Unit 4 — Brain Development

LEARNING OUTCOME

At the end of this unit, you will be able to:

1. Describe the phases of neurodevelopment and postnatal cerebral development.
2. Describe neuroplasticity and factors affecting neuroplasticity.
3. Explain the importance of environment in prenatal and postnatal brain development.
4. Evaluate the relevance of neural development and disorders of neurodevelopment to their lives.

The Case of Ali

When Ali was 3 years old, he had a streptococcal infection. He exhibited eye-rolling tics, followed by blinking, head-jerking and nose-rubbing. He also developed obsessive and compulsive disorder (OCD), such as insisting on arranging his color pencils in a particular order, repeated counting, walking in circles while he counts to three and would continually touching certain objects until they feels right. His doctor treated him with amoxicillin (antibiotic), which reduced his tics, but not his OCD.
Biopsychological research promotes greater understanding of disorders causing mental retardation at birth, and paves the way for future treatment possibilities.

Neural development is important to everyone who plans to become a parent. Tissue destined to become the nervous system is differentiated even before a woman might know she is pregnant. Thus, every woman and her male partner need to know health recommendations regarding smoking, alcohol, and caffeine intake, if there is a reasonable chance of pregnancy. Even under ideal conditions, not every baby is born perfect.

During pregnancy, a great excess of neurons is produced—perhaps twice as many as necessary, but these are winnowed out in the final month or so of pregnancy and in the months just after birth.

So great is the profusion of primitive neurons that at least fifty thousand cells are produced during each second of most of intrauterine life to provide the necessary number.

So complex are the challenges involved in developing a brain that at least one half of our entire genome (the full catalogue of human genes on all the chromosomes) is devoted to producing this organ that will constitute only two percent of our body weight. It should be realized at this point that for the nine months of intrauterine life and for a short but indeterminate postnatal period, brain growth and development will be largely genetically determined.

However, environmental (epigenetic) factors will also be involved almost from the beginning of embryonic life, and will assume an increasingly important role. It is, in fact, the complex intertwining of genetic and epigenetic factors which guarantee the uniqueness of each individual.
PHASES OF NEURAL DEVELOPMENT

During pregnancy, the neonate and infancy undergoes several stages of neural development:
1. Induction of the neural plate
2. Incomplete closure of the neural tube
3. Closure of the neural tube
4. Neural proliferation - cell differentiation and division
5. Cell migration
6. Development of temporary connections - axon and synapse formation
7. Maturation of nerve cells
8. Development of myelin sheaths
9. Development of synaptic connections among neurons

**Neural tube:** A hollow tube, closed at the rostral end, that forms from ectodermal tissue early in embryonic development; serves as the origin of the central nervous system.

**Ventricular zone:** A layer of cells that line the inside of the neural tube, contains founder cells that divide and give rise to the central nervous system.

4.1 PRENATAL DEVELOPMENT OF THE CENTRAL NERVOUS SYSTEM

4.1.1 Induction of the Neural Plate

The induction of the neural plate undergoes the following development began with a patch of ectodermal tissue on the dorsal surface of the embryo. Through the release of special chemicals from the mesoderm (the “organizer”), the overlying ectoderm is induced to divide more rapidly, forming a thickened mass called the neural plate.

The neural plate is visible three weeks after conception. It forms three layers of embryonic cells:
- a. Ectoderm – outermost,
- b. Mesoderm – middle,
- c. Endoderm - innermost

A crease or fold soon appears in this plate. The crease rapidly deepens and becomes known as the neural groove. The entire embryo is lengthening as this happens.
The neural groove continues to deepen until its sides, the neural folds, arch over and fuse with each other forming a short segment of completely enclosed tube. This newly formed "neural tube" will become the nervous system.

The actual fusion of the walls to form the tube occurs first in the center of the embryo about midway between front and rear poles of the still rapidly lengthening little organism.

