor of full-blown depression than thoughts of or wishes for death, feeling of worthlessness and guilt, psychomotor retardation, weight problems or fatigue. In the Breslau et al study, insomniacs were at nearly four times higher risk for developing a new depressive disorder in the following 3.5 years. Data from epidemiological studies indicate that the risk for developing new anxiety disorders and alcohol abuse is also greater for insomniacs.

In a review of ten epidemiological studies on the association between heart disease and insomnia, Schwartz et al concluded that sleep complaints are a marker for chronic stress which results in autonomic dysfunction and increased risk of myocardial infarction.

### **Therapy of sleep disorders**

1. Psychological treatment: providing the patient with information on normal sleep, sleep hygiene, sleep disorders in an individual or group therapy setting; relaxation strategies, behavioral therapy.

2. Pharmacological treatment: hypnotics, tranquilizers, antidepressants, neuroleptics, psychostimulants, dopaminergic agents.

## **Topic № 4**

**a) Neurotic disorders related to stress and somatoform disorders. Agoraphobia with panic attacks. Generalized anxiety disorder. Obsessive-compulsive disorder. Conversion disorders. Somatoform disorders.**

**b) Personality disorders. Clinical manifestations and treatment of mature personality disorders.**

**a) Neurotic disorders related to stress and somatoform disorders. Agoraphobia with panic attacks. Generalized anxiety disorder. Obsessive-compulsive disorder. Conversion disorders. Somatoform disorders.**

Neurosis is a class of functional mental disorders involving distress but neither delusions nor hallucinations.

Neurosis may also be called psychoneurosis or neurotic disorder.

There are many different neuroses: obsessive-compulsive disorder, obsessive-compulsive personality disorder, impulse control disorder, anxiety disorder, hysteria, and a great variety of phobias. According to C. George Boeree, professor emeritus at Shippensburg University, the symptoms of neurosis may involve: anxiety, sadness or depression, anger, irritability, mental confusion, low sense of self-

worth, etc., behavioral symptoms such as phobic avoidance, vigilance, impulsive and compulsive acts, lethargy, etc., cognitive problems such as unpleasant or disturbing thoughts, repetition of thoughts and obsession, habitual fantasizing, negativity and cynicism, etc. Interpersonally, neurosis involves dependency, aggressiveness, perfectionism, schizoid isolation, socio-culturally inappropriate behaviors, etc.

The word neurosis means “nerve disorder”, and was first coined in the late eighteenth century by William Cullen, a Scottish physician. Cullen's concept of neurosis encompassed those nervous disorders and symptoms that do not have a clear organic cause. Freud later used the term anxiety neurosis to describe mental illness or distress with extreme anxiety as a defining feature.

### **Categories**

The neurotic disorders are distinct from psychotic disorders in that the individual with neurotic symptoms has a firm grip on reality, and the psychotic patient does not. There are several major traditional categories of psychological neuroses. These include:

- Anxiety neurosis. Mental illness defined by excessive anxiety and worry, sometimes involving panic attacks and manifesting itself in physical symptoms such as tremor, chest pain, sweating, and nausea.
- Depressive neurosis. A mental illness characterized by a profound feeling of sadness or despair and a lack of interest in things that were once pleasurable.
- Obsessive-compulsive neurosis. The persistent and distressing recurrence of intrusive thoughts or images (obsessions) and repetitive behaviors or mental acts (compulsions).
- Somatization (formerly called hysterical neurosis). The presence of real and significant physical symptoms that cannot be explained by a medical condition, but are instead a manifestation of anxiety or other mental distress.
- Post-traumatic stress disorder (also called war or combat neurosis). Severe stress and functional disability caused by witnessing a traumatic event such as war combat or any other event that involved death or serious injury.
- Compensation neurosis. Not a true neurosis, but a form of malingering, or feigning psychological symptoms for monetary or other personal gain.

### **Neurotic, stress-related and somatoform disorders**

(F40-F48)

Excl.: when associated with conduct disorder in F91.- (F92.8)

### **F40 Phobic anxiety disorders**

A group of disorders in which anxiety is evoked only, or predominantly, in certain well-defined situations that are not currently dangerous. As a result these situations are characteristically avoided or endured with dread. The patient's con-

cern may be focused on individual symptoms like palpitations or feeling faint and is often associated with secondary fears of dying, losing control, or going mad. Contemplating entry to the phobic situation usually generates anticipatory anxiety. Phobic anxiety and depression often coexist. Whether two diagnoses, phobic anxiety and depressive episode, are needed, or only one, is determined by the time course of the two conditions and by therapeutic considerations at the time of consultation.

#### **F40.0 Agoraphobia**

A fairly well-defined cluster of phobias embracing fears of leaving home, entering shops, crowds and public places, or travelling alone in trains, buses or planes. Panic disorder is a frequent feature of both present and past episodes. Depressive and obsessional symptoms and social phobias are also commonly present as subsidiary features. Avoidance of the phobic situation is often prominent, and some agoraphobics experience little anxiety because they are able to avoid their phobic situations.

Agoraphobia without history of panic disorder

Panic disorder with agoraphobia

#### **F40.1 Social phobias**

Fear of scrutiny by other people leading to avoidance of social situations. More pervasive social phobias are usually associated with low self-esteem and fear of criticism. They may present as a complaint of blushing, hand tremor, nausea, or urgency of micturition, the patient sometimes being convinced that one of these secondary manifestations of their anxiety is the primary problem. Symptoms may progress to panic attacks.

Anthropophobia

Social neurosis

#### **F40.2 Specific (isolated) phobias**

Phobias restricted to highly specific situations such as proximity to particular animals, heights, thunder, darkness, flying, closed spaces, urinating or defecating in public toilets, eating certain foods, dentistry, or the sight of blood or injury. Though the triggering situation is discrete, contact with it can evoke panic as in agoraphobia or social phobia.

Acrophobia

Animal phobias

Claustrophobia

Simple phobia

Excl.: dysmorphophobia (nondelusional) (F45.2); nosophobia (F45.2)

## **F41 Other anxiety disorders**

Disorders in which manifestation of anxiety is the major symptom and is not restricted to any particular environmental situation. Depressive and obsessional symptoms, and even some elements of phobic anxiety, may also be present, provided that they are clearly secondary or less severe.

