Paramedic Resource Manual

2005 UPDATED BY

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ADVANCED LIFE SUPPORT
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RESOURCE MATERIAL
The Paramedic Resource Manual Modules contain the information necessary to meet the objectives. However, for additional information, the following texts are recommended:


**Advanced Trauma Life Support Manual**. Committee on Trauma, American College of Surgeons, 2004.


Paramedic Resource Manual

RESPIRATORY SYSTEM
SECTION ONE

2005 Update by
Ontario Base Hospital Group Education Subcommittee

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RESPIRATORY SYSTEM: OBJECTIVES

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. identify and locate, relative to other anatomic structures, the components of the upper and lower respiratory tracts.

2. describe the function of the components of the upper and lower respiratory tracts.

3. describe the mechanics of inspiration and expiration, in association with the respiratory pressures in each phase.

4. state normal pulmonary volumes and capacities in the adult patient.

5. define compliance.

6. briefly explain ventilation/perfusion ratio.

7. describe the influence of the central nervous system on respiration.

8. briefly describe the pathophysiology and clinical presentation of:
   - Asthma
   - COPD
   - Emphysema
   - Bronchitis.

9. relate the objectives to clinical situations you may encounter in the field, utilizing the EMCA level skills of patient assessment and airway management.

If you have studied this subject previously, you may test your ability using the self assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.
Adequate patient care demands that each paramedic be able to assess a patient’s airway and ventilation and manage any problems that exist. This assessment cannot be done well without a knowledge of the basic anatomy and function of the respiratory system.

The structure of the respiratory tract is closely correlated with its function. The architecture consists of a series of air passages that convey air to the lungs. As air passes through the upper conducting channels, it is filtered, warmed and humidified.

UPPER RESPIRATORY TRACT

The sequence of organs that comprise the upper conducting pathway are:

- Nasal cavity and sinuses
- Pharynx
- Larynx
- Trachea.

NASAL CAVITY

Reasons to know the nose:

- Placement of nasopharyngeal airways
- Placement of nasotracheal tubes
- Nose bleeds (epistaxis)
- Fractured nose.

The nasal cavities are separated from each other by the nasal septum. They open in front at the nostrils (anterior nares) and posteriorly into the nasopharynx through the posterior nares (choanae). The septum is composed of cartilage anteriorly and bone posteriorly. It is covered by a very vascular mucous membrane.

Most nose bleeds in younger people originate at the anterior nasal septum, while those in older people are often from the posterior nasal structures.
On the lateral wall of each nasal cavity are three scroll-like elevations of bone covered with mucous membrane. These are the superior, middle and inferior turbinates.

Displacement of the turbinates is responsible for the unpleasant "crunching" noise often encountered in placement of a nasotracheal tube. Should turbinate laceration occur, significant bleeding may result.
The cribiform plate is the thin layer of bone that separates the brain from the nasal cavity. Its multiple perforations allow entry of the olfactory nerve fibres into the nose. With facial or head trauma the cribiform plate may fracture, allowing leakage of cerebrospinal fluid into the nose (CSF rhinorrhea). Attempting to pass any tube through the nose in such a patient may result in the tube passing through the fracture into the cranium with disastrous consequences.

SINUSES

Sinus Highlights

- Sinus infections can lead to headache.
- Ethmoid sinuses occasionally rupture with pressure changes.
- Sinusitis may lead to brain abscess.
- Air-fluid level in the sphenoid sinus may indicate a basal skull fracture.

The nasal sinuses are the ethmoidal, frontal, maxillary and sphenoid. Each sinus is a mucous membrane lined, air-filled cavity within the bony architecture of the skull which drains into the nasal cavity.

Obstruction of drainage from a sinus can lead to pain and infection (sinusitis). Occasionally an infected sinus will erode through its bony box into the cranium leading to a brain abscess. With rapid depressurization, e.g. a rapid surfacing from depth, an ethmoidal sinus may rupture leading to facial subcutaneous emphysema.

**FIGURE 3: FRONTAL AND SIDE VIEW OF SKULL AND SINUSES**

![Diagram of skull and sinuses](image)
Often basal skull fractures extend into the sphenoidal sinuses, resulting in bleeding. The presence of an air fluid level in the sphenoid sinus following head trauma may be the only radiographic sign of the fracture.

**THE PHARYNX**

Pharyngeal Facts

- Foreign bodies may lodge in the pharynx (especially laryngopharynx).
- Abscesses in this area may lead to airway obstruction.
- Swelling secondary to allergic reactions or burns may lead to airway obstruction

The pharynx is the space extending from the base of the skull to the larynx. The part behind the nose is the nasal pharynx (nasopharynx), behind the mouth is the oral pharynx (oropharynx) and behind the larynx is, you guessed it, the laryngeal pharynx (laryngopharynx).

![FIGURE 4: LATERAL ASPECT OF THE PHARYX](image)
When looking though the mouth into the oral pharynx one can see the soft palate, uvula and tonsils.

**FIGURE 5: VIEWTHROUGH OPEN MOUTH**

![Diagram of the oral anatomy showing the soft palate, uvula, tonsil, and tongue.]

**THE LARYNX**

Don't be lax about the larynx

- Airway obstruction, often due to foreign bodies at this level.
- Any mucosal swelling of larynx can cause airway obstruction.
- The larynx is very vulnerable to direct trauma.
- Trauma to the larynx is often associated with cervical spine injury.
- Emergency airway can be obtained by inserting cannulae through the cricothyroid membrane.
Disruption of normal laryngeal function or structure may lead to airway compromise. Common problems are:

1. Laryngeal foreign bodies
2. Epiglottitis
3. Edema, secondary to either burns, chemical inflammation, or allergic reactions
4. Trauma, with secondary bleeding and swelling, e.g., inexpert attempts at intubation
5. Trauma, with gross disruption of laryngeal structures with associated major airway obstruction. Often air will leak out of the larynx leading to subcutaneous emphysema.

The larynx extends from the hyoid bone to the lower border of the cricoid cartilage. It lies anterior to the 3rd to the 6th cervical vertebrae (hence the common association of laryngeal and cervical fractures). The larynx is composed of cartilages united by ligaments and moved by muscles. The whole structure is lined with mucous membrane.

The four major laryngeal cartilages are the epiglottis, thyroid, arytenoids, and cricoid.
Take a moment to palpate your own neck while referring to Figure 7 and reading the discussion which follows.

Immediately below your chin you will find a mobile bone of 4-5 mm in width which moves superiorly when you swallow. This is the hyoid bone. It resembles a horseshoe in shape.

Below the hyoid bone one next encounters the thyroid cartilage. It is easily identified by its most prominent protuberance, the "Adam's Apple". The thyroid cartilage acts as both an attachment for and protection to the vocal cords. The ligament that connects the hyoid bone and thyroid cartilage is thyrohyoid ligament.

Immediately below the thyroid cartilage one can palpate a gap and below this which is the cricoid cartilage. The ligament between the thyroid and cricoid cartilages is the cricothyroid ligament (and membrane).
You must be able to reliably locate the space between the thyroid and cricoid cartilages. A cannula may be placed through this membrane to allow oxygenation and ventilation of the patient (cricothyroidotomy).

The epiglottis is found behind the hyoid. It cannot be palpated through the skin.

The epiglottis is a semirigid cartilagenous structure found immediately posterior to the base of the tongue. It is directly behind the hyoid bone. (During oral-tracheal intubation, one must lift the epiglottis anteriorly in order to visualize the vocal cords). The epiglottis is 2-3 mm thick and has a curved shape when viewed from above. It is normally pale pink in colour. During swallowing, the epiglottis slides superiorly and posteriorly to cover the top of the larynx. This prevents aspiration of foreign material into the larynx. (The coughing and sputtering we have all experienced when something "goes down the wrong way" illustrates what happens if the epiglottis doesn't prevent this). Any swelling of the epiglottis can lead to significant airway obstruction.

The vocal cords (ligaments) stretch from the back of the thyroid cartilage to the arytenoid cartilages. They are mobile structures capable of opening and closing.

**FIGURE 8: THE VOICE APPARATUS (INTERIOR OF LARYNX, SUPERIOR ASPECT)**
The vocal cords are opened by muscles controlled by the recurrent laryngeal nerves. Damage to one recurrent laryngeal nerve results in ipsilateral cord paralysis. This results in a hoarse voice. Damage to both nerves leads to marked airway obstruction.

**TRACHEA**

Below the cricoid cartilage one may palpate the first few tracheal rings. The trachea extends from the cricoid cartilage, opposite the sixth cervical vertebra, to the fifth thoracic vertebra.

The trachea bifurcates into the right and left mainstem bronchi. The area of bifurcation is called the carina.

The trachea is about 10 cm long and lies immediately anterior to the esophagus.

**Clinical vignette**

Trachea is only 10 cm long, therefore beware you don’t insert the endotracheal tube too deeply.
Each of the 16-20 tracheal cartilages is horseshoe shaped with the opening facing posteriorly.

**FIGURE 10: RELATIONSHIP OF TRACHEA AND ESOPHAGUS**

The lack of cartilagenous continuity of the tracheal rings is important for food to move through the esophagus, i.e. the posterior wall of the trachea can bulge anteriorly allowing most objects to pass.

**Clinical vignette**

Because the rings of the trachea a semi-lunar and do not completely encircle the trachea, the posterior wall is soft and compressible. A large foreign body lodged in the esophagus can obstruct the trachea and drastically restrict air movement. This is especially true in infants and smaller children. Glucagon, given IV and in a higher dose than that which is used to raise blood sugar levels, will relax the esophagus and may relieve an obstruction.

The trachea is lined by ciliated columnar epithelium. It is these cilia that sweep foreign matter including bacteria from the trachea.

In summary, the upper respiratory tract consists of the nose, larynx, pharynx and trachea. It serves to conduct air to the lower respiratory tract and warms, moistens and filters the air during passage.
LOWER RESPIRATORY TRACT

When air has passed through the structures of the upper respiratory tract, it enters the **lower respiratory tract** which consists, in a descending order, of:

1. Right and left mainstem bronchi
2. Secondary bronchi
3. Tertiary bronchi
4. Bronchioles
5. Terminal bronchioles
6. Respiratory bronchioles
7. Alveolar ducts
8. Alveolar sacs
9. Alveoli

There are also associated structures, including the **pleura, pleural cavity, bony thorax** and muscles of respiration.

BRONCHI

The trachea ends by dividing into the right and left mainstem bronchi (singular= bronchus). These are also referred to as the primary bronchi. Each bronchus enters the lung through the **hilum**, as shown in Figure 11. The mainstem bronchi are similar in structure to the trachea, in that they have the characteristic rings of cartilage and their lining is ciliated epithelium. However, as progressive subdivision ensues, less and less cartilage is present in their walls. The cartilage is replaced by smooth muscle.