However, you can probably visualize how the newly formed section of neural tube rapidly begins to roof over in both a frontward (anterior or rostral) and a backward (posterior or caudal) direction.

It is as if there were two zippers in the newly formed roof of the developing neural tube. As these zippers are pulled simultaneously away from each other toward the two ends of the embryo, the neural folds come together and the neural tube lengthens progressively in both directions.

Finally, the neural tube is almost completely enclosed in both directions, leaving only a small unroofed portion or opening at each end. These residual openings are called neuropores and under normal developmental conditions will soon be closed, thereby forming a complete neural tube.

During this process a front-back polarity has been established in the still-lengthening embryo. Accordingly, the small unroofed area of the neural tube at the front end is called the anterior neuropore; the one at the rear end, the posterior neuropore.

4.1.2 Incomplete Closure Of The Tube

The capacity of the developing nervous system to follow an incredibly complex series of developmental rules laid down progressively by the genes is remarkable.

Nonetheless, errors occur, and the roofing over of the neural groove to form the neural tube represents one point where disturbed development can severely affect the growing embryo.

Incomplete closure of the anterior or posterior neuropore represents two such developmental errors during the first trimester which radically alter the future life of the embryo/fetus and infant.

If the anterior neuropore fails to close, the resulting deficit leads to varying degrees of incomplete development of the cerebral hemispheres and brain stem.

One of the most frequent and dramatic resulting anomalies is the fetus which is born without cerebral hemispheres and usually without any skull above the level of the eyes. This is the so-called anencephalic child (a- or an- without: cephalon- brain) Strangely enough, this type of extreme anomaly may come to term and under some conditions, live for a week or two following birth.

Such a severely deformed infant has only a brain stem (the upward continuation of the spinal cord within the skull) on which to depend for its behavior. This takes care of its basic
breathing, cardiovascular, suckling and elimination reflexes. However, little else is possible for the infant and it usually dies within a few days or weeks of birth.

If incomplete closure persists at the posterior neuropore, the fetus will be born with some variant of spina bifida (bifida- split). In the most severe of these, the posterior portion of the spinal cord is totally or partially undeveloped and the entire lower back may be open. Some defects of this sort may be amenable to restorative surgery while others are not compatible with life.

There is a more subtle form of this anomaly known as spina bifida occulta (occulta- hidden) where the only residual pathology is a tract or canal, usually of microscopic size, running between the subdural space surrounding the lower tip of the spinal cord and the skin of the lower back.

Often, the only sign of such an anomaly is a little patch of hair in the middle of the lower back just above the beginning of the cleft between the buttocks. Although usually asymptomatic, this tiny canal can become infected, usually through trauma, and can form a painful pus-filled sac known as a pilonidal cyst.

### 4.1.3 Closure Of The Neural Tube

With successful closure of the neural tube, the anterior or rostral (rostral- front) end develops three vesicles which demarcate the territory for cerebral hemispheres and brain stem.

Of these, the first and third divide once more forming a series of five vesicles which will become the major portions of the central nervous system within the skull. These consist of the cerebral hemispheres, diencephalon, midbrain, pons and cerebellum and medulla oblongata.

**Stem cells**

Neural plate cells are often referred to as stem cells. Stem cells seem to have an unlimited capacity for self-renewal and can develop into different mature cell types (totipotent).

The nervous system develops from embryonic tissue called the ectoderm. As the neural tube develops specificity increases, resulting in glial and neural stem cells (multipotent) The first sign of the developing nervous system is the neural plate that can be seen at about the 16th day of development. Over the next few days, a “trench” is formed in the neural plate - this creates a neural groove.

By the 21st day of development, a neural tube is formed when the edges of the neural groove meet. The rostral (front) part of the neural tubes goes on to develop into the brain and the rest of the neural tube develops into the spinal cord. Neural crest cells become the peripheral nervous system.
At the front end of the neural tube, three major brain areas are formed: the prosencephalon (forebrain), mesencephalon (midbrain) and rhombencephalon (hindbrain). By the 7th week of development, these three areas divide again. This process is called encephalization.

