

NURS 6501

Knowledge Check: Module 6 Student Response

This Knowledge Check reviews the topics in Module 6 and is formative in nature. It is worth 20 points where each question is worth 1 point. You are required to submit a sufficient response of at least 2-4 sentences in length for each question.

Scenario 1: Schizophrenia

A 21-year-old male college student was brought to Student Health Services by his girlfriend who was concerned about changes in her boyfriend's behaviors. The girlfriend says that recently he began hearing voices and believes everyone is out to get him. The student says he is unable to finish school because the voices told him he was not smart enough. The girlfriend relates episodes of unexpected rage and crying. Past medical history noncontributory but family history positive for a first cousin who "had mental problems". Denies current drug abuse but states he smoked marijuana every day during his junior and senior years of high school. He admits to drinking heavily on weekends at various fraternity houses. Physical exam reveals thin, anxious disheveled male who, during conversations, stops talking, cocks his head and appears to be listening to something. There is poor eye contact and conversation is rambling.

Based on the observed behaviors and information from girlfriend, the APRN believes the student has schizophrenia.

Question 1 of 4:

Describe the positive symptoms of schizophrenia and relate those symptoms to the case study patient.

Schizophrenia often changes how individuals think, feel, and behave and vary from person to person. The symptoms can appear and disappear suddenly. No individual has all the symptoms at a time. Positive symptoms of schizophrenia include the highly exaggerated ideologies, perceptions, or actions that show that an individual cannot tell what is real and what is not. Positive symptom include hallucinations, in which the individual sees, hears, smells, or feels things that no one else does; delusion, which implies the beliefs that seem strange to most people and are easy to prove wrong. However, the individual affected might think that some other person is trying to control their brain through things such as TV or the belief that someone is out to get them; confused thoughts and disorganized speech; trouble in concentrating, movement disorders. For instance, in this scenario, the patient believes that someone is out to get him, when that is not true. Besides, the patient changed his behaviors and begun to drink heavily on weekends.

Question 2 of 4:

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Explain the genetics of schizophrenia.

The exact cause of schizophrenia is unknown but a combination of genetics, environment, and altered brain chemistry and structure may play a significant role in its development. Besides, the inheritance pattern is not known. However, the risk of developing schizophrenia is higher among individuals with family member who has schizophrenia compared to the general population. Most individuals with a close relative with schizophrenia will, however not develop the disorder themselves. Genetically, schizophrenia and bipolar disorders have some similarities in that the two share similar risk genes. A predisposition to schizophrenia can run in families. However, in the general population, only one percent of individuals develop it over their lifetime, but if one of the parents had schizophrenia, the children have a 10 percent chance of developing schizophrenia. In the case scenario, the patient has a family history positive for a first cousin who “had mental problems”, implying that he is also at high risk for schizophrenia.

Question 3 of 4:

The APRN reviews recent literature and reads that neurotransmitters are involved in the development of schizophrenia. What roles do neurotransmitters play in the development of schizophrenia?

There are about 100 neurotransmitters in the brain charged with carrying messages from the end of one nerve branch to the cell body of another. In the brain of a person with schizophrenia, something goes wrong with this system of communication. Notably, two neurotransmitters play a substantial role in the development of schizophrenia. They include DOPAMINE and SEROTONIN. An increase in dopamine in certain areas of the brain results in overstimulation and excess sensory information that causes difficulty with concentration, thought process, reality orientation, feelings and behaviors besides, any abnormalities in serotonin activities results in a sensitive brain, to appear as if the nerve cells were sandpapered, thus leading to schizophrenia. The action of dopamine and serotonin explains why the patient in this case scenario experiences episodes of unexpected rage and crying.

Question 4 of 4:

The APRN reviews recent literature and reads that structural problems in the brain may be involved in the development of schizophrenia. Explain what structural abnormalities are seen in people with schizophrenia.

Structural problems in the brain such as complications brought about by other diseases may cause structural damage to the brain, which may develop into schizophrenia in later years. During older ages, structural changes result in alterations or abnormalities of both the white and gray matter. These changes lead to brain abnormalities and start prior to the onset of clinical symptoms of schizophrenia, especially those that concern language processing. The other

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abnormalities include neuroanatomical abnormalities before and after the onset of psychosis, leading to schizophrenia.