### **F41.0 Panic disorder [episodic paroxysmal anxiety]**

The essential feature is recurrent attacks of severe anxiety (panic), which are not restricted to any particular situation or set of circumstances and are therefore unpredictable. As with other anxiety disorders, the dominant symptoms include sudden onset of palpitations, chest pain, choking sensations, dizziness, and feelings of unreality (depersonalization or derealization). There is often also a secondary fear of dying, losing control, or going mad. Panic disorder should not be given as the main diagnosis if the patient has a depressive disorder at the time the attacks start; in these circumstances the panic attacks are probably secondary to depression.

Panic:

- attack
- state

Excl.: panic disorder with agoraphobia (F40.0)

### **F41.1 Generalized anxiety disorder**

Anxiety that is generalized and persistent but not restricted to, or even strongly predominating in, any particular environmental circumstances (i.e. it is “free-floating”). The dominant symptoms are variable but include complaints of persistent nervousness, trembling, muscular tensions, sweating, lightheadedness, palpitations, dizziness, and epigastric discomfort. Fears that the patient or a relative will shortly become ill or have an accident are often expressed.

Anxiety:

- neurosis
- reaction
- state

Excl.: neurasthenia (F48.0)

### **F41.2 Mixed anxiety and depressive disorder**

This category should be used when symptoms of anxiety and depression are both present, but neither is clearly predominant, and neither type of symptom is present to the extent that justifies a diagnosis if considered separately. When both anxiety and depressive symptoms are present and severe enough to justify individual diagnoses, both diagnoses should be recorded and this category should not be used.

Anxiety depression (mild or not persistent)

### **F41.3 Other mixed anxiety disorders**

Symptoms of anxiety mixed with features of other disorders in F42 – F48. Neither type of symptom is severe enough to justify a diagnosis if considered separately.

### **F42 Obsessive-compulsive disorder**

The essential feature is recurrent obsessional thoughts or compulsive acts. Obsessional thoughts are ideas, images, or impulses that enter the patient's mind again and again in a stereotyped form. They are almost invariably distressing and the patient often tries, unsuccessfully, to resist them. They are, however, recognized as his or her own thoughts, even though they are involuntary and often repugnant. Compulsive acts or rituals are stereotyped behaviours that are repeated again and again. They are not inherently enjoyable, nor do they result in the completion of inherently useful tasks. Their function is to prevent some objectively unlikely event, often involving harm to or caused by the patient, which he or she fears might otherwise occur. Usually, this behaviour is recognized by the patient as pointless or ineffectual and repeated attempts are made to resist. Anxiety is almost invariably present. If compulsive acts are resisted the anxiety gets worse.

Incl.: anankastic neurosis; obsessive-compulsive neurosis

Excl.: obsessive-compulsive personality (disorder) (F60.5)

### **F42.0 Predominantly obsessional thoughts or ruminations**

These may take the form of ideas, mental images, or impulses to act, which are nearly always distressing to the subject. Sometimes the ideas are an indecisive, endless consideration of alternatives, associated with an inability to make trivial but necessary decisions in day-to-day living. The relationship between obsessional ruminations and depression is particularly close and a diagnosis of obsessive-compulsive disorder should be preferred only if ruminations arise or persist in the absence of a depressive episode.

### **F42.1 Predominantly compulsive acts (obsessional rituals)**

The majority of compulsive acts are concerned with cleaning (particularly handwashing), repeated checking to ensure that a potentially dangerous situation has not been allowed to develop, or orderliness and tidiness. Underlying the overt behaviour is a fear, usually of danger either to or caused by the patient, and the ritual is an ineffectual or symbolic attempt to avert that danger.

### **F42.2 Mixed obsessional thoughts and acts**

### **F43 Reaction to severe stress, and adjustment disorders**

This category differs from others in that it includes disorders identifiable on the basis of not only symptoms and course but also the existence of one or other of two causative influences: an exceptionally stressful life event producing an acute stress reaction, or a significant life change leading to continued unpleasant circumstances that result in an adjustment disorder. Although less severe psychosocial



stress (“life events”) may precipitate the onset or contribute to the presentation of a very wide range of disorders classified elsewhere in this chapter, its etiological importance is not always clear and in each case will be found to depend on individual, often idiosyncratic, vulnerability, i.e. the life events are neither necessary nor sufficient to explain the occurrence and form of the disorder. In contrast, the disorders brought together here are thought to arise always as a direct consequence of acute severe stress or continued trauma. The stressful events or the continuing unpleasant circumstances are the primary and overriding causal factor and the disorder would not have occurred without their impact. The disorders in this section can thus be regarded as maladaptive responses to severe or continued stress, in that they interfere with successful coping mechanisms and therefore lead to problems of social functioning.

### **F43.0 Acute stress reaction**

A transient disorder that develops in an individual without any other apparent mental disorder in response to exceptional physical and mental stress and that usually subsides within hours or days. Individual vulnerability and coping capacity play a role in the occurrence and severity of acute stress reactions. The symptoms show a typically mixed and changing picture and include an initial state of “daze” with some constriction of the field of consciousness and narrowing of attention, inability to comprehend stimuli, and disorientation. This state may be followed either by further withdrawal from the surrounding situation (to the extent of a dissociative stupor – F44.2), or by agitation and over-activity (flight reaction or fugue). Autonomic signs of panic anxiety (tachycardia, sweating, flushing) are commonly present. The symptoms usually appear within minutes of the impact of the stressful stimulus or event, and disappear within two to three days (often within hours). Partial or complete amnesia (F44.0) for the episode may be present. If the symptoms persist, a change in diagnosis should be considered.

Acute:

- Crisis reaction
- Reaction to stress
- Combat fatigue
- Crisis state
- Psychic shock

### **F43.1 Post-traumatic stress disorder**

Arises as a delayed or protracted response to a stressful event or situation (of either brief or long duration) of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone. Predisposing factors, such as personality traits (e.g. compulsive, asthenic) or previous history of neurotic illness may lower the threshold for the development of the syndrome or aggravate

its course, but they are neither necessary nor sufficient to explain its occurrence. Typical features include episodes of repeated reliving of the trauma in intrusive memories (“flashbacks”), dreams or nightmares, occurring against the persisting background of a sense of “numbness” and emotional blunting, detachment from other people, unresponsiveness to surroundings, anhedonia, and avoidance of activities and situations reminiscent of the trauma. There is usually a state of autonomic hyperarousal with hypervigilance, an enhanced startle reaction, and insomnia. Anxiety and depression are commonly associated with the above symptoms and signs, and suicidal ideation is not infrequent. The onset follows the trauma with a latency period that may range from a few weeks to months. The course is fluctuating but recovery can be expected in the majority of cases. In a small proportion of cases the condition may follow a chronic course over many years, with eventual transition to an enduring personality change (F62.0).