**4.1.4 Neural Proliferation**

Neural plate folds to form the neural groove which then fuses to form the neural tube. Inside will be the cerebral ventricles and neural tube. Neural tube cells proliferate in species-specific ways – 3 swellings at the anterior end in humans will become the forebrain, midbrain, and hindbrain

**Cerebral cortex (cortex means “bark”):** The outmost layer of gray matter of the cerebral hemispheres that is about 3 mm thick.

**Radial glia:** Special glia with fibers that grow radially outward from the ventricular zone to the surface of the cortex; provide guidance for neurons migrating outward during brain development.
4.1.5 Cell Migration

Once cells have been created through cell division in the ventricular zone of the neural tube they migrate. Migrating cells are immature, lacking axons and dendrites.

1. Radial migration – towards the outer wall of the tube
2. Tangential migration – at a right angle to radial migration, parallel to the tube walls

Most cells engage in both types of migration.

Two types of neural tube migration:

1. Radial migration – moving out – usually by moving along radial glial cells
2. Tangential migration – moving up

Figure 4.3: Two types of neural tube migration.

Two methods of migration:
1. Somal – an extension develops that leads migration, cell body follows
2. Glial-mediated migration – cell moves along a radial glial network
Neural crest: A structure dorsal to the neural tube and formed from neural tube cells; develops into the cells of the peripheral nervous system; cells migrate long distances.

Aggregation: The process of cells that are done migrating aligning themselves with others cells and forming structures.

Cell-adhesion molecules (CAMs): Aid both migration and aggregation; CAMs found on cell surfaces, recognize and adhere to molecules.

4.1.6 Axon Growth and Synapse Formation

Once migration is complete and structures have formed (aggregation), axons and dendrites begin to grow.

Growth cone: At the growing tip of each extension, extends and retracts filopidia as if finding its way

Chemoaffinity hypothesis: Postsynaptic targets release a chemical that guides axonal growth, but this does not explain the often circuitous routes often observed

Axon growth

To find their proper place in the brain, axons often stretch for several feet, making their way through surrounding tissues and around a myriad of obstacles until they reach their final target. The growth cone then forms a synapse, or a tiny gap where nerve messages are transmitted, with the dendrites of the target neuron.

Cell adhesion molecules are found on neuron surfaces and bind to similar proteins on nearby cells. By knocking out the genes for specific molecules, these proteins found in different combinations on different nerve fibers, help axons recognize and track along paths established by related axons.

Growing axons can also change course to follow gradients of certain "attraction" molecules that spread out from target cells and provide long-range cues. An axon's response to different molecules is determined by proteins called receptors on the surface of the growth cone that the molecules fit into much as a key fits a lock. When a molecule attaches to these receptors, it causes the growth cone to grow or stop or turn.

Cells can change the receptors and other molecules that are active at a given time. Thus, growth cones can respond to different guidance molecules at different stages during their development and change direction.
Mechanisms underlying axonal growth are the same across species. A series of chemical signals exist along the way—attracting and repelling. Such guidance molecules are often released by glia. Adjacent growing axons also provide signals.

**Pioneer growth cones:** The 1st to travel a route—follow guidance molecules.

**Fasciculation:** The tendency of developing axons to grow along the paths established by preceding axons.

**Topographic gradient hypothesis:** Seeks to explain topographic maps. Gopnick et al. (1999) describe neurons as growing telephone wires that communicate with one another.

Following birth, the brain of a newborn is flooded with information from the baby’s sense organs. This sensory information must somehow make it back to the brain where it can be processed.

To do so, nerve cells must make connections with one another, transmitting the impulses to the brain. Continuing with the telephone wire analogy, like the basic telephone trunk lines strung between cities, the newborn’s genes instruct the "pathway" to the correct area of the brain from a particular nerve cell.

