

McCance: Pathophysiology, 6th Edition

Chapter 16: Alterations in Cognitive Systems, Cerebral Hemodynamics, and Motor Function

Key Points – Print

SUMMARY REVIEW

Alterations in Cognitive Systems

1. Full consciousness is an awareness of oneself and the environment and includes an ability to respond to external stimuli with a wide variety of responses.
2. Consciousness has two components: arousal and content of thought.
3. Decreased level of arousal can occur because of diffuse bilateral cortical dysfunction, bilateral subcortical (reticular formation, brainstem) dysfunction, or localized hemispheric dysfunction.
4. An alteration in breathing pattern and level of coma reflects the level of brain dysfunction.
5. Pupillary changes reflect changes in level of brainstem function, drug action, and response to hypoxia and ischemia.
6. Abnormal eye movements, including nystagmus and divergent gaze, reflect alterations in brainstem function.
7. Level of brain function manifests by changes in generalized motor responses or no responses.
8. Loss of cortical inhibition associated with decreased consciousness includes abnormal flexor and extensor movements.
9. Cerebral death or irreversible coma represents permanent brain damage, with an ability to maintain cardiac, respiratory, and other vital functions.
10. Brain death results from irreversible brain damage that includes an inability to maintain internal homeostasis.
11. Arousal returns in the VS and MCS, but content of thought is absent or markedly reduced, respectively.
12. Seizures represent abnormal, excessive hypersynchronous discharges of cerebral neurons with transient alterations in brain function. Seizures may be generalized or focal. There are three categories of epileptic syndrome: location-related, generalized, and undetermined.
13. With a deficit in selective attention, mediated by the brainstem, parietal lobe structures, and the pulvinar nucleus of the thalamus, the individual cannot focus on selective stimuli and thus neglects those stimuli.
14. In dysmnesia and amnesia, some memories are not retrieved and new memories cannot be stored.
15. Frontal areas mediate vigilance, detection, and working memory. With a vigilance deficit, the person cannot maintain search and scanning activities. With a detection deficit, the person is unmotivated and unable to use feedback.

16. Some specific disorders of content of thought (cognition) are agnosias, dysphasias, acute confusional states, and dementias, including AD.
17. Agnosias are a defect of recognition and may be tactile, visual, or auditory. They are caused by dysfunction in the primary sensory area or the interpretive areas of the cerebral cortex.
18. Dysphasia is an impairment of comprehension or production of language. Dysphasia may be expressive or sensory.
19. Aphasia is loss of language comprehension or production.
20. Wernicke dysphasia is a disturbance in understanding all language—verbal and reading comprehension.
21. Conductive dysphasias result from disruption of temporal lobe fibers, with a failure to repeat words but an ability to initiate speech, writing, and reading aloud.
22. Anomic dysphasia is an inability to name objects, people, or qualities.
23. Transcortical dysphasias involve an ability to repeat and recite.
24. Broca aphasia is an expressive dysphasia of speech and writing but with retention of comprehension.
25. Global aphasia involves anterior and posterior speech areas, with expressive and receptive aphasia.
26. Acute confusional states are characterized chiefly by defects in attention and coherence of thoughts and actions and, in the case of delirium, an intense autonomic nervous system hyperactivity.
27. AD is a chronic, irreversible dementia.

Alterations in Cerebral Hemodynamics

1. Cerebral oxygenation is a critical management issue.
2. Cerebral perfusion pressure determines cerebral blood flow.
3. An injured brain may experience cerebral oligemia, normal cerebral blood flow but with increased intracranial pressure, or cerebral hyperemia.
4. Increased intracranial pressure may result from edema, excess CSF, hemorrhage, or tumor growth. When intracranial pressure approaches arterial pressure, hypoxia and hypercapnia produce brain damage.
5. Cerebral edema is an increase in the fluid content of the brain resulting from infection, hemorrhage, tumor, ischemia, infarct, or hypoxia.
6. The shifting or herniation of brain tissue from one compartment to another disrupts the blood flow of both compartments and damages brain tissue.
7. Supratentorial herniation involves temporal lobe and hippocampal gyrus shifting from the middle fossa to the posterior fossa; transtentorial herniation with a downward shift of the diencephalon through the tentorial notch; and shifting of the cingulate gyrus herniation under the falx.