Traumatic neurosis

### **F43.2 Adjustment disorders**

State of subjective distress and emotional disturbance is interfering with performance and social functioning, which arise in the period of adaptation to a significant life change or a stressful life event. The stressor may have affected the integrity of an individual's social network (bereavement, separation experiences) or the wider system of social supports and values (migration, refugee status), or represented a major developmental transition or crisis (going to school, becoming a parent, failure to attain a cherished personal goal, retirement). Individual predisposition or vulnerability plays an important role in the risk of occurrence and the shaping of the manifestations of adjustment disorders, but it is nevertheless assumed that the condition would not have arisen without the stressor. The manifestations vary and include depressed mood, anxiety or worry (or mixture of these), a feeling of inability to cope, plan ahead, or continue in the present situation, as well as some degree of disability in the performance of daily routine. Conduct disorders may be an associated feature, particularly in adolescents. The predominant feature may be a brief or prolonged depressive reaction, or a disturbance of other emotions and conduct.

- Culture shock
- Grief reaction
- Hospitalism in children

Excl.: separation anxiety disorder of childhood (F93.0)

### **F44 Dissociative (conversion) disorders**

The common themes that are shared by dissociative or conversion disorders are a partial or complete loss of the normal integration between memories of the past, awareness of identity and immediate sensations, and control of bodily move-

ments. All types of dissociative disorders tend to remit after a few weeks or months, particularly if their onset is associated with a traumatic life event. More chronic disorders, particularly paralyses and anaesthesias, may develop if the onset is associated with insoluble problems or interpersonal difficulties. These disorders have previously been classified as various types of “conversion hysteria”. They are presumed to be psychogenic in origin, being associated closely in time with traumatic events, insoluble and intolerable problems, or disturbed relationships. The symptoms often represent the patient's concept of how a physical illness would be manifest. Medical examination and investigation do not reveal the presence of any known physical or neurological disorder. In addition, there is evidence that the loss of function is an expression of emotional conflicts or needs. The symptoms may develop in close relationship to psychological stress, and often appear suddenly. Only disorders of physical functions normally under voluntary control and loss of sensations are included here. Disorders involving pain and other complex physical sensations mediated by the autonomic nervous system are classified under somatization disorder (F45.0). The possibility of the later appearance of serious physical or psychiatric disorders should always be kept in mind.

Incl.: conversion:

- hysteria
- reaction
- hysteria
- hysterical psychosis

Excl.: malingering (conscious simulation) (Z76.5)

#### **F44.0 Dissociative amnesia**

The main feature is loss of memory, usually of important recent events, that is not due to organic mental disorder, and is too great to be explained by ordinary forgetfulness or fatigue. The amnesia is usually centered on traumatic events, such as accidents or unexpected bereavements, and is usually partial and selective. Complete and generalized amnesia is rare, and is usually part of a fugue (F44.1). If this is the case, the disorder should be classified as such. The diagnosis should not be made in the presence of organic brain disorders, intoxication, or excessive fatigue.

Excl.: alcohol – or other psychoactive substance-induced amnesic disorder (F10 – F19 with common fourth character .6); amnesia (anterograde (R41.1), retrograde (R41.2), nonalcoholic organic amnesic syndrome (F04), postictal amnesia in epilepsy (G40.-)

#### **F44.1 Dissociative fugue**

Dissociative fugue has all the features of dissociative amnesia, plus purposeful travel beyond the usual everyday range. Although there is amnesia for the peri-

od of the fugue, the patient's behaviour during this time may appear completely normal to independent observers.

Excl.: postictal fugue in epilepsy (G40.-)

#### **F44.2 Dissociative stupor**

Dissociative stupor is diagnosed on the basis of a profound diminution or absence of voluntary movement and normal responsiveness to external stimuli such as light, noise, and touch, but examination and investigation reveal no evidence of a physical cause. In addition, there is positive evidence of psychogenic causation in the form of recent stressful events or problems.

Excl.: organic catatonic disorder (F06.1)

stupor:

- Stupor (R40.1)
- catatonic (F20.2)
- depressive (F31-F33)
- manic (F30.2)

#### **F44.3 Trance and possession disorders**

Disorders in which there is a temporary loss of the sense of personal identity and full awareness of the surroundings. Include here only trance states that are involuntary or unwanted, occurring outside religious or culturally accepted situations.

Excl.: states associated with:

- acute and transient psychotic disorders (F23.-)
- organic personality disorder (F07.0)
- postconcussional syndrome (F07.2)
- psychoactive substance intoxication (F10-F19 with common fourth character .0)
- schizophrenia (F20.-)

#### **F44.4 Dissociative motor disorders**

In the commonest varieties there is loss of ability to move the whole or a part of a limb or limbs. There may be close resemblance to almost any variety of ataxia, apraxia, akinesia, aphonia, dysarthria, dyskinesia, seizures, or paralysis.

Psychogenic:

- aphonia
- dysphonia

#### **F44.5 Dissociative convulsions**

Dissociative convulsions may mimic epileptic seizures very closely in terms of movements, but tongue-biting, bruising due to falling, and incontinence of urine are rare, and consciousness is maintained or replaced by a state of stupor or trance.

#### **F44.6 Dissociative anaesthesia and sensory loss**

Anaesthetic areas of skin often have boundaries that make it clear that they are associated with the patient's ideas about bodily functions, rather than medical

knowledge. There may be differential loss between the sensory modalities which cannot be due to a neurological lesion. Sensory loss may be accompanied by complaints of paraesthesia. Loss of vision and hearing are rarely total in dissociative disorders.

Psychogenic deafness

**F44.7 Mixed dissociative (conversion) disorders**

Combination of disorders specified in F44.0-F44.6

**F44.8 Other dissociative (conversion) disorders**

- Ganser syndrome
- Multiple personality

*Psychogenic:*

- confusion
- twilight state

**F45 Somatoform disorders**

The main feature is repeated presentation of physical symptoms together with persistent requests for medical investigations, in spite of repeated negative findings and reassurances by doctors that the symptoms have no physical basis. If any physical disorders are present, they do not explain the nature and extent of the symptoms or the distress and preoccupation of the patient.