For example, nerve cells in the retina of the eye send impulses to the primary visual area in the occipital lobe of the brain and not to the area of language production (Wernicke’s area) in the left posterior temporal lobe.

The basic trunk lines have been established, but the specific connections from one house to another require additional signals.
Synaptogenesis

Synaptogenesis is the formation of new synapses. Neurons that are stimulated by input from the surrounding environment continue to establish new synapses. Synaptogenesis depends on:

- The presence of glial cells, especially astrocytes;
- High levels of cholesterol are needed – supplied by astrocytes;
- Chemical signal exchange between pre and postsynaptic neurons is needed; and
- A variety of signals act on developing neurons.

Neurons seldom stimulated soon lose their synapses, a process called synaptic pruning.

Neuron Death and Synapse Rearrangement

50% more neurons than are needed are produced, death is normal. Neurons die due to failure to compete for chemicals provided by targets:

- Increase targets leads to decreased death;
- Destruction of some cells leads to increased survival of remaining cells; and
- Increase number of innervating axons leads to decreased proportion survive.
**Life-preserving chemicals**

Neurotrophins promote growth and survival, guide axons, stimulate synaptogenesis. Both passive cell death (necrosis) and active cell death (apoptosis) occurs. Apoptosis is safer than necrosis. It is “cleaner”, leaving less debris and glutamate in the brain.

**The central nervous system in the womb**

Ten week human fetus is 1.25 cm (0.5 in.) long and consists mostly of ventricle. Twenty weeks fetus is 5 cm (2 in.) long with basic brain shape. At times during brain development, as many as 250,000 neurons are added per minute.

At birth, almost all the neurons that the brain will ever have are present. However, the brain continues to grow for a few years after birth. By the age of 2 years old, the brain is about 80% of the adult size. End product at adulthood is approximately 1400 g (3 lb)

Glia continues to divide and multiply. Glia carries out many important functions for normal brain function including insulating nerve cells with myelin. The neurons in the brain also make many new connections after birth.

**Age Brain Weight**

Average brain weights at different times of development:

<table>
<thead>
<tr>
<th>AGE</th>
<th>BRAIN WEIGHT</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 weeks of gestation</td>
<td>100 grams</td>
</tr>
<tr>
<td>Birth</td>
<td>400 grams</td>
</tr>
<tr>
<td>18 months old</td>
<td>800 grams</td>
</tr>
<tr>
<td>3 years old</td>
<td>1100 grams</td>
</tr>
<tr>
<td>Adult</td>
<td>1300-1400 grams</td>
</tr>
</tbody>
</table>

**ACTIVITY 4.1**

(a) Briefly explain the phases of neurodevelopment.
(b) Explain the different kinds of neural tube migration.
Postnatal growth is a consequence of:
- Synaptogenesis
- Myelination – sensory areas and then motor areas. Myelination of prefrontal cortex continues into adolescence
- Increased dendritic branches

Overproduction of synapses may underlie the greater plasticity of the young brain.

### 4.2.1 Development of the Prefrontal Cortex

Believed to underlie age-related changes in cognitive function, no single theory explains the function of this area. Prefrontal cortex plays a role in working memory, planning and carrying out sequences of actions, and inhibiting inappropriate responses.

### 4.2.2 Effects of Experience on Neural Circuits

Neurons and synapses that are not activated by experience usually do not survive – use it or lose it. Humans are uniquely slow in neurodevelopment – allows for fine-tuning.

When a baby is born he has billions of brain cells, and that many of these brain cells are not connected. Relate early experience to how nature and nurture interact to modify the early development, maintenance, and reorganization of neural circuits discussed previously.

#### Early Studies of Experience and Neurodevelopment

- Early visual deprivation:
  - Fewer synapses and dendritic spines in 1° visual cortex
  - Deficits in depth and pattern vision

- Enriched environment:
  - Thicker cortices
  - Greater dendritic development
  - More synapses per neuron

The impact we can have on those first 3 years a child’s brain is critical to every type of development (cognitive, emotional, & physical).