Scenario 2: Bipolar Disorder

A 34-year-old female was brought to the Urgent Care Center by her husband who is very concerned about the changes he has seen in his wife for the past 3 months. He states that his wife has had been depressed and irritable, has complaints of extreme fatigue, has lost 10 pounds and has had insomnia. He has come home from work to find his wife sitting in front of the TV and not moving for hours. In the past few days, she suddenly has become very hyperactive, has been talking incessantly, has been easily distracted and seems to “flit from one thing to another.” She hasn’t slept in 3 days. The wife went on an excessive shopping spree for new clothes that resulted in their credit card being denied for exceeding the line of credit. The wife is unable to sit in the exam room and is currently pacing the hallway muttering to herself and is reluctant to talk with or be examined by the APRN. Physical observation shows agitated movements, rapid fire speech, and hyperactivity. Based on the history and observable symptoms, the APRN suspects that the patient has bipolar type 2 disorder. The APRN refers the patient and husband to the Psychiatric Mental Health Nurse Practitioner for evaluation and treatment.

Question 1 of 6:

Discuss the role genetics plays in the development of bipolar 2 disorders.

Bipolar 2 disorder is not only caused by one single gene, but by multiple genes, with each gene contributing only a small percentage of vulnerability, though acting together with other environmental factors like stress, lifestyle, habits and sleep to cause bipolar 2 disorder. The genes involved in bipolar disorder include G72/DAOA, DISC1, NRG1, TPH2, BDNF, 5-HTT, and DAT1. Therefore, genetics play a significant role in the development of bipolar disorder even though the exact inheritance pattern is not clear, but variations in many genes likely combine to increase an individual’s chance of developing bipolar 2 disorders. This combination results in mood swings, ranging from depressive lows to manic highs, as witnessed in this patient. The patient went on a spree of shopping until they run out of credit.

Question 2 of 6:

Explain how the hypothalamic-pituitary-adrenal (HPA) system may be associated with bipolar type 2 disease.

Hypothalamic-pituitary-adrenal (HPA) system is associated with bipolar 2 disorders because it is central to the pathogenesis of depressive symptoms and cognitive deficits, which may further lead to neurocytotoxic effects of raised cortisol levels. Dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis leads to manic episodes experienced by individuals with bipolar 2 disorders may be preceded by an increase in ACTH and cortisol levels, resulting in cognitive

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problems and functional impairments. For instance, the patient in this scenario had irritable behavior and depressed, indicating a compromised HPA system.

Question 3 of 6:

Discuss the role inflammatory cytokines play in the development and exacerbation of bipolar type 2 symptoms

Inflammatory cytokines play some role in the development and exacerbation of bipolar type 2 symptoms. Cytokines are proteins involved in the regulation and orchestration of the immune response. Cytokines can directly affect neuronal activity, thus inducing neuronal excitability and plastic changes in the mood regulating areas of the brain and neuroprogression of the bipolar diathesis. Besides, inflammatory cytokines influence the symptoms and exacerbation of type 2 bipolar by influencing the HPA through effects on HPA axis as it increases the levels of cortisol hormone.

Question 4 of 6:

Discuss the role of the amygdala in bipolar disorder.

Amygdala plays the role of mediating in not only physiologic but also behavioral arousal in response to environmental stimuli. In bipolar disorder, amygdala plays a role in emotion-related brain function. Any damage to amygdala leads to changes in the mood states such as facial expression and social withdrawal, as noted in the patient in this scenario. The patient has suddenly become very hyperactive and has been talking incessantly, indicating changes in behavioral arousal and changes in emotions.

Question 5 of 6:

How does neurochemical dysregulation contribute to bipolar disorders?

Bipolar 2 disorders occur as a result of chemical imbalance in the brain. Therefore, neurochemical dysregulation means that the neurotransmitters, the chemical responsible for controlling the functions of the brain such as noradrenaline, serotonin and dopamine are not balanced. Any faulty in homeostasis between noradrenaline, serotonin and dopamine transporter and receptors lead to depressive and manic phases of bipolar disorders.

Question 6 of 6:

What is the current status of the use of nutraceuticals in management of depression?

Currently, nutraceuticals are increasingly being used in the management and treatment of depression due to limited prescription options. However, research indicates that patients with long-standing depression are not likely candidates for nutraceuticals, which tend to be more

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effective in patients with milder forms of depressive illnesses. Therefore, physicians are encouraged to always monitor the side effects and possible drug-drug interactions, and utilize their critical judgment in deciding if the agents are making any difference.