Excl.:

- dissociative disorders (F44.-)
- hair-plucking (F98.4)
- lalling (F80.0)
- lisping (F80.8)
- nail-biting (F98.8)
- psychological or behavioral factors associated with disorders or diseases classified elsewhere (F54)
- sexual dysfunction, not caused by organic disorder or disease (F52.-)
- thumb-sucking (F98.8)
- tic disorders (in childhood and adolescence) (F95.-)
- Tourette syndrome (F95.2)
- trichotillomania (F63.3)

**F45.0 Somatization disorder**

The main features are multiple, recurrent and frequently changing physical symptoms of at least two years duration. Most patients have a long and complicated history of contact with both primary and specialist medical care services, during which many negative investigations or fruitless exploratory operations, may have been carried out. Symptoms may be referred to any part or system of the body. The course of the disorder is chronic and fluctuating, and is often associated with dis-

ruption of social, interpersonal, and family behaviour. Short-lived (less than two years) and less striking symptom patterns should be classified under undifferentiated somatoform disorder (F45.1).

- Multiple psychosomatic disorder

Excl.: malingering (conscious simulation) (Z76.5)

#### **F45.1 Undifferentiated somatoform disorder**

When somatoform complaints are multiple, varying and persistent, but the complete and typical clinical picture of somatization disorder is not fulfilled, the diagnosis of undifferentiated somatoform disorder should be considered.

Undifferentiated psychosomatic disorder

#### **F45.2 Hypochondriacal disorder**

The essential feature is a persistent preoccupation with the possibility of having one or more serious and progressive physical disorders. Patients manifest persistent somatic complaints or a persistent preoccupation with their physical appearance. Normal or commonplace sensations and appearances are often interpreted by patients as abnormal and distressing, and attention is usually focused upon only one or two organs or systems of the body. Marked depression and anxiety are often present, and may justify additional diagnoses.

- Body dysmorphic disorder
- Dysmorphophobia (nondelusional)
- Hypochondriacal neurosis
- Hypochondriasis
- Nosophobia

Excl.: delusional dysmorphophobia (F22.8); fixed delusions about bodily functions or shape (F22.-)

#### **F45.3 Somatoform autonomic dysfunction**

Symptoms are presented by the patient as if they were due to a physical disorder of a system or organ that is largely or completely under autonomic innervation and control, i.e. the cardiovascular, gastrointestinal, respiratory and urogenital systems. The symptoms are usually of two types, neither of which indicates a physical disorder of the organ or system concerned. First, there are complaints based upon objective signs of autonomic arousal, such as palpitations, sweating, flushing, tremor, and expression of fear and distress about the possibility of a physical disorder. Second, there are subjective complaints of a nonspecific or changing nature such as fleeting aches and pains, sensations of burning, heaviness, tightness, and feelings of being bloated or distended, which are referred by the patient to a specific organ or system.

(Cardiac neurosis, Da Costa syndrome, Gastric neurosis, Neurocirculatory asthenia)

Psychogenic forms of:

- aerophagy
- cough
- diarrhoea
- dyspepsia
- dysuria
- flatulence
- hiccough
- hyperventilation
- increased frequency of micturition
- irritable bowel syndrome
- pylorospasm

Excl.: psychological and behavioural factors associated with disorders or diseases classified elsewhere (F54)

#### **F45.4 Persistent somatoform pain disorder**

The predominant complaint is of persistent, severe, and distressing pain, which cannot be explained fully by a physiological process or a physical disorder, and which occurs in association with emotional conflict or psychosocial problems that are sufficient to allow the conclusion that they are the main causative influences. The result is usually a marked increase in support and attention, either personal or medical. Pain presumed to be of psychogenic origin occurring during the course of depressive disorders or schizophrenia should not be included here.

Psychalgia

Psychogenic:

- backache
- headache

Somatoform pain disorder

Excl.: backache NOS (M54.9)

pain:

- NOS (R52.9)
- acute (R52.0)
- chronic (R52.2)
- intractable (R52.1)
- tension headache (G44.2)

#### **F45.8 Other somatoform disorders**

Any other disorders of sensation, function and behaviour, not due to physical disorders, which are not mediated through the autonomic nervous system, which are limited to specific systems or parts of the body, and which are closely associated in time with stressful events or problems.

Psychogenic:

- dysmenorrhea
- dysphagia, including “globus hystericus”
- pruritus
- torticollis
- Teeth-grinding

## **F48 Other neurotic disorders**

### **F48.0 Neurasthenia**

Considerable cultural variations occur in the presentation of this disorder, and two main types occur, with substantial overlap. In one type, the main feature is a complaint of increased fatigue after mental effort, often associated with some decrease in occupational performance or coping efficiency in daily tasks. The mental fatiguability is typically described as an unpleasant intrusion of distracting associations or recollections, difficulty in concentrating, and generally inefficient thinking. In the other type, the emphasis is on feelings of bodily or physical weakness and exhaustion after only minimal effort, accompanied by a feeling of muscular aches and pains and inability to relax. In both types a variety of other unpleasant physical feelings is common, such as dizziness, tension headaches, and feelings of general instability. Worry about decreasing mental and bodily well-being, irritability, anhedonia, and varying minor degrees of both depression and anxiety are all common. Sleep is often disturbed in its initial and middle phases but hypersomnia may also be prominent.

Fatigue syndrome

Use additional code, if desired, to identify previous physical illness.

Excl.: asthenia NOS (R53), burn-out (Z73.0), malaise and fatigue (R53), post viral fatigue syndrome (G93.3), psychasthenia (F48.8)

### **F48.1 Depersonalization-derealization syndrome**

A rare disorder in which the patient complains spontaneously that his or her mental activity, body, and surroundings are changed in their quality, so as to be unreal, remote, or automatized. Among the varied phenomena of the syndrome, patients complain most frequently of loss of emotions and feelings of estrangement or detachment from their thinking, their body, or the real world. In spite of the dramatic nature of the experience, the patient is aware of the unreality of the change. The sensorium is normal and the capacity for emotional expression intact. Depersonalization-derealization symptoms may occur as part of a diagnosable schizophrenic, depressive, phobic, or obsessive-compulsive disorder. In such cases the diagnosis should be that of the main disorder.