8. The most common infratentorial herniation is a shift of the cerebellar tonsils through the foramen magnum.
9. Hydrocephalus comprises a variety of disorders characterized by an excess of fluid within the cranial vault, subarachnoid space, or both. Hydrocephalus occurs because of interference with CSF flow caused by increased fluid production or obstruction within the ventricular system or by defective reabsorption of the fluid.
10. Hydrocephalus can be treated by reducing CSF in the ventricles through the use of shunts and diuretic therapy if resection of the cause is not possible.

Alterations in Motor Function

1. Motor dysfunction may be characterized as alterations of motor tone, movement, and complex motor performance.
2. Hypotonia and hypertonia are the main categories of altered tone.
3. Four types of hypertonia exist: spasticity, gegenhalten, dystonia, and rigidity.
4. Paresis, paraplegia, hyperkinesia, and hypokinesia are the main categories of altered movement.
5. Two subtypes of paresis and paralysis are described: upper motor neuron and lower motor neuron.
6. An upper motor neuron syndrome is characterized by paresis or paralysis, hypertonia, and hyperreflexia.
7. Interruption of the pyramidal tract below the pons results in spinal shock.
8. Lower motor neuron syndromes manifest with impaired voluntary and involuntary movements.
9. Partial paralysis occurs with only partial loss of alpha motor neurons, and total paralysis is complete loss of alpha motor neurons. Loss of gamma motor neurons impairs muscle tone and decreases tendon reflexes.
10. Amyotrophy (e.g., poliomyelitis) is a lower motor neuron syndrome involving the anterior horn cells, with loss of muscle tone and strength resulting in segmental paresis and hyporeflexia.
11. Nuclear palsies involve damage to the cranial nerve nuclei.
12. Bulbar palsies involve cranial nerves IX, X, and XII.
13. Included in the category of hyperkinesia are chorea, athetosis, ballism, akathisia, tremor, and myoclonus.
14. HD (chorea) is a rare hereditary disease involving the basal ganglia and cerebral cortex. It is inherited as an autosomal dominant trait and commonly manifests between 25 and 45 years of age.
15. The major pathologic feature of HD is severe degeneration of the basal ganglia and the frontal cerebral cortex. The basal ganglia and the substantia nigra exhibit a depletion of

neurons that secrete GABA (an inhibitory neurotransmitter). This depletion leads to an excess of dopaminergic activity that causes involuntary, fragmentary movements.

16. No known treatment is effective in halting the degenerative process in HD.
17. Types of hypokinesia include akinesia, bradykinesia, and loss of associated movements.
18. PD is a common degenerative disorder of the basal ganglia (corpus striatum) involving degeneration of the dopamine-secreting nigrostriatal pathway.
19. Degeneration of the dopaminergic nigrostriatal pathway allows overactivity by the STN to excessively inhibit motor thalamus and motor cortex causing rigidity and bradykinesia. Progressive dementia may be associated with an advanced stage of the disease.
20. Treatment of PD is symptomatic, involving levodopa (L-dopa), a precursor of dopamine.
21. Alterations in complex motor performance include disorders of posture (stance), disorders of gait, and disorders of expression.
22. Disorders of posture include dystonic posture, decerebrate posture, basal ganglion posture, and senile posture.
23. Disorders of gait include upper motor neuron gaits, cerebellar gait, basal ganglion gait, and senile gait.
24. Disorders of expression include hypermimesis, hypomimesis, and dyspraxia or apraxia.
25. Dyspraxia is an impairment of the conceptualization or execution of a complex motor act.
26. Extraparamidal motor syndromes include basal ganglia and cerebellar motor syndromes.
27. Basal ganglia disorders manifest with alterations in muscle tone and posture, including rigidity, involuntary movements, and loss of postural reflexes.
28. Cerebellar motor syndromes result in loss of muscle tone, difficulty with coordination, and disorders of equilibrium and gait.