**FIGURE 11: BRONCHI AT HILI OF LUNGS**
RIGHT BRONCHUS

The right mainstem bronchus, approximately five centimeters long, is shorter, more vertical in direction and of larger calibre than the left. It passes to the root or hilum of the corresponding lung (Figure 11).

Clinical vignette

Because the right mainstem bronchus is almost in a direct line with the trachea, most foreign bodies that pass the carina end up in the right mainstem bronchus. This is also true for endotracheal tubes that have been inserted too far.

LEFT BRONCHUS

The left bronchus is a little longer than five centimeters and leaves the tracheal bifurcation at a greater angle (about 45°) than does the right (about 25°) (Figure 12).

BRONCHIAL TREE

Each bronchus, upon entering the lung, will divide into secondary bronchi (Figure 12), which in turn will sub-divide into smaller units, the tertiary bronchi. Each tertiary bronchus leads to a bronchopulmonary segment. There are ten such segments in the right lung and nine in the left.

The division of the bronchi continues, in tree-like fashion and indeed, the resultant structure is referred to as the bronchial tree. The fine bronchial tubes at the periphery of the "tree" are the bronchioles.

FIGURE 12: THE BRONCHIAL TREE
BRONCHIOLES

The terminal branches of the bronchioles are the respiratory bronchioles (Figure 13), so called because they are the first sites where gas exchange takes place. Each respiratory bronchiole gives off several branches called alveolar ducts. The alveoli open directly into the duct or pass through an intervening structure, the alveolar sac.

FIGURE 13: TERMINAL DIVISION OF THE BRONCHIOLES

Terminal Bronchiole
Respiratory Bronchiole
Alveolar Duct
Alveolus
Alveolar Sac
ALVEOLUS

The structure separating the air in the alveoli from the blood in the capillaries is called the respiratory membrane. It consists of a thin film of surfactant, a layer of alveolar epithelial cells, a tiny interstitial space, the capillary basement membrane and finally the endothelial cell forming the capillary cell wall.

The total alveolar surface area is approximately 70 m². Carefully arranged, you could park 20 cars in a space this size. It is through the respiratory membrane that gas exchange occurs. Anything that thickens this membrane will impair gas exchange, e.g. interstitial edema often associated with congestive heart failure and interstitial pneumonia.

FIGURE 14: STRUCTURE OF ALVEOLI AND SURROUNDING CAPILLARY NETWORK
Blood returning to the lungs from the tissues has reduced levels of O₂ and increased levels of CO₂ as a consequence of cellular respiration. During passage around the alveolus, excess CO₂ is unloaded and O₂ stores replenished. Oxygen is taken up from the alveolus while carbon dioxide is released into the alveolus (Figure 15).

Oxygen exchange can be markedly impaired by a number of alveolar processes, such as:

- excess alveolar fluid as in pulmonary edema
- alveolar collapse as in atelectasis or a pneumothorax resulting in shunting.
LUNGS

The lungs are cone-shaped organs, which occupy most of the thorax, excepting the mediastinum, which separates them and contains the heart and major vessels. The base of each lung lies in contact with the upper surface of the diaphragm while the apex extends about 2 cm above the level of the clavicle (collar bone).

On the medial surface of each lung is a slit, the hilum, where structures enter and leave the lung. The bronchi, blood vessels, nerves and lymphatic vessels that enter and leave the lung through the hilum, form the root of the lung which constitutes its only firm attachment.

![Diagram of Medial Aspect of the Left Lung]

FIGURE 17: MEDIAL ASPECT OF THE LEFT LUNG

- Apex
- Hilum
- Pulmonary Artery
- Bronchus
- Pulmonary Veins
- Base Diaphragm Surface
- Root
The right lung, which is larger than the left, is divided by horizontal and oblique fissures into superior, middle and inferior lobes (Figure 18). The smaller and narrower left lung is divided by an oblique fissure into superior and inferior lobes. The lingula, a small tongue of lung tissue between the oblique fissure and the cardiac notch, is a part of the upper lobe (Figure 18).

Location of these lobes assumes clinical importance when reporting findings during chest auscultation. One must also understand the normal sounds you expect to find in each area. Auscultate from apices to bases. For confirmation of endotracheal tube (ETT) placement: Auscultate just below the xiphoid (to rule our breath sounds in the stomach from a misplaced ETT in the esophagus), then over the right chest, high mid-axillary line, then over the left chest, high mid-axillary line.
PLEURAE

The pleurae are two serous sacs enclosing the lungs. The pleura that adheres to the surface of the lung (the visceral pleura), is reflected from the root of the lung onto the inner surface of the chest wall, diaphragm and the lateral surface of the mediastinum, to form the parietal pleura (Figure 19). The parietal pleura then has costal, diaphragmatic and mediastinal parts.

FIGURE 19: PLEURAE
PLEURAL CAVITY

The parietal and visceral layers of the pleurae are moist and separated by only a thin layer of serous fluid, perhaps less than 0.02 mm in thickness (Figure 19). Normally the parietal and visceral surfaces are in apposition so that the pleural cavity is a potential space rather than a real space. There is a negative intrapleural pressure within this "space" when it is intact. The adherence of the pleural layers is by a bond similar to that holding two plates of glass together. This pleural bond can be easily destroyed if air (pneumothorax) or fluid accumulates in the pleural space.

PLEURAL RECESSES

In quiet respiration there are certain places where the lungs do not completely fill the pleural cavity. These unfilled areas of the pleural sac are known as pleural recesses. Two of the more obvious and important ones are the right and left costodiaphragmatic recesses, where the periphery of the diaphragm is close to the costal wall. Small pleural effusions will accumulate in these recesses when the patient is erect.
THORAX

The thorax is the bony structure that forms part of an air-tight box around the lungs.

The boundaries of the thorax are:

- Superiorly - thoracic inlet
- Laterally - ribs
- Anteriorly - sternum and ribs
- Posteriorly - ribs, thoracic vertebrae (spine)
- Inferiorly - diaphragm.

The thoracic inlet is the opening that marks the boundary between the neck and the thorax.

FIGURE 21: ANATOMY OF THE THORACIC CAVITY
THORACIC CAVITY

The thoracic (chest) cavity is divided, for descriptive purposes, into three main parts. The right and left pleural cavities and the mediastinum, which lies in the midline.

The pleural cavities contain the lungs and pleurae, while the mediastinum contains important midline structures, e.g. heart, trachea, esophagus.

FIGURE 22: CROSS SECTIONAL VIEW OF THE THORAX
MECHANICS OF VENTILATION

Air, like water, flows from areas of higher pressure to areas of lower pressure. Where there is no pressure gradient, there is no flow. For inspiration to occur the alveolar pressure must be less than the atmospheric pressure at the mouth. There are only two possible means to achieve this pressure gradient:

1. The alveolar pressure can be lowered below atmospheric pressure.

2. Atmospheric pressure can be increased above the normal resting alveolar pressure.

With normal inspiration, contraction of the respiratory muscles results in an enlargement of the thoracic cage and expansion of the lungs. This expansion reduces the pressure within the lungs and a pressure gradient of approximately 3 mm is established between the mouth and the alveoli. The result is a flow of atmospheric air into the lungs.

Consider a closed syringe, on which you pull back on the plunger thereby expanding the volume in the barrel. This is comparable to the expansion of the thorax by inspiratory muscular contraction. One readily recognizes the negative pressure that has been created within the syringe. If you open the syringe end, atmospheric air flows into the barrel, as it would flow from the mouth to alveoli during inspiration.
FIGURE 23: MECHANICS OF VENTILATION

Sternum Moves Outward

Diaphragm Contracts

Sternum Moves Inward

Diaphragm Relaxes

Ribs move outward and upward

Diaphragm contracts
**MUSCLES OF INSPIRATION**

The principle muscle of inspiration is the diaphragm. It is a large dome-shaped sheet of muscle that separates the thoracic and abdominal cavities. In quiet relaxed breathing it may be the only muscle utilized. The diaphragm is anchored about the circumference of the thorax.

Muscular contraction results in a downward movement of the central part, similar to movement of a piston within a cylinder. The main action of the diaphragm is to enlarge the thoracic cavity downward. During maximum ventilation in a healthy individual, its excursion may be as much as ten centimeters. The motor nerves of the diaphragm exit from the spinal cord from the third to fifth cervical vertebrae and run downward as the phrenic nerves.

**ACCESSORY MUSCLES OF INSPIRATION**

The intercostal muscles increase the anterior-posterior diameter of the thorax by moving the anterior end of each rib in an outward and upward motion. As well, their contraction tenses the intercostal spaces and keeps them from being sucked in during inspiration. These muscles are innervated by the intercostal nerves which leave the spinal cord between the first and eleventh thoracic vertebrae, corresponding with muscle position.

---

**Clinical vignette**

You can now readily see that if the spinal cord is transected above C-3, all muscles of ventilation are paralyzed as there is a loss of nervous control. If the cord is injured below C-5, the diaphragm continues to work and some ventilation occurs.

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Other important accessory muscles of inspiration, which come into play when ventilation is laboured, are the **scalenes** and the **sternocleidomastoids**. Contraction of these muscles function to raise the anterior end of the first rib, together with the manubrium and sternum. In addition to increasing the anterior dimension of the upper outlet of the thorax, this also stabilizes the upper thoracic cage so that contraction of the intercostal muscles results in elevation of the remaining ribs.

Maximal contraction of the inspiratory muscles of respiration can lower the intra pleural pressure to as much as 60-100 mmHg below atmospheric.
MUSCLES OF EXPIRATION

Expiration is usually a passive process. Contraction of the inspiratory muscles causes the elastic tissues of the lungs and thorax to be stretched, and thus potential energy is stored in them.Expiration usually occurs as a result of the recoil of the stretched tissues and release of stored energy. Only at very high rates of ventilation (above 40 litres per minute), or with moderately severe airway obstruction (as seen in asthma or emphysema) do the muscles of expiration actively contract.

The abdominal muscles are the most important muscles of expiration. They include:

- External obliques
- Internal obliques
- Rectus abdominus
- Transversus abdominus.

Contraction of these muscles increases intra-abdominal pressure thereby moving the diaphragm upward. Additionally, contraction depresses the lower ribs thereby decreasing the circumference of the thorax.

Contraction of the internal intercostal muscles depresses the ribs, moving them downward and inward. They also stiffen the intercostal spaces so they do not "bulge out" during expiratory efforts such as coughing.

RESPIRATORY REFLEXES

1. Cough

A cough is a violent expiratory blast against a partially closed glottis. The cough reflex is induced by irritation of sensory nerve endings in the larynx, trachea or the larger bronchi.