### **F48.8 Other specified neurotic disorders**

- Dhat syndrome



- Occupational neurosis, including writer cramp
- Psychasthenia
- Psychasthenic neurosis
- Psychogenic syncope

**b) Personality disorders. Clinical manifestations and treatment of mature personality disorders.**

Personality disorders are conditions in which an individual differs significantly from an average person, in terms of how they think, perceive, feel or relate to others.

Changes in how a person feels and distorted beliefs about other people can lead to odd behaviour, which can be distressing and may upset others.

**Common features include:**

- being overwhelmed by negative feelings such as distress, anxiety, worthlessness or anger
- avoiding other people and feeling empty and emotionally disconnected
- difficulty managing negative feelings without self-harming (for example, abusing drugs and alcohol, or taking overdoses) or, in rare cases, threatening other people
- odd behaviour
- difficulty maintaining stable and close relationships, especially with partners, children and professional carers
- sometimes, periods of losing contact with reality

**Symptoms typically get worse with stress.**

People with personality disorders often experience other mental health problems, especially depression and substance misuse. Personality disorders typically emerge in adolescence and continue into adulthood. They may be mild, moderate or severe, and people may have periods of “remission” where they function well.

The following information is reproduced verbatim from the ICD-10 Classification of Mental and Behavioral Disorders, World Health Organization, Geneva, 1992. (Since the WHO updates the overall ICD on a regular basis, individual classifications within it may or may not change from year to year; therefore, you should always check directly with the WHO to be sure of obtaining the latest revision for any particular individual classification.) It provides the common description and guidelines referenced by the diagnostic criteria for each of the individual personality disorders. It is a severe disturbance in the characterological constitution and behavioral tendencies of the individual, usually involving several areas of the personality, and nearly always associated with considerable personal and social disruption. Personality disorder tends to appear in late childhood or adolescence and continues to be manifest into adulthood. It is therefore unlikely that the diag-

nosis of personality disorder will be appropriate before the age of 18 years. General diagnostic guidelines applying to all personality disorders are presented below; supplementary descriptions are provided with each of the subtypes.

Conditions not directly attributable to gross brain damage or disease, or to another psychiatric disorder, meeting the following criteria:

1) markedly disharmonious attitudes and behaviour, involving usually several areas of functioning, e.g. affectivity, arousal, impulse control, ways of perceiving and thinking, and style of relating to others;

2) the abnormal behaviour pattern is enduring, of long standing, and not limited to episodes of mental illness;

3) the abnormal behaviour pattern is pervasive and clearly maladaptive to a broad range of personal and social situations;

4) the above manifestations always appear during childhood or adolescence and continue into adulthood;

5) the disorder leads to considerable personal distress but this may only become apparent late in its course;

6) the disorder is usually, but not invariably, associated with significant problems in occupational and social performance.

For different cultures it may be necessary to develop specific sets of criteria with regard to social norms, rules and obligations. For diagnosing most of the subtypes listed below, clear evidence is usually required of the presence of at least three of the traits or behaviours given in the clinical description.

**F 60.0 Paranoid personality disorder:** A person with a paranoid personality disorder is extremely distrustful and suspicious. Other features include:

A. The general criteria of personality disorder (F60) must be met.

B. At least four of the following must be present:

(1) Excessive sensitivity to setbacks and rebuffs. (2) Tendency to bear grudges persistently, e.g. unforgiveness of insults, injuries or slights. (3) Suspiciousness and a pervasive tendency to distort experience by misconstruing the neutral or friendly actions of others as hostile or contemptuous. (4) A combative and tenacious sense of personal rights out of keeping with the actual situation. (5) Recurrent suspicions, without justification, regarding sexual fidelity of spouse or sexual partner. (6) Persistent self-referential attitude, associated particularly with excessive self-importance. (7) Preoccupation with unsubstantiated “conspiratorial” explanations of events around the subject or in the world at large.

**F 60.1 Schizoid personality disorder:** Someone with a schizoid personality disorder may appear cold and detached, and avoid making close social contact with others. Other features include:

A. The general criteria of personality disorder (F60) must be met.

B. At least four of the following criteria must be present:

(1) Few, if any, activities provide pleasure. (2) Displays emotional coldness, detachment, or flattened affectivity. (3) Limited capacity to express warm, tender feelings for others as well as anger. (4) Appears indifferent to either praise or criticism of others. (5) Little interest in having sexual experiences with another person (taking into account age). (6) Almost always chooses solitary activities. (7) Excessive preoccupation with fantasy and introspection. (8) Neither desires, nor has, any close friends or confiding relationships (or only one). (9) Marked insensitivity to prevailing social norms and conventions; if these are not followed this is unintentional.

### **F60.2 Dissocial personality disorder**

A person with an antisocial personality disorder sees other people as vulnerable and may intimidate or bully others without remorse. They lack concern about the consequences of their actions. Symptoms include:

A. The general criteria of personality disorder (F60) must be met.

B. At least three of the following must be present:

(1) Callous unconcern for the feelings of others. (2) Gross and persistent attitude of irresponsibility and disregard for social norms, rules, and obligations. (3) Incapacity to maintain enduring relationships, though having no difficulty to establish them. (4) Very low tolerance to frustration and a low threshold for discharge of aggression, including violence. (5) Incapacity to experience guilt, or to profit from adverse experience, particularly punishment. (6) Marked proneness to blame others, or to offer plausible rationalizations for the behaviour bringing the subject into conflict with society.

Comments: Persistent irritability and the presence of conduct disorder during childhood and adolescence, complete the clinical picture but are not required for the diagnosis. It is suggested that sub-criteria should be developed to operationalize behaviour patterns specific to different cultural settings concerning social norms, rules and obligations where needed (such as examples of irresponsibility and disregard of social norms).

### **F60.3 Emotionally unstable personality disorder**

#### **F60.30 Impulsive type**

A. The general criteria of personality disorder (F60) must be met.

B. At least three of the following must be present, one of which is (2):

(1) A marked tendency to act unexpectedly and without consideration of the consequences. (2) A marked tendency to quarrelsome behaviour and to conflicts with others, especially when impulsive acts are thwarted or criticized. (3) Liability to outbursts of anger or violence, with inability to control the resulting behavioral explosions. (4) Difficulty in maintaining any course of action that offers no immediate reward. (5) Unstable and capricious mood.

