Understandings:

1. Explain excitatory and inhibitory neurons.

- Excitatory neurotransmitters depolarize the membrane voltage, making it closer to the voltage threshold. Example is Acetylcholine.

Inhibitory neurotransmitters hyperpolarize the membrane voltage (increases the negative potential difference), making it further away from the voltage threshold. Example is GABA, where these cause an influx in chloride ions, hence make the membrane potential more negative.

2. Explain summation.

- The sum of these excitatory and inhibitory neurons is the result of our decision. Signals usually come from many different synapses.

3. Distinguish between fast and slow neurotransmitters.

- Fast neurotransmitters act within 1 ms after they have been bound to a receptor on the post-synaptic neuron. And these are the ones that have effect on ion channels! So basically, what we have talked about so far is only fast neurotransmitters.

Slow neurotransmitters, also known as neuromodulators, act after longer than 100 ms after they have been bound to a receptor on the post-synaptic neuron (and even on other neurons too because they have time to diffuse!). These are not responsible for ion channels but rather elicit a secondary messenger inside the post-synaptic neuron.

The next question would be what these secondary messengers do. This will be explained right below.

4. Explain the link between memory and learning with neurotransmitters.

- We know that slow neurotransmitters make post-synaptic neurons produce secondary messengers. These secondary messengers can modulate (adjust) hence the name neuromodulators for slow neurotransmitters.

If a post-synaptic neuron is frequently used, it will develop by these secondary messengers increasing the number of synapses, number of receptors or chemical modification of receptors. When a receptor is “chemically modified” in a positive way, it can increase the rate of ions moving when a fast neurotransmitter excites a voltage gated ion channel next time. Essentially, strengthening signals would mean that the brain is storing a memory or learning (which is a form of memory indeed).

In more professional terms, this persistent strengthening of synapses is called long-term potentiation.

5. Explain what psychoactive drugs are.

- A psychoactive drug is a fancy word for a chemical that (most of the time) acts on the central nervous system to temporarily alter one's perception, consciousness and emotions.

Some drugs may excite a certain action or some drugs may inhibit a certain action.

Examples of excitatory drugs we should know: Nicotine, cocaine, amphetamines.

Examples of inhibitory drugs we should know: Benzodiazepines, alcohol, tetrahydrocannabinol.

These names though…

Anyways, many of you may wonder what kind of drug morphine is. It is in fact both excitatory and inhibitory! So it is not always clear-cut. Dopamine is also not clear-cut.

6. Explain how anesthetics work.

- Anesthesia basically blocks the neurotransmitters from the sensory neurons so the "it hurts" signal never reaches the brain! Or they could just block the ion channels which some anesthetic chemicals do, like cocaine (nowadays doctors use a chemical similar to cocaine, but works in the same way). Others may keep the GABA receptor open so the inhibiting signals keep coming.

Really, there are many different methods and many anesthetic chemicals. But I don't think scientists really know exactly how these works, but we know they work *thumbs up*!

7. Explain stimulant drugs.

- Stimulant drugs basically stimulate a person. The response is very similar to what sympathetic nervous system (response during threat) gives us. It makes us excited, increased heartbeat, increased blood pressure, etc.

These would be a type of excitatory neurotransmitters that specifically increases the factors a sympathetic nervous system would control. The three excitatory neurotransmitters mentioned above are all also stimulant drugs.

This is important to treat depressed patients! Another very useful drug to treat depressed patients is serotonin (an inhibitory drug). Lack of serotonin cause depression so extra doses are given.

Extra notes

- We should be able to explain how an excitatory drug like cocaine affects the brain. First of all, state that it is an excitatory drug. It affects synapses that use dopamine. In normal conditions, dopamine is reabsorbed by presynaptic membrane. Cocaine blocks dopamine reuptake transporters at the presynaptic membrane. Dopamine remains in the synapse and binds again at the postsynaptic membrane. This makes the person feel pleasure.

8. Explain what addiction is in a more neuroscientific way.

- Addiction is a type of neurological chronic disease. Symptoms are:

1. Inability to consistently **A**bstain
2. Impairment in **B**ehavioral control
3. **C**raving for drugs or rewarding experiences
4. **D**iminished recognition of social relationships
5. Dysfunctional **E**motional response

Addiction has to do with the white matter (axon and synapse connections) in the frontal lobe. When the frontal lobe is unable to inhibit certain reward gratification, it can be seen as a start in addiction and increased dopamine secretion. The factors affecting a start in addiction are various and stretch to bio-psycho-socio factors. But some are just genetically more prone to become addicted, some people have bad circumstances (stress, traumatic experience, etc.), and the drug itself once consumed directly affect the dopamine synapses.

Applications and skills:

1. Explain the effects on the nervous system of two stimulants and two sedatives.

- Stimulants and sedatives are two opposite type of drugs. We know that stimulants make people awake, but sedatives (soothing agent) make people drowsy.

Stimulant	Sedative
Pramipexole: This is a molecule that mimics dopamine. Since it directly binds to the dopamine receptor, it is an agonistic drug. Cocaine: This is a molecule that binds to dopamine transporters that reabsorb the dopamine. Thus when it is bound, dopamine repeatedly binds to its receptor. This is an example of antagonistic drug.	Diazepam: This molecule acts similar to GABA. It causes an additional intake of chloride ions that cause hyperpolarization. THC (tetrahydrocannabinol): These inhibits the release of excitatory neurotransmitters.

2. Explain the effect of anesthetics on awareness.

- As mentioned, there are many types of anesthetics. Some may result in loss of complete consciousness and some to a lesser extent depending on what type of surgery it is.

3. Explain how endorphins can act as painkillers.

- Endorphin comes from the word endogenous morphine. These are basically natural pain killers that are secreted by pituitary gland. These act by binding to receptors on pain sensing neurons and block them. In that way, it prevents any pain sensing. In addition it may even block the release of some neurotransmitters.

So during stress and pain, endorphins are released.

4. Be able to evaluate data showing the impact of MDMA (ecstasy) on serotonin and dopamine metabolism in the brain.

- Do the data based question on this!!!

MDMA/ecstasy is a "feel good"-drug. Serotonin is a neurotransmitter related to depression and other emotions. Lack of serotonin would make the person prone to become depressed.