The actual stimulus may be:

- Inflammatory e.g. infection
- Mechanical e.g. dust, smoke
- Chemical e.g. irritating, noxious gases
- Thermal e.g. cold air.

Once the nerve endings are irritated, impulses are transmitted to the "cough centre" in the medulla. This triggers the following sequence of events:

1. Deep inspiration
2. Tight closing of glottis
3. Expiration against closed glottis
4. Glottis partially opens
5. Expulsion of foreign material.
The closing of the glottis allows for pressure to accumulate within the respiratory passages. The sequential partial opening of the glottis allows for rapid expiratory airflow and expulsion of the foreign substance.

2. **Sneeze**

The sneeze is a defence mechanism against irritant materials in the upper respiratory tract. Irritation of the sensory endings in the nasal mucous membrane results in a deep inspiration which is followed by a violent expiration, with the mouth closed, so that the expiratory blast is discharged through the nose.

**PULMONARY COMPLIANCE**

Healthy lungs contain a great deal of elastic tissue. This must be stretched when the lungs expand. Elastance is defined as that property of matter which allows it to return to its original shape after having been deformed by some external force.

Compliance in pulmonary physiology usually refers to the amount of pressure that must be generated to expand the lungs with a given volume.

We all recognize that some balloons are harder to inflate than others; the same is true of lungs or even the same lung at different times. The more pressure that has to be exerted to inflate the lung, the less its compliance.

Clinically, compliance is of great importance. Decreasing compliance usually indicates increasing disease. Poorly compliant lungs are often referred to as "stiff", e.g. worsening interstitial pneumonia or increasing pulmonary fibrosis.

You will often note changes in compliance when you are manually squeezing a bag to ventilate a patient. If compliance is improving you'll need to exert less pressure to move the same volume of air.

Unfortunately in many clinical scenarios the reverse situation is encountered, e.g. a patient with increasing pulmonary edema will require greater pressure to move the same amount of air.

**SURFACE TENSION**

If one imagines the cells of the alveoli wall, each having a fair amount of iron in their nucleus, and then pictures what would happen if a magnet were placed in the center of this alveolus - the walls would move inward. The closer the walls got to the magnet, the stronger would be the magnetic pull. This is a crude example to illustrate the essential nature of the force known as surface tension.

If one had to overcome the force of surface tension to expand the hundreds of millions of alveoli with each breath, it would require exhaustive muscular effort that could not be sustained over
long periods of time. Fortunately the alveolar cells produce a protein substance known as **pulmonary surfactant**. Surfactant dramatically reduces surface tension and consequently the muscular work required to expand the alveoli.

A striking example of the crucial nature of surfactant is seen in the disease known as Infant Respiratory Distress Syndrome (IRDS). It frequently strikes premature infants who, because of the physical immaturity of the surfactant producing cells, have insufficient surfactant present in their alveoli. As a result, the infant requires strenuous muscular effort to inspire. Treatment of this condition requires maintenance of a positive airway pressure to overcome the surface active forces present, instilling exogenous surfactant into the lungs, and in some instances mechanical ventilation.

**MECHANICAL WORK OF BREATHING**

It can be demonstrated mathematically that there is an optimal tidal volume and respiratory frequency at which the mechanical work of breathing, and jointly the oxygen consumption, is minimal. It has been shown that both normal subjects, as well as those with pre-existing pulmonary disease, breathe at a tidal volume and frequency at which the work of breathing is least.

In the normal individual the respiratory muscles require between 3 - 14 mL of oxygen (<5% of the total body's oxygen consumption).

In severe cardiac or pulmonary pathological states, the oxygen cost of breathing can dramatically increase. For example, an emphysemic patient at rest may utilize 25% of his total oxygen consumption for the mechanical work of breathing.

Additionally cardiopulmonary pathologies will result in a disproportionate increase in oxygen consumption with increases in ventilation.
LUNG VOLUMES AND CAPACITIES

It is important that the paramedic understand a few of the commonly used terms relating to lung volumes and capacities.

1. **Tidal Volume** \((T_V \text{ or } V_T)\) is the measure of the volume of air inspired and exhaled with each breath at rest. In the healthy adult it is usually 400 - 500 mL, however it can be substantially higher.

2. **Residual Volume** is the air remaining after expiration. Even with a maximal expiratory effort, all the air can not be emptied from the lungs. This remaining air is the residual volume.

3. **Functional Residual Capacity** (FRC) is the volume of gas remaining in the lungs at the end of normal tidal exhalation. Note: A Positive End Expiratory Pressure, or P.E.E.P. device, limits exhalation (i.e. increases the FRC) and keeps the alveoli from collapsing.

4. **Total Lung Capacity** is the maximum amount of air the lungs can contain.

5. **Forced Vital Capacity** is the maximum amount of air that can be exhaled after a maximal inspiration.

6. **FeV\(_1\)** (forced expiratory volume in one second). This is a measure of the amount of air that can be exhaled in the first second of a maximal forced exhalation. It is a very useful measure of the degree of obstruction present in asthma, e.g. the lower the FeV\(_1\) the more severe the obstruction.

A reduction in the lung volumes obtained by spirometry suggest the presence of a "restrictive" type of pulmonary disease. This is, consistent with a reduced distensibility of the lungs as seen in pulmonary fibrosis, or a reduced distensibility of the chest wall symptomatic of kyphoscoliosis, and certain neurological diseases.

An increase in the measured lung volumes indicates that the lungs are hyperinflated because of obstruction of the airways (as in asthma), or a loss of lung elasticity (as in emphysema). Consistent with this loss of elasticity is a reduced ability to rapidly exhale the inspired air (decreased flowrates).

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**Clinical vignette**

"Spirometry is the process of measuring volumes of air moving in and out of the lungs. A spirometer is the device used to measure these volumes."
CENTRAL NERVOUS SYSTEM CONTROL OF VENTILATION

Control of ventilation is principally governed by the respiratory center in the brainstem. It responds to increased blood CO₂ levels by increasing ventilation - increased respiratory rate (RR) and/or increased tidal volume. Ventilation decreases when our blood CO₂ level is below normal.

In patients with chronic markedly elevated CO₂ levels, the respiratory center may be inactivated. These people breathe based on their hypoxic drive. Hypoxia is sensed principally by the carotid and aortic arch chemoreceptors. When they are stimulated, the rate and/or volume of respiration is increased.

We also can exert a voluntary control over our respirations, e.g. breath holding.

Some drugs may directly stimulate the respiratory center resulting in hyperventilation, e.g. ASA toxicity. Many other drugs markedly depress our respiratory drive, e.g. narcotic or sedative overdoses.

Metabolic acidosis markedly stimulates our respiratory center resulting in hyperventilation. This lowers the blood’s CO₂ content and will elevate the serum pH towards normal.

A wide variety of CNS insults result in abnormal respiratory patterns varying from apnea to central neurogenic hyperventilation, discussion of which is beyond the scope of this text.
VENTILATION PERFUSION RATIO

Ventilation (V) is defined as the volume of air which moves into or out of the mouth. Minute ventilation (or volume) equals the number of breaths per minute times the volume of each breath (TV).

**EXAMPLE**

<table>
<thead>
<tr>
<th>If:</th>
<th>Respiratory rate</th>
<th>= 12 breaths/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tidal volume</td>
<td>= 500 cc</td>
<td></td>
</tr>
<tr>
<td>Minute ventilation</td>
<td>= 12 x 500 cc</td>
<td>= 6000 cc per minute</td>
</tr>
</tbody>
</table>

Perfusion (Q) is defined as the flow of blood through tissues. At rest the normal cardiac output is approximately 6L/min, i.e. the lungs are perfused with 6L of blood per minute.

The ventilation perfusion ratio, (ventilation/perfusion) is commonly expressed as V/Q or V:Q.

Under ideal conditions:

\[ V = Q \]

Anything that decreases the amount of air entering an area of lung tissue will decrease its V/Q ratio (V/Q < 1), e.g. 5L V/6L Q = .83. If no air can enter a segment of lung then its V/Q = 0 (since 0 divided by any number is still 0) – e.g. foreign body obstruction (FBO) of the airway.

**FIGURE 24: VENTILATION/PERFUSION RATION**

A. \( \frac{V}{Q} = 1 \)  
   Normal ventilation  
   Normal perfusion  

B. \( \frac{V}{Q} < 1 \)  
   ↓ ventilation  
   normal perfusion  

C. \( \frac{V}{Q} = 0 \)  
   Occlusion  
   No ventilation  
   Normal perfusion
A common clinical example of a ventilatory disorder causing V/Q mismatch is in the asthma patient. When totally well, his V/Q = 1 (Figure 24A). As bronchospasm increases, less air can be moved into his alveoli, therefore, V/Q < 1 (Figure 24B). If his condition worsens and some airways become totally occluded (often due to bronchospasm and mucous plugging), no ventilation of these alveoli occurs, therefore, V/Q = 0 (Figure 24C).

Intrapulmonary shunting occurs with any illness that permits blood to flow through the lungs without picking up enough O₂ to fully saturate the hemoglobin. A shunt will result in a decreased arterial oxygen concentration.

Abnormalities of decreased perfusion, e.g. secondary to a large myocardial infarction will result in less blood being circulated around the ventilated alveoli. Because of this, less than normal amounts of oxygen are extracted from the alveoli. One can quickly appreciate that if the cardiac output is reduced by one third, e.g. 4 instead of 6 L/min, then only two-thirds as much oxygenated blood can be delivered to the tissues, despite normal ventilation.

One should now be able to recognize two types of V/Q mismatch leading to tissue hypoxia that will not respond well to increasing the percentage of inspired oxygen. In the circumstance where a large amount of intrapulmonary shunting is occurring and a large volume of poorly oxygenated blood is being mixed with maximally oxygenated blood, the amount of oxygen available for tissue metabolism will still be less than normal (Figure 26).
FIGURE 26: EFFECT OF INCREASING INSPIRED O₂ CONCENTRATION

A. 21% O₂ (room air)

B. 100% O₂
In the theoretical patient illustrated in Figure 26, with a 33% shunt, increasing inspired \( O_2 \) from 21% to 100% only increased hemoglobin saturation by 1.3%.

**HYPOPERFUSION STATES**

Each gram of Hgb can combine with 1.4 mL of \( O_2 \) at 100% saturation. Each Hgb can carry 4 molecules of \( O_2 \). Hgb is 95-98% saturated with \( O_2 \) when the inspired \( O_2 \) concentration is 21% (room air).