**F60.31 Borderline type**

A. The general criteria of personality disorder (F60) must be met.

B. At least three of the symptoms mentioned above in criterion B (F60.30) must be present, and in addition at least two of the following:

(6) Disturbances in and uncertainty about self-image, aims and internal preferences (including sexual). (7) Liability to become involved in intense and unstable relationships, often leading to emotional crises. (8) Excessive efforts to avoid abandonment. (9) Recurrent threats or acts of self-harm. (10) Chronic feelings of emptiness.

**F 60.4 Histrionic personality disorder:** A person with histrionic personality disorder is anxious about being ignored. As a result, they feel a compulsion (overwhelming urge) to be noticed and the centre of everyone's attention. Features include:

A. The general criteria of personality disorder (F60) must be met.

B. At least four of the following must be present:

(1) Self-dramatization, theatricality, or exaggerated expression of emotions.

(2) Suggestibility easily influenced by others or by circumstances. (3) Shallow and labile affectivity. (4) Continually seeks excitement and activities in which the subject is the centre of attention. (5) Inappropriately seductive in appearance or behaviour. (6) Overly concerned with physical attractiveness.

Comments: Egocentricity, self-indulgence, continuous longing for appreciation, lack of consideration for others, feelings that are easily hurt and persistent manipulative behaviour complete the clinical picture, but are not required for the diagnosis.

**F60.5 Anankastic personality disorder**

Note: Often referred to as obsessive-compulsive personality disorder.

A. The general criteria of personality disorder (F60) must be met.

B. At least four of the following must be present:

(1) Feelings of excessive doubt and caution. (2) Preoccupation with details, rules, lists, order, organization or schedule. (3) Perfectionism that interferes with task completion. (4) Excessive conscientiousness and scrupulousness. (5) Undue preoccupation with productivity to the exclusion of pleasure and interpersonal relationships. (6) Excessive pedantry and adherence to social conventions. (7) Rigidity and stubbornness. (8) Unreasonable insistence that others submit to exactly his or her way of doing things, or unreasonable reluctance to allow others to do things.

**F 60.6 Anxious (avoidant) personality disorder:** A person with avoidant personality disorder appears painfully shy, is socially inhibited, feels inadequate and is extremely sensitive to rejection. Unlike people with schizoid personality

disorders, they desire close relationships with others, but lack the confidence and ability to form them.

A. The general criteria of personality disorder (F60) must be met.

B. At least four of the following must be present:

(1) Persistent and pervasive feelings of tension and apprehension. (2) Belief that oneself is socially inept, personally unappealing, or inferior to others. (3) Excessive preoccupation about being criticized or rejected in social situations. (4) Unwillingness to get involved with people unless certain of being liked. (5) Restrictions in lifestyle because of need of security. (6) Avoidance of social or occupational activities that involve significant interpersonal contact, because of fear of criticism, disapproval or rejection.

**F 60.7 Dependent personality disorder:** A person with dependent personality disorder feels they have no ability to be independent. They may show an excessive need for others to look after them and are “clingy”. Other features include:

A. The general criteria of personality disorder (F60) must be met.

B. At least four of the following must be present:

(1) Encouraging or allowing others to make most of one's important life decisions. (2) Subordination of one's own needs to those of others on whom one is dependent, and undue compliance with their wishes. (3) Unwillingness to make even reasonable demands on the people one depends on. (4) Feeling uncomfortable or helpless when alone, because of exaggerated fears of inability to care for oneself. (5) Preoccupation with fears of being left to take care of oneself. (6) Limited capacity to make everyday decisions without an excessive amount of advice and reassurance from others.

### **F61 Mixed and other personality disorders**

It has not been attempted to provide standard sets of criteria for these mixed disorders, since those doing research in this field will prefer to state their own criteria depending upon the purpose of the study.

#### **F61.0 Mixed personality disorders**

Features of several of the disorders in F60.- are present, but not to the extent that the criteria for any of the specified personality disorders in F60 are met.

#### **F61.1 Troublesome personality changes, not classifiable in F60 or F62**

Not classifiable in F60.- or F62.- and regarded as secondary to a main diagnosis of a coexisting affective or anxiety disorder.

## **Topic № 5**

**a) Organic mental disorders, differential diagnosis, treatment.**

**b) Types of dementia. Differential diagnosis, treatment.**

**c) Epilepsy**

### **a) Organic Mental Disorders – Condition and Symptoms**

Organic Mental Disorders, also referred to as chronic Organic Brain Syndromes, are afflictions of the brain that can lead to severe mental or behavioral problems. They may be permanent or temporary, and can be either hereditary or caused by injury, disease, or a structural or systemic defect in body chemistry or hormones. Organic Mental Disorders do not include disorders that result from substance abuse, nor do they include psychiatric disorders.

Common symptoms of Organic Mental Disorders include confusion, memory loss, loss of brain function, and agitation, but symptoms can differ somewhat based on the condition.

#### **Causes**

Listed below are disorders associated with Organic Mental Syndromes (OBS).

#### **Brain injury caused by trauma**

- Bleeding into the brain (intracerebral hemorrhage)
- Bleeding into the space around the brain (subarachnoid hemorrhage)
- Blood clot inside the skull causing pressure on brain (subdural hematoma)
- Concussion

#### **Breathing conditions**

- Low oxygen in the body (hypoxia)
- High carbon dioxide levels in the body (hypercapnia)
- Cardiovascular disorders

Dementia due to many strokes (multi-infarct dementia), Heart infections (endocarditis, myocarditis), Stroke, Transient ischemic attack (TIA)

#### **Degenerative disorders**

- Alzheimer disease (also called senile dementia, Alzheimer's type)
- Creutzfeldt-Jacob disease
- Diffuse Lewy Body disease
- Huntington disease
- Multiple sclerosis
- Normal pressure hydrocephalus
- Parkinson disease
- Pick disease
- Dementia due to metabolic causes

#### **Kidney disease**

- Liver disease
- Thyroid disease (hyperthyroidism or hypothyroidism)
- Vitamin deficiency (B1, B12, or folate)

## **Infections**

- Any sudden onset (acute) or long-term (chronic) infection
- Blood poisoning (septicemia)
- Brain infection (encephalitis)
- Meningitis (infection of the lining of the brain and spinal cord)
- Prion infections, such as mad cow disease
- Late-stage syphilis
- Complications of cancer can also lead to OBS.