Scenario 3: Panic Disorders and Attacks

A 27-year-old female presents to the Emergency Room, with a chief complaint of palpitations, rapid heart rate, sweating, tremors, and inability to catch her breath. The symptoms started about 10 hours ago and have gotten worse. She states she has some chest pain that remains constant no matter what. She also has numbness and tingling around her mouth and lips. She says she knows something “terrible is going to happen”. She denies having any similar episode in the past. Past medical history noncontributory. Social history significant for recent stressor of applying for medical school and taking the Medical College Admission Test (MCAT). She had not received the results prior to the episode but is sure that she failed the test. Says she doesn’t know if anyone else in her family has had similar episodes. Physical exam reveals a thin, anxious appearing female who is profusely sweating despite cool ambient air temperature. BP 176/88, Pulse 136, and respirations 26. Electrocardiogram negative for evidence of myocardial infarction and all lab data within normal limits except for mild respiratory alkalosis. The patient’s symptoms are subsiding and the patient states she is feeling better. The APRN suspects the patient has just experienced a panic attack.

Question 1 of 2:

What are panicogens and how do they contribute to the development of panic attack symptoms?

Panicogens are substances that cause panic or anxiety. They include severe stress, Yohimbine, CCK tetrapeptide (CCK-4), caffeine, *m*-chlorophenylpiperazine, and benzodiazepine partial inverse agonist FG 7142. These agents cause panic attack by increasing anxiety, nervousness, fear, nausea, palpitations, restlessness, and tremors. For instance, the patient in this scenario had chief complaint of palpitations, rapid heart rate, sweating, and tremors, which are all triggered by the panicogenic agents.

Question 2 of 2:

How does the GABA-benzodiazepine (BZ) receptor system contribute to panic attacks/disorders?

The GABA-benzodiazepine (BZ) receptor system contributes to panic attacks because their blockage with antagonists results in severe anxiety that eventually leads to panic attack. However, increasing GABA receptor function with antagonists reduces anxiety and changes of panic disorders.

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Scenario 4: Social Anxiety Disorder (SAD)

A 21-year-old female college junior makes an appointment to see the APRN in the Student Health Clinic. The student tells the APRN that it has gotten harder and harder for her to attend classes, especially her history class where the class is preparing for the semester's end presentations. She says she is terrified to speak to the class and is considering dropping the class so she will not have to present. She has a significant impairment in social activities and has resigned from her sorority. She is unable to go to the library to study as she feels everyone is looking at her and mocking her. She admits to having some of these symptoms in high school, but the guidance counselor was able to work with her to decrease some of her symptoms. Past medical history noncontributory except for the milder symptoms exhibited in high school. Family history noncontributory. Social history positive for anxiety related to social situations that has had a negative impact on both her scholarly and social endeavors. The APRN diagnoses the student with social anxiety disorder (SAD).

Question 1 of 2:

Describe the areas of the brain that are associated with social anxiety disorder.

The areas of the brain that are mostly associated with social anxiety include brain stem, prefrontal cortex, and amygdala, which play a key role in modulating fear and anxiety. Prefrontal cortex is responsible for pathological anxiety responses related to negative emotions caused by amygdala, while brain stem controls the rate of breathing and any fault may result in social anxiety. In these cases, the patient's social anxiety possibly originated from the prefrontal cortex which concerns negative emotions. The patient has a significant impairment in social activities and has resigned from her sorority and is also unable to go to the library to study as she feels everyone is looking at her and mocking her, which are all negative social emotions.

Question 2 of 2:

How is oxytocin associated with SAD?

Oxytocin hormone is associated with SAD as it is known to promote positive social interactions such as feelings of love, social bonding, and well-being. The hormone act by reducing the impact of SAD on and individual and enhance emotional health.

Scenario 5: Generalized Anxiety Disorder (GAD)

A 36-year-old female comes to see the APRN in clinic with a chief complaint of "I'm so and I feel all keyed up all the time". She states she feels restless, keyed up, and on edge most of the time. She fatigues easily and has difficulty concentrating and says her mind goes blank. She admits to being irritable and snapping at her coworkers which she worries will affect her job. She says the symptoms have been present for about 8 or 9 months. and Increased muscle tension. She has had difficulty falling asleep or stay sleeping. Further questioning revealed that prior to her symptoms, her parents got divorced which has been a great stressor for her. Past medical

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history noncontributory. Social history positive for a case of “nerves” when she was in high school that seemed to resolve after she graduated from college. No drug or alcohol history. The APRN believes the patient has generalized anxiety disorder (GAD).

Question 1 of 2:

Discuss the role of neurotransmitters in the expression of GAD.

Several neurotransmitters are involved in GAD such as **serotonin**, glutamate, gamma-amino butyric acid, Cholecystokinin, Adenosine and many others. However, some of these neurotransmitters are inhibitory while others are excitatory. Therefore, these neurotransmitters play a key role in up-regulation or down-regulation of GAD. Neurotransmitters in the brain modulate the neural circuitry involved in anxiety. For instance, the dysregulation of GABA, an inhibitory neurotransmitter, especially the GABA_A variant, tend to elevate the amygdala activity in the brain, thus causing GAD.