A patient, with a Hgb concentration of 150 gm/L and a cardiac output of 6L, has a MI and his cardiac output drops to 4L. Using the formula footnoted below:

- calculate the effect that this decreasing cardiac output will have on oxygen availability for tissue metabolism
- determine if increasing the percentage of inspired \( O_2 \) will correct the problem.

**Total \( O_2 \) available for tissue metabolism:**

1. Prior to MI; cardiac output at 6L, breathing 21% \( O_2 \) \( 6 \times 159 \times 1.4 \times 98\% = 1235 \) mL \( O_2 \)
2. Post MI; cardiac output at 4L, breathing 21% \( O_2 \) \( 4 \times 150 \times 1.4 \times 98\% = 823 \) mL \( O_2 \)
3. Post MI; cardiac output at 4L, breathing 100% \( O_2 \) \( 4 \times 150 \times 1.4 \times 100\% = 840 \) mL \( O_2 \)

It is not the detail of these calculations that is important to the Paramedic, but rather the concept. These calculations allow us to appreciate how little we improved the amount of \( O_2 \) available for tissue metabolism by increasing his inspired \( O_2 \) from 21% to 100%, i.e. from 823 to 840 mL \( O_2 \). Therefore, if we are to increase the total \( O_2 \) available for tissue metabolism significantly, we must correct the underlying problem of decreased cardiac output.

---

*Formula for Calculation*

\[
\text{Total } O_2 \text{ available/minute} = \text{Cardiac} \times \text{Hb} \times \text{mL } O_2/\text{g Hb} \times \% \text{ Saturation} \\
\text{Output} \times \text{Conc. (at 100% Saturation)}
\]
COMMON RESPIRATORY ILLNESSES

ASTHMA

Asthma is defined by the American Thoracic Society as a “chronic inflammatory disease of the airways” and is characterized by increased responsiveness of the trachea and bronchi to various stimuli and manifested by widespread narrowing of the airways that changes in severity either spontaneously or as a result of therapy.

Early in an acute asthmatic attack, the airway narrowing is caused by smooth muscle contraction. As the attack continues, further airway narrowing results from mucosal edema and increased mucous production. This may progress to total obstruction of peripheral airways.

An attack of asthma may be triggered by many stimuli. Some of the more common are allergens, infections, cold temperatures and exercise.

The symptoms early in an acute asthmatic attack consist of dyspnea, wheezing and cough. The physical findings may consist of cough, anxiety, tachycardia, increased respiratory effort including use of accessory muscles, nasal flaring, and inability to lie down. Inspiratory and expiratory wheezing (due to large and small airway constriction) will be heard in the asthma patient that is still ventilating adequately. Prolongation of the time required for inspiration and expiration is usually evident in these patients.
If an attack is allowed to progress and worsen, the severity of the symptoms and signs will increase. However the patient's ability to compensate for increased airway narrowing by increasing respiratory effort is finite. When this is exceeded, respiratory effort decreases; the patient becomes confused (decreased O₂ and increased CO₂); wheezing decreases (the worst asthmatics have silent chests); and unless rapid intervention occurs, respiratory arrest followed by cardiac arrest is imminent.

**Clinical vignette**

The number of words the patient is able to speak between breaths is a good indicator of disease severity. e.g. 2-word dyspnea would likely signify moderate to severe respiratory distress.

**Chronic Obstructive Pulmonary Disease (COPD)**

COPD is generally used to describe patients who have chronic airway obstruction. Chronic is defined as occurring on most days for at least three months in the year, for at least two successive years.

The causes of airway obstruction that fall within the COPD basket are emphysema, bronchitis and asthma.

**Emphysema** is defined as a condition of the lung characterized by abnormal, permanent enlargement of the air spaces distal to the terminal bronchiole, accompanied by destruction of their walls.

**Bronchitis** is defined as chronic or recurrent excess mucous secretion in the bronchial tree, in most instances accompanied by cough.

**Asthma** is defined as a chronic inflammatory disease of the airways with hyperreactivity of the trachea and bronchi to various stimuli and manifested by widespread narrowing of the airways that changes in severity either spontaneously or as a result of therapy.

Most patients with COPD have a combination of all three of these illnesses, although the fraction that each contributes to the respiratory obstruction varies from patient to patient, e.g. patient A's obstruction may be predominantly due to bronchitis and bronchospasm with no emphysematous component, while in patient B the obstruction may be due to emphysema and bronchitis with no bronchospasm. Many other combinations of the three illnesses are possible.

As the combination of the illnesses leading to airway obstruction varies, so do the symptoms and signs and frequency of exacerbations.

In order for you to assess how much of each illness is responsible for your individual patient's respiratory obstruction, you must understand the presentation of each illness in its pure form. However, you must also realize that in most COPD patients, respiratory obstruction is due to a combination of two or three of these illnesses.
EMPHYSEMA

Patients with emphysema have had significant destruction of their alveolar septa (walls) and obliteration of the pulmonary vascular bed. The alveolar septa are the springs that keep the bronchi open. Without their support, the bronchioles collapse.

This collapse of airways leads to two problems:

- Increased air flow obstruction
- Overinflation of the air sacs.

The classic picture of patient whose COPD is caused only by emphysema is that of the pink puffer. These patients are thin, anxious, alert, oriented, dyspneic, tachypneic and hyperventilating. Dyspnea is the hallmark.

Accessory muscles of breathing are used in the fight for breath. Pursed lips on expiration increases airway pressure, thereby internally splinting the airway. These patients have minimal respiratory reserve and are largely sedentary. Due to the chronic over distension of their airways, these patients have increased A/P chest diameters and low immobile diaphragms. They usually have normal colour (pink) and near normal arterial blood gases.

Unfortunately, due to their poor respiratory reserve, many patients with severe emphysema can not tolerate any increased respiratory insult. A pneumonia or even a single fractured rib may tip
them into respiratory failure. Excessive coughing in emphysems can also lead to barotrauma such as a pneumothorax. Markedly diminished or absent air entry in a dyspneic emphysemic may signal a tension pneumothorax and is an ominous sign!

Unfortunately there is no therapy short of lung transplantation to correct emphysema.

**BRONCHITIS**

Patients whose COPD is due solely to chronic bronchitis are often referred to as "blue bloaters". This refers to chronic central cyanosis due to the shunting (V/Q mismatch) that occurs in the lung, resulting in ↓ O₂ saturation. This person is relatively stocky or obese and has central and peripheral cyanosis. They have less of an increase in their A/P chest diameter than the emphysematous patient, and their diaphragms are not abnormally low. They usually have a chronic cough, often productive of sputum.

These patients may deteriorate suddenly (over a few minutes) and if they do, either a pneumothorax, pulmonary embolism or atelectasis is usually the cause. The atelectasis is caused by secretions obstructing a major airways.

Increasing secretion production causes a more gradual deterioration. This is usually due to an "allergic cause" or infection. Pneumonias can be devastating in these patients. One must be even more wary of rib fractures in the "blue bloater" than the "pink puffer". If these people don't cough vigorously they don't clear their secretions and substantial airway collapse may occur.

We have already discussed the third illness in the triad causing COPD, i.e. asthma.
MANIFESTATIONS OF RESPIRATORY DISEASE

SYMPTOMS AND SIGNS

EXCESSIVE NASAL SECRETIONS

This may be the result of irritation of the mucosa of the upper respiratory tract due to infection, allergens, or mechanical or chemical sources.

SNEEZING

Forced exhalation of air through the nose is an attempt to remove nasal mucosal irritants.

COUGHING

Coughing is a defense mechanism of the lower respiratory tract, functioning to clear airways of irritants. The initiating stimulus may be:

- inflammatory, e.g. infection, asthma
- mechanical, e.g. dust, aspirated material
- chemical, e.g. irritating noxious gases, smoke, Cl2
- thermal, e.g. cold air.

Coughing may become chronic in nature, e.g. bronchitis.

EXPECTORATION OF SPUTUM OR BLOOD

It is important to determine colour, volume, and consistency of sputum. For example:

- duration of sputum production will indicate an acute or chronic disease process
- mucoid, sticky, grey or white sputum may be normal for persons with COPD, e.g. chronic bronchitis
- yellow or green sputum may indicate infection
- blood may be mixed with sputum in varying degrees or it may comprise the entire expectorate.

CHEST PAIN

Determining the exact cause of chest pain is often difficult. One should know the possible sources of chest pain and the usual type of pain produced by each. Sources of chest pain are:

CHEST WALL

- Skin, e.g. burns - this is constant, mild to excruciating in nature
- Ribs, e.g. fractures - usually associated with trauma and increased by anything that moves the rib(s), e.g. breathing/movement/palpation, moderate to severe in nature
- Costal cartilages, *e.g.* costochondritis - similar to pain of rib fracture but less severe
- Muscles, *e.g.* muscle sprain - similar to pain of rib fractures, especially sore when using involved muscles, mild to moderate in severity
- Nerves, *e.g.* shingles (Herpes Zoster) - constant boring, burning pain, maximal in the involved dermatome, mild to moderate in severity.

**PLEURA**

- Pleuritic pain is stabbing, knifelike, aggravated by breathing, movement and coughing. This pain is usually moderate to excruciating. Common causes of pleural irritation are infections, *e.g.* pleurisy and pneumonia, or pulmonary infarctions.

**PERICARDIAL**

- This pain is often similar to pleural pain in nature and often worsened by breathing. It is commonly decreased when leaning forward. Pericardial inflammation is usually due to pericardial infection or subsequent to myocardial infarction. Moderate to severe.

**DIAPHRAGM**

- Pain similar in character and severity to pleural pain is due to diaphragmatic irritation from above, *e.g.* lower lobe pneumonia or pulmonary infarction, or below, *e.g.* free peritoneal blood or air. This is often associated with referred pain to one or both shoulders.

**ESOPHAGEAL IRRITATION**

- This pain can vary from the burning associated with heartburn to pain indistinguishable from that of myocardial infarction, mild to severe in nature.

**CARDIAC ISCHEMIA**

- This is extensively described elsewhere. Classically described as tightness, heaviness, pressure, weight originating from center of chest. It may radiate to arms and/or neck and jaw and occasionally to the back. This pain is not increased with chest movement or breathing. It may be associated with sweating, nausea and/or vomiting.