## **Delirium**

Also known as acute confusional state or acute brain syndrome. It is common on medical and surgical wards —a third of elderly patients in hospital have an episode of delirium – so all doctors should be able to recognize and manage it.

Clinical features of delirium: Clouding of consciousness is the most important diagnostic sign. It refers to drowsiness, decreased awareness of surroundings, disorientation in time and place, and distractibility. At its most severe the patient may be unresponsive, but more commonly the impaired consciousness is quite subtle. Because clouding of consciousness may not be apparent, the first clue to the presence of delirium is often one of its other features:

- Fluctuating course, worse at night.
- Visual hallucinations.
- Transient persecutory delusions.
- Irritability and agitation, or somnolence and decreased activity.
- Impaired concentration and memory.
- The differential diagnosis includes dementia

Usually the clinical picture (especially the acute onset and rapid fluctuations), recognition of delirium is followed by an urgent

## **Management of delirium**

Delirium is managed where it occurs – usually in general hospitals.

- In practice (and in an exam), emphasize both the need to search for a cause and for environmental steps whilst this is ongoing. The latter may avoid the need for medication, which can complicate the problem, and should only be used when necessary.

- Antipsychotics are the first-line pharmacological treatment. Haloperidol is often used, by intramuscular injection if it cannot be taken orally. It can be given intravenously but this is rarely required.

- A delirious person may occasionally be a risk to self, other patients, or staff. Call for help, ensure safety, and use physical restraint if essential (e.g. to allow drug to be administered).

- Patients with delirium are often incapable of giving informed consent. Treatment is therefore given under the common law. If continuing interventions without consent are anticipated, the Mental Health Act may be required.

### **Prognosis of delirium**

Prognosis depends on the cause. Within a week the patient is usually better or has died. A quarter have died by 3 months.

### **Case**

A 75-year-old lady was found lying on the floor and taken to hospital. She is drowsy, disorientated in time and place, distractible, and unable to give any history. She thinks you are trying to kill her. She is febrile and hypotensive, but has no neurological signs or injuries. Blood tests and X-rays are performed. She is given oxygen, and antibiotics for the clinical suspicion of septicemia. Her agitation worsens but settles with haloperidol. The GP tells you that there is no past history of note. Blood cultures grow an organism sensitive to the antibiotic. Her condition improves over 72 hours. The haloperidol is tailed off and her cognitive function returns to normal.

### **Organic psychiatric disorders**

The diagnostic rule is to preface the psychiatric label with “organic” and state the etiology. For example, ‘Organic anxiety disorder due to thyrotoxicosis. Each organic syndrome is very rare compared to its “functional” counterpart. In areas with good primary care, psychiatrists rarely see undiagnosed organic psychiatric disorders. However, when an organic syndrome does occur it is essential to recognize it. Detecting an organic disorder requires that you:

- Consider the possibility, with every patient. If this is done, it is easier not to forget to take a brief medical history and conduct a relevant physical examination and order investigations.

- Always include an organic disorder on your list of differential diagnoses in an exam situation.

- Be suspicious if aspects of the psychiatric presentation are unusual. For example, organic syndromes often produce abnormalities in unexpected functions – e.g. anosmia in depression due to a frontal meningioma. There may or may not be an effective treatment for the organic disorder. Regardless, the psychiatric symptoms are still treated with the appropriate pharmacological, psychological and social interventions.

- As a rule, treatment response in an organic disorder is similar to that of its functional equivalent; for example, depression responds to antidepressants whether it is organic or not. The presence of the organic disorder may, however, affect the choice of drug (e.g. avoid TCAs in depression following myocardial infarction).



### **Amnesic syndrome**

Amnesic (or amnestic) syndrome completes the triad of conditions (with dementia and delirium) which affect memory and which always have an organic cause. Its features are

- Selective loss of recent memory.
- Confabulation: the unconscious fabrication of recent events to cover gaps in memory.
- Time disorientation.
- Attention and immediate recall intact.
- Long-term memory and other intellectual faculties intact.

Amnesic syndrome is rare, and in practice difficult to distinguish from some dementias. It is due to damage to the mammillary bodies, hippocampus or thalamus. The usual cause is alcohol induced thiamine deficiency (Korsakov's syndrome), which is treated with thiamine and abstinence. Other causes include herpes simplex encephalitis, severe hypoxia and head injury. The memory deficits are often irreversible.

#### **b) Types of dementia differential diagnosis, treatment.**

According to ICD-10 dementia included in the following sections:

**Dementia (F00-F03)** is a syndrome due to disease of the brain, usually of a chronic or progressive nature, in which there is disturbance of multiple higher cortical functions, including memory, thinking, orientation, comprehension, calculation, learning capacity, language, and judgement. Consciousness is not clouded. The impairments of cognitive function are commonly accompanied, and occasionally preceded, by deterioration in emotional control, social behaviour, or motivation. This syndrome occurs in Alzheimer's disease, in cerebrovascular disease, and in other conditions primarily or secondarily affecting the brain.

Use additional code, if desired, to identify the underlying disease.

#### **F00 Dementia in Alzheimer's disease ( G30.-+ )**

Alzheimer's disease is a primary degenerative cerebral disease of unknown etiology with characteristic neuropathological and neurochemical features. The disorder is usually insidious in onset and develops slowly but steadily over a period of several years. Alzheimer's disease is named after Dr. Alois Alzheimer. In 1906, Dr. Alzheimer noticed changes in the brain tissue of a woman who had died of an unusual mental illness. Her symptoms included memory loss, language problems, and unpredictable behavior. After she died, he examined her brain and found many abnormal clumps (now called amyloid plaques) and tangled bundles of fibers (now called neurofibrillary, or tau, tangles). These plaques and tangles in the brain are still considered some of the main features of Alzheimer's disease. Another feature is the loss of connections between nerve cells (neurons) in the brain. Neurons

transmit messages between different parts of the brain, and from the brain to muscles and organs in the body. During this preclinical stage of Alzheimer's disease, people seem to be symptom-free, but toxic changes are taking place in the brain. Abnormal deposits of proteins form amyloid plaques and tau tangles throughout the brain, and once-healthy neurons stop functioning, lose connections with other neurons, and die.