#### Competitive Nature of Experience and Neurodevelopment

Monocular deprivation changes the pattern of synaptic input into layer IV of V1. Altered exposure during a sensitive period leads to reorganization. Active motor neurons take precedence over inactive ones.
Effects of Experience on Topographic Sensory Cortex Maps

Cross-modal rewiring experiments demonstrate the plasticity of sensory cortices – with visual input, auditory cortex can see. Change input, change cortical topography - shifted auditory map in prism-exposed owls.

Neural activity prior to sensory input plays a role in development – ferret visual development disrupted by interference with neuronal activity prior to eye opening. Early music training influences the organization of human auditory cortex – fMRI studies

Mechanisms by Which Experience Might Influence Neurodevelopment

Many possibilities

• Neural activity regulates the expression of genes that direct the synthesis of CAMs
• Neural activity influences the release of neurotrophins
• Some neural circuits are spontaneously active and this activity is needed for normal development

4.2.3 Cerebral Hemispheres

Lateralization: The specialization of one of the cerebral hemispheres to handle a particular function; myelinization of the corpus callosum.

Left hemisphere: The hemisphere that controls the right side of the body, coordinates complex movements, and, in 95% of right-handers and 62% of left-handers, controls most functions of speech and written language.

Right hemisphere: The hemisphere that controls the left side of the body and that, in most people, is specialized for visual-spatial perception and interpreting nonverbal behavior.

Unilateral neglect: Patients with right hemisphere damage may have attentional deficits and be unaware of objects in the left visual field

Right Hemisphere’s Role In Emotion

The right hemisphere is involved in our expression of emotion through tone of voice and facial expressions; controls the left side of the face, which usually conveys stronger emotion than the right side of the face.

Lawrence Miller describes the facial expressions and the voice inflection of people with right hemisphere damage as “often strangely blank–almost robotic”
Handedness, Culture, And Genes

The corpus callosum of left-handers is 11% larger and contains up to 2.5 million more nerve fibers than that of right-handers. In general, the two sides of the brain are less specialized in left-handers.

Left-handers tend to experience less language loss following an injury to either hemisphere. Left-handers tend to have higher rates of learning disabilities and mental disorders than right-handers.

**ACTIVITY 4.2**

(a) Explain the effects of an enriched and deprived environment on a child’s brain.
(b) Explain the specialization of the right and left cerebral hemispheres.
NEUROPLASTICITY

Brain facts – True or false?

Mature brain changes and adapts

FACT 1: Neuroplasticity includes several different processes that take place throughout a lifetime.

Neuroplasticity does not consist of a single type of morphological change, but rather includes several different processes that occur throughout an individual’s lifetime. Many types of brain cells are involved in neuroplasticity, including neurons, glia, and vascular cells.

FACT 2: Neuroplasticity has a clear age-dependent determinant.

Although plasticity occurs over an individual’s lifetime, different types of plasticity dominate during certain periods of one’s life and are less prevalent during other periods.

FACT 3: Neuroplasticity occurs in the brain under two primary conditions:

1. During normal brain development when the immature brain first begins to process sensory information through adulthood (developmental plasticity and plasticity of learning and memory).
2. As an adaptive mechanism to compensate for lost function and/or to maximize remaining functions in the event of brain injury.

FACT 4: The environment plays a key role in influencing plasticity.

In addition to genetic factors, the brain is shaped by the characteristics of a person’s environment and by the actions of that same person.
4.3.1 Brain Plasticity: Synaptic Pruning

Over the first few years of life, the brain grows rapidly. As each neuron matures, it sends out multiple branches (axons, which send information out, and dendrites, which take in information), increasing the number of synaptic contacts and laying the specific connections from house to house, or in the case of the brain, from neuron to neuron.