**AORTIC DISSECTION**

- Sudden onset of severe centralized chest pain is the most common complaint. The pain often begins in the anterior chest and may "migrate" to the interscapular region and/or down the back. In fact, migrating pain is highly specific to thoracic aortic dissection. It may also be described as a "ripping" or "tearing". One article reported that 71% of patients had migration of pain with their dissections.¹

  It is often associated with sweating, nausea and vomiting. A difference in systolic blood pressures between the right and left arm of ≥15 mmHg may suggest the aortic dissection has taken a path along one of the subclavian arteries. Hypotension is an ominous sign.
ESOPHAGEAL RUPTURE

- This is a rare occurrence and usually occurs as the result of an instrument being introduced into the esophagus for medical purposes. Signs & symptoms include severe central chest and/or epigastric pain that is usually preceded by a sudden increase in intrathoracic pressure, **e.g. of other causes**: vomiting, violent coughing, weight lifting **e.g. of instrumentation**: insertion of esophageal/airway device, gastric lavage, esophagoscopy. The pain is often pleuritic in nature but may be constant. It may radiate to the back and is often increased by swallowing or neck movement.

- You will already have noted that the pain associated with chest wall, pleural, pericardial and diaphragmatic hernia or esophageal rupture share common features. Differentiation is often very difficult.
DYSPNEA

Dyspnea is defined as a sensation of difficulty breathing. This may be normal or abnormal, e.g. the dyspnea associated with bronchospasm, COPD, chest wall injury or foreign body obstruction. The causes of abnormal dyspnea may be:

- Physical, causes of which include anything that increases the body’s O₂ consumption or decreases its ability to deliver O₂ to the tissues.

<table>
<thead>
<tr>
<th>Normal</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-psychogenic</td>
<td>* Psychogenic: e.g. hyperventilation</td>
</tr>
<tr>
<td>Cardiac. e.g. Left ventricular failure</td>
<td>Pulmonary</td>
</tr>
<tr>
<td>Haematologic. e.g. Severe anemia</td>
<td>Metabolic. e.g. hyperthyroidism, CO poisoning</td>
</tr>
<tr>
<td>Obstructive e.g. asthma</td>
<td>Restrictive e.g. pulmonary fibrosis</td>
</tr>
</tbody>
</table>
SHORTNESS OF BREATH (SOB)

Conversely, shortness of breath is the subjective sensation of not being able to catch one’s breath. e.g. as might be experience in the setting of hypovolemia where oxygenation is inadequate due to hypoperfusion and decreased oxygen carrying capacity from lost red blood cells. Or, the SOB experienced by some in the setting of acute myocardial infarction where cardiac output, and consequently tissue perfusion and oxygenation is impaired. In the healthy state, SOB may occur with exercise.

HYPOXIA

Hypoxia is defined as a lack of sufficient O₂ to allow proper functioning of the brain and other vital organs and tissues. Hypoxia can result from:

- Respiratory causes, e.g. severe pulmonary edema
- Cardiovascular causes, e.g. cardiogenic shock or hypovolemia
- Haematological causes, e.g. severe anemia or CO poisoning.

As the definition implies, CNS signs are often prominent in hypoxia. These may range from anxiety, combativeness, visual disturbances, in-coordination and dysarthria to confusion or coma and even seizures. Other signs of hypoxia may include cyanosis, cardiac arrhythmias and signs of sympathetic stimulation such as tachycardia and diaphoresis.

Hypoxia is a major stimulant of respiration when the PaO₂ falls to below 60 mmHg (normal PaO₂ is 80-100 mmHg). However, in severe pre-terminal hypoxia, spontaneous respiration may be absent. Correction of hypoxia takes highest precedence in the treatment of any patient.

CYANOSIS

Cyanosis is defined as a diffuse bluish discolouration of the skin and mucous membranes, secondary to an increased amount of deoxygenated (reduced) hemoglobin in the bloodstream. Approximately 5 g deoxygenated hemoglobin per 100 ml of blood, or the presence of abnormal hemoglobin (methaemoglobin or sulfhaemoglobin), is required to produce cyanosis in normal persons. Polycythemic patients (those with abnormally high red blood cell count [hemoglobin]) may exhibit cyanosis at lower levels.

Cyanosis is usually a late sign of hypoxia.

HYPERCAPNIA

An increased blood level of carbon dioxide (PaCO₂) results when ventilation is insufficient to remove the carbon dioxide produced. Most hypercarbic patients are hypoventilating. The common causes are:

- decreased central respiratory drive, e.g. narcotic OD
- spinal cord transection
- Neuromuscular diseases, e.g. myesthenia gravis.
- COPD (chronic CO₂ retainers)
Hypercarbia can, however, occur in the presence of an increased respiratory rate and effort, e.g. severe asthma. These patients are also in great danger as despite maximal ventilatory effort, they cannot compensate for their respiratory disease.

Hypercarbic patients may complain of a headache (increased PaCO₂ produces cerebral vasodilation) and if the CO₂ level continues to rise, the patient may become confused, somnolent or comatose.

References:

ADVANCED LIFE SUPPORT
PRE COURSE
RESPIRATORY SYSTEM

SELF-ASSESSMENT

MARKS

[2] 1. You have unsuccessfully attempted a nasotracheal intubation and now have a substantial epistaxis from the same nostril. What two general sites are the most likely to have been injured?

[2] 2. You are attempting a blind nasotracheal intubation on a different patient (without the use of a laryngoscope). This patient has presented with hypoventilation secondary to a drug overdose. The tube advances without difficulty but does not enter the trachea.

[1] a) What is the most likely anatomic location of the distal end of the tube at the end of the unsuccessful pass?

[2] b) Your intubation attempt has stimulated the patient's rate and depth of ventilation. Without using any instruments or tools, how would you know that the tube is not in the trachea?

[2] 3. List in order of anatomical position the following structures, starting with the most anterior:

   a) esophagus
   b) vertebral body
   c) larynx
   d) spinal cord

[3] 4. At the cephalic end of the trachea is a) ____________________________.
The trachea terminates at the bifurcation into the mainstem bronchi. This area is called b) __________________. The trachea is usually c) __________________ _______ in length.

5. On arrival at hospital, the emergency physician tells you that your patient's endotracheal tube was in a mainstem bronchus.


[2] b) Name the most likely mainstem bronchus involved.

[2] c) What is the anatomical reason for this position?

[1] d) This same patient exhibited enroute to hospitals an increasing degree of cyanosis and an increased heart rate. The most likely cause is ________.

[2] e) You have increased the oxygen concentration administered to this patient in response to d). Will the patient respond positively to this? (Justify your answer in 10 words or less.)
6. You are called to the scene of a karate tournament where a combatant is having a great deal of difficulty breathing following a blow to the anterior neck.

[1] a) Identify the vulnerable structure most likely involved.

[2] b) Identify the risk to the patient and the mechanism by which it occurs.

7. Anatomically orient the following structures starting with the most cephalad:
   - Thyroid cartilage
   - Hyoid bone
   - Tracheal rings
   - Cricoid cartilage
   - Mandible
   - Cricothyroid ligament

[2] 8. a) Why do very sick asthmatics often have very little wheezing?

[1] b) What causes the wheezing in asthma?

[1] a) The potential for airway obstruction (exists/does not exist) _______________

[2] b) Give the anatomical reason for your answer.

10. Name the three pathologies involved in COPD.

11. You are ventilating an intubated patient with asthma and COPD. You are noticing that you have to use increasing force to move the air. Using medical terminology (two words) state what is happening.

12. You notice that the nasal secretions of a patient who has suffered a severe head injury are seen to consist of dilute blood. When this falls on the sheet it is seen to form a dark red inner circle and a pink outer circle.

[1] a) What is the likely anatomic site of injury?

[1] b) Name the secretion.
c) What are the implications in terms of choice of airway protection?

13. You have a patient with a C-3 cord transection. Describe the nature of this patient's respiratory effort.

14. a) Define tidal volume.

b) What is the normal adult range?

40 TOTAL SCORE
ADVANCED LIFE SUPPORT
PRECURSE
RESPIRATORY SYSTEM

SELF-ASSESSMENT ANSWERS

1. Nasal septum
   Turbinates

2. a) Esophagus
   b) No air movement would be felt or heard.

3. Larynx Esophagus Vertebral body Spinal cord

4. a) cricoid cartilage
   b) the carina
   c) 10 cm

5. a) This is not desirable since air will only enter one lung. The result will be a hypoxic patient.
   b) Right
   c) The right mainstem bronchus is almost in a straight line with the distal end of the trachea, while the left mainstem bronchus takes a much sharper turn.
   d) Hypoxia
   e) Anticipate no response. You have created a large shunt.

6. a) Larynx
   b) Airway obstruction due to mucosal swelling, or mechanical obstruction.

7. Mandible
   Hyoid bone
   Thyroid cartilage
   Cricothyroid ligament
   Cricoid cartilage
   Tracheal rings

8. a) The production of sound is dependent upon adequate ventilation. Decreased ventilation, movement of air results in decreased wheezing. Decreased movement of air results from bronchoconstriction, mucosal edema and increased mucous production which causes obstruction of flow.
   b) Airway constriction.
9.  
   a) Exists
   b) Tracheal rings are horseshoe-shaped, open at the posterior aspect. A large object in the esophagus could then protrude into the posterior aspect of the trachea.

10. 
    Asthma
    Bronchitis
    Emphysema

11. Decreasing compliance.

12. 
    a) Cribiform plate (with or without basal skull fracture).
    b) CSF
    c) Anything put into the patient's nose is done with the risk of penetrating the cranium via the cribiform plate. In this instance if the oral route of airway support is possible, it is preferable.

13. None. Ventilatory support is required.

14. 
    a) Volume of air inspired and expired at rest.
    b) 400-500 mL
ADVANCED LIFE SUPPORT
PRE COURSE
THE RESPIRATORY SYSTEM

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.
   
   Reading ___________________ hours
   Self assessment ___________ hours
   Total time _____________ hours

2. Were the objectives of the module clearly stated?
   
   [ ] yes  [ ] no
   If no, please comment.

3. Did you see any of the resource materials?
   
   [ ] yes  [ ] no
   If yes, which items
   __________________________________________________________
   __________________________________________________________
   __________________________________________________________
   Were they helpful?
   __________________________________________________________

4. Were the reference notes adequate?
   
   [ ] yes  [ ] no
   If no, please comment.
5. Were the reference notes easy to follow?

[ ] yes [ ] no
If no, please comment.

6. Were the examples provided satisfactory?

[ ] yes [ ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[ ] yes [ ] no
If yes, please specify.

1. Was the level of the module satisfactory for your program of study?

[ ] yes [ ] no
If no, please comment.