Question 2 of 2:

Explain the structural brain changes that occur in people with GAD.

In people with GAD, brain structural changes occurs a result of alterations of the gray and white matter. The structural changes in gray matter include macrostructural changes such as decreased cortical thickness and volume, as well as through microstructural changes such as increased gray matter mean diffusivity. On the other hand, structural changes in white matter include changes in macrostructural lesions measured by the total white matter hyper-intensity (WMH) burden and through microstructural damage in the white matter tracts.

Scenario 6: Post-Traumatic Stress Disorder (PTSD)

A 27-year-old man comes to the Veteran’s Administration Hospital at the insistence of his fiancée who accompanies him to the appointment. She tells the APRN that her fiancée has not “been the same” since he returned from his second tour in Iraq. He was an infantryman with a local Marine Reserve unit and served 2 tours and was honorably discharged. Since his return, he has had difficulty sleeping, and says he “sleeps with one eye open” and fears sleep. Deep sleep brings vivid nightmares. He grudgingly admits to having experienced several traumatic events during his second tour of duty. He is unwilling to discuss them and will not reveal specific details. He is short tempered and irritable and is afraid to be around people as he doesn’t want to snap at people and alienate them. He startles easily at loud noises, especially the sounds of cars backfiring. He admits to thinking there are threats everywhere and spends an excessive amount of time searching for them but never finding any. He has intrusive memories almost every day and says he really isn’t interested in doing much of anything. He is very worried that these symptoms are irreparably hurting his relationship with his fiancée who he loves very much. The APRN diagnoses him with post-traumatic stress disorder (PTSD).

Question 1 of 2:

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Describe the changes seen in the brain structure in patients with PTSD.

Changes in the brain structure in patients with PTSD emanates from severe emotional trauma that causes lasting alterations in the ventromedial prefrontal cortex region of the brain. Prefrontal cortex is responsible for the regulation of responses triggered by amygdale. PTSD patients show a marked decrease in the volume of the ventromedial prefrontal cortex and the functional ability of this region. For instance, the patient in this scenario grudgingly admits to having experienced several traumatic events during his second tour of duty, indicating alterations in the ventromedial prefrontal cortex region of the brain.

Question 2 of 2:

Briefly discuss the role glucocorticoids may have on the development of PTSD

Glucocorticoids play an important role in the development of PTSD. Notably, moderate secretion of glucocorticoids enhances a patients' coping mechanism by helping them to process information in a way that reduces the retrieval of fear-evoking memories. However, when there is inadequate production of glucocorticoids such as cortisol, which is a stress hormone, the individuals may have problems to emotionally adapt following a traumatic event as seen in the patient in this scenario.

Scenario 7: Obsessive-Compulsive Disorder (OCD)

A 17-year-old male high school junior comes to the clinic to establish care. He recently moved from a relatively urban area to a very rural area and has just started his junior year in a new school. The mother states that she has noticed that her son has been frequently washing his hands and avoids contact with any dirty or soiled object. He uses paper towels or napkins over the knob on a door when opening it. According to the mother, this behavior has just appeared since moving. The patient, upon close questioning, admits that he is "grossed out" by some of the boys in the boys' room since they use the toilet and do not wash their hand afterwards. He is worried about all the germs the boys are carrying around. Past medical history is noncontributory. Social history -lives with parents and 2 siblings in a house in a new town. Is an honors student. Based on these behaviors, The APRN thinks the patient has obsessive-compulsive disorder (OCD).

Question 1 of 2:

What is primary pathophysiology of OCD?

Obsessive-compulsive disorder (OCD) is a mental disorder that is characterized by absurd, recurrent, and uncontrollable thoughts that lead to anxiety, followed by repetitive behaviors aimed at reducing anxiety. The primary pathophysiology of OCD is overly persistent and uncontrolled neural activity in SMS, possibly as a result of dopamine-serotonin imbalance. For instance, in the case scenario, the patient admits that he is "grossed out" by some of the boys in the boys' room since they use the toilet and do not wash their hand afterwards. He is also

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worried about all the germs the boys are carrying around; indicating that he is has anxiety from the thought that other boys' hands may be contaminated with germs.

Question 2 of 2:

Describe the role the dorsal anterior cingulate cortex (dACC) has in reinforcement of obsessive behaviors.

In patients with OCD, the dACC acts as a hub that processes negative emotional and reinforcing information and then uses the information to direct motivated behavior. Therefore, it is likely that OCD processes such as anxiety and depression can be reinforced by dACC modulation.