At birth, each neuron in the cerebral cortex has approximately 2,500 synapses. By the time an infant is two or three years old, the number of synapses is approximately 15,000 synapses per neuron (Gopnick, et al., 1999). This amount is about twice that of the average adult brain. As we age, old connections are deleted through a process called synaptic pruning.

Synaptic pruning eliminates weaker synaptic contacts while stronger connections are kept and strengthened. Experience determines which connections will be strengthened and which will be pruned; connections that have been activated most frequently are preserved.

Neurons must have a purpose to survive. Without a purpose, neurons die through a process called apoptosis in which neurons that do not receive or transmit information become damaged and die. Ineffective or weak connections are "pruned" in much the same way a gardener would prune a tree or bush, giving the plant the desired shape.

It is plasticity that enables the process of developing and pruning connections, allowing the brain to adapt itself to its environment.

4.3.2 Injury-induced Plasticity: Plasticity and Brain Repair

During brain repair following injury, plastic changes are geared towards maximizing function in spite of the damaged brain.

In studies involving rats in which one area of the brain was damaged, brain cells surrounding the damaged area underwent changes in their function and shape that allowed them to take on the functions of the damaged cells. Although this phenomenon has not been widely studied in humans, data indicate that similar (though less effective) changes occur in human brains following injury.

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4.3.3 Effects of Experience on the Reorganization of the Adult Cortex

Tinnitus (ringing in the ears) – produces major reorganization of 1° auditory cortex. Adult musicians who play instruments fingered by hand have an enlarged representation of the hand in right somatosensory cortex. Skill training leads to reorganization of motor cortex.

4.4 DISORDERS OF NEURODEVELOPMENT

4.4.1 Autism

Autism is found in 4 of every 10,000 individuals. There are 3 core symptoms:

1. Reduced ability to interpret emotions and intentions
2. Reduced capacity for social interaction
3. Preoccupation with a single subject or activity

Intensive behavioral therapy may improve function.

Autism is heterogenous, level of brain damage and dysfunction varies. Most have some abilities preserved – rote memory, ability to complete jigsaw puzzles, musical ability, artistic ability.

Savants are intellectually handicapped individuals who display specific cognitive or artistic abilities. 1/10 autistic individuals display savant abilities. Perhaps it is a consequence of compensatory functional improvement in the right hemisphere following damage to the left.

A brief observation in a single setting cannot present a true picture of an individual’s abilities and behaviors. Parental (and other caregivers’ and/or teachers) input and developmental history are very important components of making an accurate diagnosis.

There are no medical tests for diagnosing autism. An accurate diagnosis must be based on observation of the individual’s communication, behavior, and developmental levels.

At first glance, some persons with autism may appear to have mental retardation, a behavior disorder, problems with hearing, or even odd and eccentric behavior. To complicate matters further, these conditions can co-occur with autism.

However, it is important to distinguish autism from other conditions, since an accurate diagnosis and early identification can provide the basis for building an appropriate and effective educational and treatment program.

Early Diagnosis

Research indicates that early diagnosis is associated with dramatically better outcomes for individuals with autism. The earlier a child is diagnosed, the earlier the child can begin benefiting from one of the many specialized intervention approaches.
**Diagnosis Tools for Autism**

The characteristic behaviors of autism spectrum disorders may or may not be apparent in infancy (18 to 24 months), but usually become obvious during early childhood (24 months to 6 years).

The National Institute of Child Health and Human Development (NICHD) lists five behaviors that signal further evaluation is warranted:

1. Does not babble or coo by 12 months
2. Does not gesture (point, wave, grasp) by 12 months
3. Does not say single words by 16 months
4. Does not say two-word phrases on his or her own by 24 months
5. Has any loss of any language or social skill at any age.

Having any of these five "red flags" does not mean your child has autism.

**Neural Basis of Autism**

**Genetic basis**

Siblings of the autistic have a 5% chance of being autistic; 60% concordance rate for monozygotic twins. Several genes interacting with the environment may cause autism. There is evidence for a role of a gene on chromosome 7.