Base Hospital

9. General comments or suggested improvements.
Paramedic Resource Manual

OXYGEN DELIVERY
SECTION TWO

2005 Update by
Ontario Base Hospital Group Education Subcommittee

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OBJECTIVES: OXYGEN DELIVERY

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. briefly explain the principle of partial pressures of gases.
2. describe the factors affecting oxygen and carbon dioxide transport and release in the body.
3. briefly describe the factors affecting oxygen dissociation.
4. define hypoxia and briefly explain the difference between the two main types of hypoxia.
5. list the causes of hypoxia and give examples.
6. briefly describe the chemical control of ventilation.
7. explain the reason for the use of non-rebreather masks and nasal cannulae in the A.L.S. system.
8. Identify and explain the function of an aerosol mask and nebulizer.
9. State the flow rates and oxygen concentration achieved with:
   a) Nasal cannulae
   b) Non-rebreather mask
   c) Aerosol mask
   d) Bag-valve-mask.
10. compare and contrast the bag-valve-mask with the pressure-driven (powered) system.
11. state the reasons for using humidified oxygen.
12. Accurately calculate the duration of an oxygen cylinder, given the flow rate, cylinder size and gauge pressure.
13. Apply the information within the above objectives to clinical situations.

If you have studied this subject previously, you may test your ability using the self-assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.
INTRODUCTION

The unit on the respiratory system examines both the anatomical and physiological aspects of the respiratory system important in pulmonary mechanics and ventilation. This unit is intended to be a continuation of the discussion on the respiratory system focusing on the principles of gas transport, factors affecting transport, the chemical control of the respiration and the practical application and usage of oxygen delivery systems.

A review of the respiratory system, as well as a brief review of acid-base balance, is suggested before attempting this unit.

PARTIAL PRESSURES

PRINCIPLE

Gas modules are in fluid motion all around us. The earth’s atmosphere is made up of many different gases, each one comprising a certain percentage of the total amount.

Like other molecules, gases have weight and create a downward force as a result of the earth’s gravity. The total downward force of these gases is known as atmospheric pressure.

At sea level, this downward pressure is sufficient to support a column of mercury (Hg) 760 millimeters (mm) high. Therefore, 1 Atmosphere is equal to 760 mmHg.

Gases are also measured in “torr” units. One torr unit equals one mmHg. Therefore:

\[ 1 \text{ Atmosphere} = 760 \text{ mmHg} = 760 \text{ torr} \]

It is often important to calculate the pressure of a single gas of the mixture. This value is known as the PARTIAL PRESSURE (often called the TENSION) of that gas. The partial pressure of any gas is the pressure which it would exert if it were alone and unaffected by changes in other gases.

There are a number of gas laws which help summarize the behaviour of gases. Relevant to this discussion is Dalton’s Law which states: “The total pressure of a gas mixture is equal to the sum of the partial pressures of the component gases”.

CALCULATION OF PARTIAL PRESSURES

To calculate the partial pressure ($P$) of a particular gas, multiply the total pressure ($P_T$) of all the gases times the fraction of composition of the gas you are trying to find.

EXAMPLE 1:

At sea level (1 atm) the total gas pressure ($P_T$) is 760 mmHg.

Oxygen is approximately 20.93% of the total atmospheric composition.

Therefore, the partial pressure of $O_2$ ($P_{O_2}$) is:

$$760 \times 0.2093 = 159.1 \text{ mmHg}$$

159.1 mmHg = the approximate $P_{O_2}$ in atmospheric air

EXAMPLE 2:

The $P_T$ of the two gases in the box equals 760 mmHg.

Gas (a) equals 2/10 or 20% of the PT.  
Gas (b) equals 8/10 or 80% of the PT.

Therefore, the partial pressure of gas (a) is:

$$0.20 \times 760 = 152 \text{ mmHg}$$

the partial pressure of gas (b) is:

$$0.80 \times 760 = 608 \text{ mmHg}$$
COMPOSITION OF AIR

The understanding of partial pressures, as they relate to respiratory physiology, requires a comparison of air composition between the atmospheric and the alveolar air.

<table>
<thead>
<tr>
<th>SPECIFIC GASES</th>
<th>DRY ATMOSPHERE AIR (%) (partial pressure)</th>
<th>ALVEOLAR AIR (%) (partial pressure)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrogen (N₂)</td>
<td>79.03 600.60 mmHg</td>
<td>74.9 569.24 mmHg</td>
</tr>
<tr>
<td>Oxygen (O₂)</td>
<td>20.93 159.10 mmHg</td>
<td>13.6 103.36 mmHg</td>
</tr>
<tr>
<td>Carbon Dioxide (CO₂)</td>
<td>0.04 .30 mmHg</td>
<td>5.3 40.28 mmHg</td>
</tr>
<tr>
<td>Water (H₂O)</td>
<td>-</td>
<td>6.2 47.12 mmHg</td>
</tr>
<tr>
<td>Total</td>
<td>100.00 760.00 mmHg</td>
<td>100.0 760.0 mmHg</td>
</tr>
</tbody>
</table>

From Table 1, it can be seen that the total pressure of the atmospheric air and the alveolar air is the same. However, when comparing the two, it is important to note the difference in the percentage and partial pressure of each of the component gases.

Above any solution is the vapour of the solution (solvent) itself. This is known as the VAPOUR PRESSURE. Under equilibrium conditions, the partial pressure of a gas in a liquid is equal to the partial pressure of the gas above the liquid. The vapour pressure of water at 37°C is approximately 47 mmHg. The airways of the lungs, including the alveoli, are fully saturated with water vapour, i.e. 100% relative humidity. This means a partial pressure of water vapour within these airways equaling approximately 47 mmHg. This water vapour pressure must appear as part of the total gas pressure and this is reflected by a decrease in the partial pressures of the other gases within the alveoli.

Another gas law which may help to explain the relationship of partial pressures above and within a solution is Henry’s Law which states: “The quantity of a gas that dissolves in a volume of liquid is directly proportional to the partial pressure of that gas, the pressure remaining constant”.

DIFFUSION

It is within the alveoli that gas exchanged takes place. The exchange of gases between the alveoli and the venous blood returning to the lungs is a result of the gases diffusing across the alveolar and capillary membranes.

The ability of a gas to diffuse across these membranes and either into or out of the blood is dependent upon five factors. Certain pathologies such as COPD, pulmonary edema, tumors, fibrosis, etc. can all affect the efficiency of the gas transfer.
The factors affecting diffusion are:

1. The solubility of the gas in the fluid.
2. The concentration or pressure gradient.
3. The amount of surface area available.
4. The thickness of the membrane.
5. The temperature of the fluid.

Gases diffuse from an area of high concentration (pressure) to an area of low concentration (pressure) until an equilibrium is attained. In this way, essential gases move into and out of the blood via the lungs.

![FIGURE 1: DIFFUSION OF GASES](image)

When examining the partial pressure of gases within the blood, it can be seen (Figure 2) that the partial pressure of oxygen within the venous blood ($P_{VO_2}$) is about 40 mmHg. As the blood enters the pulmonary capillaries, via the heart and pulmonary arteries, it will come into contact with the alveolar air containing a $PO_2$ of approximately 100 mmHg. The concentration difference between the two causes the oxygen to diffuse from the alveolus to the blood until an equilibrium is reached. Blood now leaving the lungs, via the pulmonary veins, will be pumped by the heart into the arterial system.
The PO$_2$ of the newly oxygenated blood will be very close to that of the alveolus. However, there is a slight reduction due to the normal physiologic shunt (see Respiratory unit). Lung damage or disease may cause a dramatic increase in the amount of blood shunted through the lungs. This would cause a further lowering of the partial pressure of oxygen within the arterial blood (PaO$_2$).

Oxygenated blood is taken to the tissues, via the arterial and capillary systems and is exchanged as a direct result of the gas pressure differences. The PO$_2$ within the tissues can be extremely variable and is dependent upon the metabolic activity. Average PO$_2$ for tissues is considered to be approximately 40 mmHg, however, this could be considerably lower in very active tissues.

Carbon dioxide (CO$_2$) is a by-product of cell metabolism. The CO$_2$ produced by the cell diffuses into the venous blood giving it a PCO$_2$ of about 46 mmHg. As this blood comes in contact with the alveolar air (having a PCO$_2$ of 40 mmHg), there is a net diffusion of carbon dioxide out of the blood and into the lungs.
GAS TRANSPORT

OXYGEN TRANSPORT TO THE TISSUES

At the normal partial pressure of 100 mmHg, oxygen is relatively insoluble in plasma. Only about 0.3 mL of oxygen dissolves in 100 mL of plasma. The small amount of oxygen that is dissolved is totally inadequate to supply the demand by the tissues.

There are two factors which determine the quantity of oxygen delivered to the tissues. These are the:

- Blood flow (perfusion)
- Concentration of hemoglobin and the affinity of oxygen for it (oxygenation).

Actual blood flow is determined by the integrity of the cardiovascular system. Various influences upon the cardiovascular system which affect cardiac output and the degree of vasoconstriction all affect blood flow.

Hemoglobin is the red pigment found within the red blood cells (erythrocytes). As a result of its chemical configuration, hemoglobin has a strong affinity for oxygen and is the principal carrier in the blood. As stated above, only about 0.3 mL of oxygen is physically dissolved in 100 mL of blood. By contrast, hemoglobin will combine with 19-20 mL of oxygen per 100 mL of blood (usually expressed as volumes percent). This oxygen bound to hemoglobin accounts for approximately 97-98% of the total O2 carried, when the PO2 is 100 mmHg.

Hemoglobin is a complex molecule consisting of heme and globin portions. In each heme portion there are four atoms of iron, each capable of attaching to a molecule of oxygen. When oxygen is attached to deoxygenated hemoglobin (Hb), it becomes oxyhemoglobin (HbO2). Oxyhemoglobin is formed in the alveolar capillary beds due to a high PO2 and a decreased PCO2. The mechanism by which oxygen is released from hemoglobin for diffusion to the tissues is discussed in greater detail under the “Bohr Effect” on page 67. Figures 3 and 4 illustrate the transport of oxygen.

**FIGURE 3: ASSOCIATION/DISSOCIATION OF O2 AND HbO2**

<table>
<thead>
<tr>
<th>IN LUNGS</th>
<th>Hb → increasing PO2 → HbO2</th>
</tr>
</thead>
<tbody>
<tr>
<td>O2</td>
<td>+</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>IN TISSUE</th>
<th>HbO2 → Decreasing PO2 → Hb + O2</th>
</tr>
</thead>
</table>

Oxygen is transported to the tissues through the bloodstream. In the lungs, oxygen diffuses into the blood and combines with hemoglobin, forming oxyhemoglobin (HbO2). This complex is transported to the tissues where oxygen is released due to a decrease in partial pressure, allowing it to diffuse into the cells.
**OXYGEN DISSOCIATION**

The way in which oxygen is taken up and given off can be seen graphically using an oxygen-hemoglobin dissociation curve. The resulting S shaped curve will show the percentage of saturated hemoglobin (left vertical axis) at varying partial pressures of oxygen (horizontal axis). Examples of this curve are shown in Figure 5.