The damage initially appears to take place in the hippocampus, the part of the brain essential in forming memories. As more neurons die, additional parts of the brain are affected, and they begin to shrink. By the final stage of Alzheimer's, damage is widespread, and brain tissue has shrunk significantly.

Dementia in Alzheimer's disease with early onset ( G30.0+ ) Dementia in Alzheimer's disease with onset before the age of 65, with a relatively rapid deteriorating course and with marked multiple disorders of the higher cortical functions.

- Alzheimer's disease, type 2
- Presenile dementia, Alzheimer's type
- Primary degenerative dementia of the Alzheimer's type, presenile onset

**F00.1. Dementia in Alzheimer's disease with late onset ( G30.1+ )** Dementia in Alzheimer's disease with onset after the age of 65, usually in the late 70s or thereafter, with a slow progression, and with memory impairment as the principal feature.

Alzheimer's disease, type

1 Primary degenerative dementia of the Alzheimer's type, senile onset

**F00.2 Dementia in Alzheimer's disease, atypical or mixed type ( G30.8+ )** Atypical dementia, Alzheimer's type

**F01 Vascular dementia:** Vascular dementia is the result of infarction of the brain due to vascular disease, including hypertensive cerebrovascular disease. The infarcts are usually small but cumulative in their effect. Onset is usually in later life.

Includes: arteriosclerotic dementia

**F01.0 Vascular dementia of acute onset**

Usually develops rapidly after a succession of strokes from cerebrovascular thrombosis, embolism or haemorrhage. In rare cases, a single large infarction may be the cause.

**F01.1 Multi-infarct dementia**

Gradual in onset, following a number of transient ischaemic episodes which produce an accumulation of infarcts in the cerebral parenchyma.

Predominantly cortical dementia

**F01.2 Subcortical vascular dementia**

Includes cases with a history of hypertension and foci of ischaemic destruction in the deep white matter of the cerebral hemispheres. The cerebral cortex is

usually preserved and this contrasts with the clinical picture which may closely resemble that of dementia in Alzheimer's disease.

### **F01.3 Mixed cortical and subcortical vascular dementia**

### **F02 Dementia in other diseases classified elsewhere**

Cases of dementia due, or presumed to be due, to causes other than Alzheimer's disease or cerebrovascular disease. Onset may be at any time in life, though rarely in old age.

**F02.0 Dementia in Pick's disease (G31.0+)** A progressive dementia, commencing in middle age, characterized by early, slowly progressing changes of character and social deterioration, followed by impairment of intellect, memory, and language functions, with apathy, euphoria and, occasionally, extrapyramidal phenomena.

**F02.1 Dementia in Creutzfeldt-Jakob disease (A81.0+)** A progressive dementia with extensive neurological signs, due to specific neuropathological changes that are presumed to be caused by a transmissible agent. Onset is usually in middle or later life, but may be at any adult age. The course is subacute, leading to death within one to two years.

**F02.2 Dementia in Huntington's disease (G10+)** A dementia occurring as part of a widespread degeneration of the brain. The disorder is transmitted by a single autosomal dominant gene. Symptoms typically emerge in the third and fourth decade. Progression is slow, leading to death usually within 10 to 15 years.

Dementia in Huntington's chorea

**F02.3 Dementia in Parkinson's disease (G20+)** A dementia developing in the course of established Parkinson's disease. No particular distinguishing clinical features have yet been demonstrated.

Dementia in:

- paralysis agitans
- parkinsonism

**F02.4 Dementia in human immunodeficiency virus [HIV] disease (B22.0+)** Dementia developing in the course of HIV disease, in the absence of a concurrent illness or condition other than HIV infection that could explain the clinical features.

### **F02.8 Dementia in other specified diseases classified elsewhere**

Dementia in:

- cerebral lipidoses (E75.-+)
- epilepsy ( G40.-+ )
- hepatolenticular degeneration (E83.0+)
- hypercalcaemia (E83.5+)
- hypothyroidism, acquired (E01.-+ , E03.-+)

- intoxications (T36-T65+)
- multiple sclerosis (G35+)
- neurosyphilis (A52.1+)
- niacin deficiency [pellagra] (E52+)
- polyarteritis nodosa (M30.0+)
- systemic lupus erythematosus (M32.-+)
- trypanosomiasis (B56.-+ , B57.-+)
- vitamin B 12 deficiency (E53.8+)

### c) **Epilepsy**

There are several main types of epilepsy

- Consult a neurology text for general coverage of epilepsy. Relationship of psychiatric symptoms with seizures

- Complex partial epilepsy was called psychomotor epilepsy because of the frequency of psychiatric symptoms during seizures. It has also been referred to as temporal lobe epilepsy, though this is not always the site of the seizure focus.

- The risk of schizophrenia is several-fold higher in people with complex partial epilepsy, more so if the focus is in the left temporal lobe, and due to an early developmental abnormality

- The association with sexual dysfunction may be due to the epilepsy, or the medication. Psychiatric presentations can occur with other epilepsies, but are much rarer.

- Absence seizures in children produce transient lapses in concentration or simple automatisms and can be mistaken for a behavioral disorder.

- A generalized seizure disorder can present to a psychiatrist if, for example, the person was found wandering in a postictal delirium. Psychiatric disorders masquerading as epilepsy. Pseudoseizures (hysterical seizures or non-epileptic attack disorder) is a form of dissociative disorder (p. 107). It can be hard to distinguish clinically from a true seizure. EEG monitoring during attacks may be required to make the diagnosis. Pseudoseizures are commoner in people who also have epilepsy.

- Other conditions which can be misdiagnosed as epilepsy include panic attacks, hypoglycemia and schizophrenia. In children, consider temper tantrums and nightmares. Psychological problems associated with having epilepsy. Historically, epilepsy has been attributed to demonic possession and its sufferers seen as irritable, self-centered people with criminal tendencies. Although entirely false, persisting negative attitudes, acting in concert with the real disabilities, probably explain the higher incidence of psychiatric disorders and suicide in epilepsy.

- The commonest psychiatric disorders are anxiety and depressive disorders.

- The suicide risk is increased 5-fold, and more so in those with complex partial seizures.

● Anticonvulsant treatments can compound the psychiatric problems – phenobarbitone causes hyperactivity and irritability in children; phenytoin can produce ataxia and delirium.

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