Brain damage tends to be widespread, but is most commonly seen in the cerebellum. Thalidomide, given early in pregnancy, increases chance of autism.

Neurodevelopmental error occurs within 1st few weeks of pregnancy when motor neurons of the cranial nerves are developing. Consistent with observed deficits in face, mouth, and eye control. Anomalies in ear structure indicate damage occurs between 20 and 24 days after conception.

**4.4.2 Williams Syndrome**

Occurs in 1 of every 20,000 births. Williams syndrome is marked by mental retardation and an uneven pattern of abilities and disabilities.

- Sociable, empathetic, and talkative – exhibit language skills, music skills and an enhanced ability to recognize faces
- Profound impairments in spatial cognition
- Usually have heart disorders associated with a mutation in a gene on chromosome 7 – the gene (and others) are absent in 95% of those with Williams

Williams Syndrome is a rare disorder. Like autism it is caused by an abnormality in chromosome 7, and shows a wide variation in ability from person to person.

Underdeveloped occipital and parietal cortex, normal frontal and temporal “elfin” appearance – short, small upturned noses, oval ears, broad mouths.
Williams people have a unique pattern of emotional, physical and mental strengths and weaknesses.

For parents, teachers, and care workers, learning about this pattern can be a key to understanding a Williams person and in helping them achieve their full potential.

It is a non-hereditary syndrome which occurs at random and can effect brain development in varying degrees, combined with some physical effects or physical problems. These range from lack of co-ordination, slight muscle weakness, possible heart defects and occasional kidney damage.

### 4.4.3 Hypercalcaemia

A high calcium level and is often discovered in infancy, and normal development is generally delayed. The incidence is approximately 1 in 25,000. By 2002 over 1300 cases were known in the UK and similar organisations have now sprung up in the USA, New Zealand, Canada, Australia and most countries in Europe.

#### ACTIVITY

(a) Explain the differences between Autism and Williams Syndrome.

(b) How can parents and teachers help children with Autism and/or Williams Syndrome?
1. The process of the growth of a fertilized egg into a mature nervous system is called _________.

2. The first major phase of neurodevelopment is formation of the _______.

3. Most cell division in the developing neural tube occurs in the _______.

4. In addition to the radial migration of developing neurons along glial cells, there is considerable ________ migration.

5. When the lips of the neural _________ fuse, the result is the neural tube.

6. The neural crest develops into the _______.

7. An exciting recent finding is that synaptogenesis depends on the presence of _________.

8. Between birth and adulthood, the size of the human brain _______.

9. The last part of the brain to achieve full myelination is the _______.

10. The growth of new neurons in adults is called adult _________.

11. The prefrontal cortex seems to play a role in _________.

12. Neurons and synapses that are not activated by experience usually _________.

13. Adult stem cells that migrate to the olfactory bulbs are created at certain sites in the _________.

14. The area of somatosensory cortex receiving input from the left hand was found to be bigger in _________.

15. Autism usually becomes apparent _________.

16. A core symptom of autism is _________.

17. Despite being intellectually handicapped, about 10% of autistic individuals display _________ abilities.

18. ________ taken early in pregnancy causes autism in the infant.

19. Considering their mental retardation, Williams people have remarkably good _________.

Suggested Text and References

Required Reading

Suggested Readings
Self-check

1. neurodevelopment.
2. neural plate.
3. ventricular zone
4. tangential
5. groove
6. peripheral nervous system.
7. astrocytes.
8. quadruples
9. prefrontal cortex
10. neurogenesis
11. working memory, planning and carrying out sequences of action.
12. do not survive.
13. ependymal layer.
14. musicians who finger stringed instruments with the left hand.
15. before the age of 3 years.
16. reduced ability to detect emotions and intentions of others, reduced capacity for social interaction and communication.
17. Savant
18. Thalidomide
19. language skills