At maximal saturation, each gram of hemoglobin has an oxygen carrying capacity of 1.34 mL/100 mL of blood ($PO_2 = 760$ mmHg), or 4 $O_2$ molecules per hemoglobin. The average adult has between 14-16 gm of hemoglobin for every 100 mL of blood.

**Venous** blood has a $PO_2$ of 40 mmHg at rest. This means that 75% of the hemoglobin is saturated in "deoxygenated" blood.

---

**For Interest Only**

Of a 4.6 vol % of $O_2$ used by the tissues, about 4.4% was released from the hemoglobin and the further 0.2% came from the dissolved $O_2$ in the plasma. With the tissues using such a small percent of the total available $O_2$, it can be seen that there is a large reserve available for increased tissue demands, and that very active conditions can cause the $PvO_2$ to be as low as 10-20 vol %.

---

**FACTORS AFFECTING AFFINITY OF OXYGEN FOR HEMOGLOBIN**

The three major factors which affect the affinity of oxygen for hemoglobin are $pH$ (blood acidity), $PCO_2$ and temperature. The oxygen-hemoglobin dissociation curve (Figure 5) is affected by any one of these factors. The curve will shift either:

- Downward and to the right
- Upward and to the left.

An increase in hydrogen ion concentration (lowering the $pH$) causes the blood to be more acidic which causes the curve to shift downward and to the right. When the curve shifts downward and to the right, as in an acidicotic state, $O_2$ doesn’t bond as easily or as strongly at the level of the lungs, however $O_2$ is more readily released to the tissue levels. Increases in temperature also have a similar effect on the curve.

---

**Clinical vignette**

Even though hemoglobin’s affinity for $O_2$ may be diminished in an acidotic state, we can help to compensate for that by providing supplemental $O_2$. Hyperventilation may also be indicated in cases of respiratory acidosis, as blowing off $CO_2$ will cause an increase in the blood $pH$ (every $\downarrow$ in $CO_2$ of 10 mmHg = $\uparrow$ in $pH$ or 0.08).
Conversely, a decrease in hydrogen ion concentration (increase in pH or alkalosis), a reduction in PCO₂, or lowering of the temperature will cause the curve to shift upward and to the left. This causes oxygen bind more readily and more tightly to hemoglobin at the level of the lungs, however, O₂ is not as readily released from hemoglobin at the tissue level.

**Clinical vignette**

Hyperventilation (blowing off CO₂) may actually impair oxygenation at the tissue level as O₂ becomes too tightly bound to hemoglobin. Hence providing O₂ to a patient who is hyperventilating is not only indicated, but critically important for increasing the amount of dissolved O₂ in blood plasma to make it available at the tissue level.

**FIGURE 5: EFFECT OF CO₂, PH AND TEMPERATURE ON OXYGEN-HEMOGLOBIN DISSOCIATION**

Nature has provided us with a protective mechanism when it comes to oxygen transport. As seen by the flatness at the top of the curve, slight natural variances in alveolar PO₂ will not affect, to any significant degree, the amount of oxygen carried by the hemoglobin.
CARBON DIOXIDE TRANSPORT

Carbon dioxide is a byproduct of normal aerobic cellular metabolism. Under resting conditions, each 100 mL of blood gives up 4-5 mL of CO$_2$ in the lungs. Carbon dioxide is very acidic and is transported by the blood until it can be eliminated from the body by either the lungs or excreted by kidneys. Inability of the body to excrete CO$_2$ would result in the blood becoming too acidic to sustain life. Carbon dioxide is transported in the blood in three ways. These are:

- Carried in the form of bicarbonate
- Combined with hemoglobin (carbaminohemoglobin)
- Dissolved in plasma.

Although carbon dioxide is almost 20-fold more soluble than oxygen in plasma, only 7-10% is carried in this form. A larger amount (23-25%) diffuses into the red blood cell and combines with hemoglobin (Hb) to form carbaminohemoglobin (HbCO$_2$).

\[ \text{Hb} + \text{CO}_2 \rightarrow \text{HbCO}_2 \]

The largest amount of carbon dioxide (65-70%) is carried in the form of bicarbonate (HCO$_3^-$). This reaction occurs quite slowly in plasma but upon entering the red blood cell the reaction is increased almost 1000-fold by the assistance of the enzyme carbonic anhydrase.

\[
\begin{align*}
\text{carbonic} \\
\text{CO}_2 + \text{H}_2\text{O} & \rightarrow \text{H}_2\text{CO}_3 \\
\text{anhydrase}
\end{align*}
\]

Carbon dioxide combines with water to form carbonic acid. The carbonic acid (H$_2$CO$_3$) then dissociates into a hydrogen ion (H$^+$) and a bicarbonate ion (HCO$_3^-$).

\[ \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{HCO}_3^- \]

The free hydrogen ions produced by this reaction are buffered primarily by the deoxyhemoglobin. The bicarbonate ions formed diffuse into the plasma. As the bicarbonate ions move out of the cell chloride ions (Cl$^-$) move into the cell in a 1:1 relationship. This phenomenon is known as the CHLORIDE SHIFT. It occurs so that electrochemical neutrality is maintained within the cell. In the lungs this chemical reaction reverses as CO$_2$ is expelled.
BOHR EFFECT

The Bohr effect describes the changes in the affinity of oxygen to bind to hemoglobin as a result of the shift in blood pH that occurs on a breath by breath basis. Changes in hemoglobin to oxygen affinity occur at both the level of the lungs and the tissues.

Think of hemoglobin as a MAGNET that’s affected by blood pH.

- when we exhale, we blow off CO₂ and a shift of the blood’s pH toward the alkaline side occurs
- when the blood is more alkaline, hemoglobin has a greater affinity (stronger magnet) for O₂ and thus O₂ is drawn toward hemoglobin at the alveolar/capillary level.
- when the blood reaches the tissue level, CO₂, a by-product of cellular metabolism, diffuses from the tissue to the capillary blood
- this shifts the blood pH toward the acidic side which weakens hemoglobin’s hold on oxygen (weaker magnet)
- blood (hemoglobin) that shifts toward the acidic side of pH gives up O₂ readily to the tissues

The Bohr effect occurs on a breath by breath basis, facilitating the binding and releasing of oxygen from hemoglobin.

SUMMARY

ALKALINE STATE
- hemoglobin becomes a stronger magnet, drawing O₂ toward it
- if the blood remained alkaline at the tissue level, it would not release O₂ to the tissues readily (e.g. hyperventilation makes the blood alkaline, as does administering sodium bicarbonate, excessive vomiting, etc)

ACIDOTIC STATE
- when the blood is acidic, hemoglobin becomes a weak magnet and does not pick up O₂ as readily
- we attempt to compensate for this by providing the patient with supplemental oxygen which increases the amount of O₂ dissolved in blood plasma for transport.
- if blood is acidic at the tissue level, O₂ bound to hemoglobin is released to the tissue easily.
HYPOXEMIA

Hypoxemia is identified by a blood gas analysis with a partial pressure of oxygen in the arterial blood lower than normal (<80 mmHg) and usually less than 90% oxygen saturation.

HYPOXIA

The term hypoxia can be generally defined as a state of oxygen deficiency or lack of oxygen. This reduced or insufficient oxygen supply to the tissues can cause impairment of bodily functions which may become irreversible if allowed to go unmanaged.

There are four types of hypoxia, each of these having a number of possible causes:

- Hypoxic Hypoxia
- Hypemic Hypoxia
- Stagnant Hypoxia
- Histotoxic Hypoxia.

A patient with oxygen deficiency may be suffering from a single cause or any combination of causes from one or more types of hypoxia.

- **Hypoxic Hypoxia**: Breathing air or a gas which contains a lower than normal PO$_2$, *e.g.* high altitudes, rebreathing in a closed space.

- Decrease in pulmonary ventilation, *e.g.* pneumothorax, partial airway obstruction, drug induced respiratory depression.

- Abnormal lung function, *e.g.* asthma, fibrotic disease, fluid filled alveoli as with pulmonary edema, pneumonia, hemorrhage, drowning.

- Arteriovenous shunting, *e.g.* some congenital heart defects allow for mixing of arterial and venous blood.

- **Hypemic Hypoxia**: Reduced or altered Hb. In this case, blood does not have a normal O$_2$ carrying capacity. There is either a reduced concentration of hemoglobin (anemia) or the hemoglobin that is there, has a reduced ability to chemically unite with oxygen. Some common causes are:
  - Any type of anemia causing a reduction in Hb concentration.
  - Certain poisonings which chemically alter Hb.
  - Hb combined with a gas other than O$_2$, *e.g.* carbon monoxide
o **Stagnant Hypoxia**: Any shock state in which there is widespread inadequate tissue perfusion, and hence inadequate tissue oxygenation. This form of hypoxia refers to end organ perfusion. With this type of hypoxia, both the O₂ carrying capacity and the PO₂ may be normal. What causes this problem is an extreme blood flow deficit. This serious lack of blood flow may be localized to a specific region or may be generalized throughout the body. Some of the major causes are:

- General – hypovolemic shock, cardiogenic shock
- Localized – thrombosis, embolus, vasoconstriction.

o **Histotoxic Hypoxia**: While the blood’s ability to pick up and transport O₂ may remain unaffected, a problem may also exist at the cellular level. An action by a toxic substance may prevent the diffusion of O₂ into the cells or may prevent the cells from utilizing oxygen. Cyanide is a poison which will cause this type of hypoxia.

### PHYSICAL FINDINGS ASSOCIATED WITH TISSUE HYPOXIA

1. CNS impairment – restlessness, confusion, unsteady gait, slurred speech, stupor, coma.
2. Tachycardia (early) – ventricular dysrhythmias, and bradycardia (late).
3. Tachypnea, diaphoresis, pallor with or without cyanosis.

---

**Clinical vignette**

Cyanosis, due to the colour of deoxygenated blood, may be present in either type of hypoxia. It should be noted that cyanosis may be absent or reduced in the anemic patient, or the patient poisoned with carbon monoxide.
CHEMICAL CONTROL OF VENTILATION

As concentrations of carbon dioxide, oxygen and hydrogen ions vary in the blood, the respiratory system adjusts in an attempt to maintain normal tissue concentrations. The respiratory center will respond to elevated PCO₂, reduced PO₂ or a lowered pH by increasing alveolar ventilation.

CARBON DIOXIDE

The most powerful stimulant to directly affect the respiratory center is CO₂. Even a small increase, as little as 1%, can increase the respiratory minute volume whereas a small change in PO₂ has almost no effect. Conversely, if one were to voluntarily hyperventilate, causing the PCO₂ to fall below normal, respirations would cease until the PCO₂ is built up again. This situation can also occur when patients are artificially hyperventilated with adjunctive ventilatory equipment.

HYDROGEN IONS

The pH (acidity) of the blood also has a powerful effect on the respiratory center. Carbon dioxide and hydrogen ion concentration rise and fall together causing a combined effect in the control of respirations. Carbon dioxide combines with water to form carbonic acid, which can then dissociate into free hydrogen ions and bicarbonate:

\[ \text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{CHO}_3^- \]

When the CO₂ concentration increases, the chemical reaction (above) shifts to the right. This causes an increase in hydrogen ions (acidity). If there is an increase in H⁺, the reaction shifts to the left causing an increase in carbon dioxide.

The concentration of carbon dioxide and acidity of the blood affect the respiratory center via the CENTREL CHEMORECEPTORS. These chemosensitive receptors are located in the centerolateral surface of the medulla and on other areas of the brainstem. Carbon dioxide combines with water to form carbonic acid which then breaks down into H⁺ and bicarbonate. It is these hydrogen ions that excite the chemoreceptors. The stimulation of the respiratory center causes more carbon dioxide to be eliminated through the lungs, causing a reduction in acidity. This entire process is very important in the acid-base regulation of the body.

OXYGEN

Within the process of respiratory control, the role of oxygen is less important. Changes in the blood pH and levels of carbon dioxide are always fluctuating. These changes affect the chemoreceptors thereby stimulating ventilation in an attempt to maintain normal arterial oxygenation and normal body PH. However, if the PaO₂ falls below 70 mmHg (steep portion of the O₂ curve) then there is a chemical stimulation of the PERIPHERAL CHEMORECEPTORS. These receptors, located in the carotid and aortic bodies, sense the reduction in PaO₂ and stimulate the respiratory center, via nerve fibers, to increase ventilation.
FIGURE 7: PERIPHERAL CHEMORECEPTORS

- Internal Carotid Artery
- Carotid Body
- Carotid Sinus
- Aorta
- Aortic Bodies
OXYGEN DEPRIVATION

Oxygen is essential to life. It is a colourless, odourless and tasteless gas, which supports combustion. Further, it is one of the most powerful drugs used by prehospital personnel – in many cases it can make the difference between life and death.

Oxygen deficiency can have an insidious onset giving little or no warning. It causes an impairment of judgement which may not allow the patient to realize what has happened. There are both subjective and objective signs of oxygen deprivation.

Subjective signs of oxygen deprivation are: dizziness, headache, restlessness, air hunger, visual changes, auditory changes, tingling, apprehension.

Objective signs of oxygen deprivation are: increased ventilatory function, unsteady gait, tachycardia (early), dysrhythmias, cyanosis (late), bradycardia and hypertension (late), unconsciousness (late).

Clinical vignette
Oxygen therapy should be initiated on any patient who is suspect of having one or more of the above signs.

To Review the Rationale for Oxygen Therapy

The purposes of oxygen therapy are to:

1. Increase PO$_2$ in the alveoli and the blood.
2. Reduce the ventilatory workload.
3. Reduce the myocardial workload.

The major function of the cardiovascular and respiratory system is to both supply oxygen to the tissues and remove metabolic waste. Reducing the workload causes a decrease in oxygen utilization and waste production.
Pulse oximetry and End Tidal CO₂ monitoring

**Pulse oximetry**
- A pulse oximeter (SpO₂ monitor) is a non-invasive device which measures the amount of oxygen bound to hemoglobin. The device emits red and infrared light through vascular tissue, such as a nailbed, and measures the amounts of absorbed light on the other side.
- Because hemoglobin changes its shape depending on whether or not it is carrying oxygen molecules, light absorption also changes depending on whether the hemoglobin is carrying oxygen or not.
- Hemoglobin is the oxygen transporting part of the red blood cell.
- Approximately 98% of oxygen is transported bound to hemoglobin - the remaining 2% is transported dissolved in blood plasma.
- Each hemoglobin can carry four oxygen molecules.
- The blood is said to be fully “saturated” with oxygen if every hemoglobin has bound to it, four oxygen molecules.
- A pulse oximeter measures “saturation” of oxygen bound to hemoglobin by percentage - i.e., if all hemoglobin were carrying four molecules of oxygen each, the saturation would be 100%.
- Normal saturation is between 95% and 100%.

**End Tidal CO₂ or ETCO₂**
- The ETCO₂ device measures the amount of exhaled carbon dioxide.
- The measurement is taken at the end of exhaled tidal volume; hence “End Tidal CO₂” or ETCO₂ for short.
- Measurements taken earlier in the exhalation phase would be misleading, as these gases would include oxygen and traces of other gases.
Oxygenation & Ventilation
They’re not the same!

Pulse oximetry and End Tidal CO₂ monitoring are two very valuable adjuncts to prehospital care. However, there are some key respiratory concepts that are essential to review before using these sophisticated, yet simple diagnostic tools.

OXYGENATION
- From a treatment perspective, oxygenation of the patient simply means to provide oxygen. To hyperoxygenate a patient is to provide supplemental oxygen in a high concentration.
- Providing supplemental oxygen affects the PaO₂ level (partial pressure exerted by dissolved oxygen in arterial blood plasma). Supplemental O₂ also affects SpO₂ or the amount of oxygen bound to hemoglobin.
- There is a correlation between SpO₂ and PaO₂. That correlation will be described later with the oxyhemoglobin dissociation curve.

Oxygenation - Sequence of events
- When we inhale atmospheric gas, oxygen diffuses across the alveolar-capillary membrane
- It dissolves in blood plasma
- 98% of it is quickly then taken up and bound to hemoglobin, while the rest remains dissolved in plasma
- At the tissue level, oxygen bound to hemoglobin is released, dissolves in plasma, then diffuses into the tissues

VENTILATION
- Breathing affects primarily the PaCO₂ level (partial pressure exerted by carbon dioxide in arterial blood plasma) – ETCO₂ is an approximation of PaCO₂
- Normal PaCO₂ is 35-45 mmHg
- Hyperventilation blows off CO₂ and therefore may result in PaCO₂ (or ETCO2) level of less than 35 mmHg.
- A patient who is hypoventilating (e.g. narcotic overdose) or a patient who has difficulty exhaling CO₂ because of inflamed bronchioles or mucous plugs in the smaller airways (e.g. emphysems) will retain CO₂. Consequently, the PaCO₂ (or ETCO₂) level may be above 45 mmHg. This may be a “relative” normal finding for emphysems. For that reason, emphysems are sometimes referred to as “CO₂ retainers”.
- In the normal health lung, providing ventilatory support or even hyperventilation (with an FiO₂ of 21%) has little to no effect on PaO₂ or SpO₂, unless supplemental oxygen is added. Hyperventilation does however dramatically lower the ETCO₂ level.
FICK PRINCIPLE

The Fick Principle describes oxygenation from the starting point of oxygen content in the atmosphere (FiO₂) to oxygen utilization at the tissue level.

In the process of troubleshooting why a patient’s SpO₂, consider the following:

Is there?
- **adequate FiO₂ and PAO₂ (atmospheric O₂ in inspired air)**?
  - patient in a chemical vat where the FiO₂ is less than 21% because oxygen is displaced by other gases
  - the patient is not receiving the high FiO₂ you are trying to deliver because the stretcher wheel is overttop of the oxygen tubing, or the O₂ tank has run dry or has not been turned on.
  - at high altitude there is a lower PAO₂
- **adequate diffusion of O₂ across the alveolar-capillary membrane**?
  - a shunt, such as exudate from pneumonia, pulmonary edema, bronchospasm and mucous plugs, is impairing gas diffusion and exchange.
- **adequate affinity for O₂ binding**?
  - in an acidic state, oxygen doesn’t bind as well to hemoglobin
  - in an alkoldotic state, oxygen binds very tightly to hemoglobin but does not release easily at the tissue level
  - carbon monoxide (CO) has a greater affinity to hemoglobin that oxygen. Hemoglobin preferentially binds to CO which reduces the oxygen carrying capacity
- **adequate O₂ carrying capacity**?
  - remember, 98% of oxygen transported in the blood is bound to hemoglobin. A patient who is anaemic or hypovolemic may have an saturation (SpO₂) of 100%, but their oxygen carrying capacity is low. Therefore a patient may be hypoxic despite a normal to high saturation reading.
  - Carbon dioxide (CO) will also bind preferentially to hemoglobin. This not only impairs the body’s oxygen carrying capacity, but because a pulse oximeter cannot differentiate between O₂ and CO, you are likely to see a falsely high SpO₂ reading.
- **adequate perfusion**?
  - shock states or conditions such as a pulmonary embolus reduce or stop blow flow to the lungs resulting in hypoxia
- **adequate release of O₂ at the cellular level**?
  - a patient may have an SpO₂ of 100%, but if the hemoglobin is not releasing oxygen at the tissue level, hypoxia results. This occurs when blood pH is alkalotic.
- **ability of the cell to utilize the O₂ that is delivered**?
  - cyanide poisoning prevents the utilization of O₂ at the cellular level. CO, in addition to its affinity for hemoglobin, also impairs the utilization of O₂ at the cellular level.

When you’re dealing with a patient who is deteriorating despite your efforts at providing them with supplemental oxygen and/or ventilatory support, let the Fick Principle guide you through your problem solving process.
OXYGEN DELIVERY DEVICES

There are two main categories of patients for which oxygen is administered – those who are spontaneously breathing and can accept free flow oxygen and those who are not breathing of have such a poor ventilatory status that they require assisted ventilation.

There is a wide variety of oxygen delivery systems on the market today and no one device is perfect for every job. Since every device cannot be available at all times, those devices with the broadest spectrum of usage will be discussed in this unit. (It is these devices that are most frequently used by paramedics).

The oxygen delivery systems with the broadest spectrum of usage include:
1. Nasal prongs (cannulae)
2. Non-rebreather mask
3. Aerosol mask
4. Bag-valve-mask

The first three oxygen delivery devices listed above are to be used with spontaneously breathing patients and are considered low flow devices. Low flow devices utilize an oxygen reservoir either located within the device itself or within the body’s own anatomic reservoir, Therefore, it should be understood that the system itself does not supply all the inspired gas that a patient requires. Variations in the breathing patterns of the patients can widely affect the FIO₂ (Fraction of Inspired Oxygen)* administered to the patient.

By contrast, high flow systems such as venturi masks are designed to deliver the total gas requirements of the patient while providing a constant and exact FIO₂. The problem with these devices is that they are generally more expensive and cannot provide as high an FIO₂ as a low flow system.

Although not perfect, the low flow systems seem to have the widest flexibility in prehospital care.

Clinical vignette
In the past there has been concerns over how much oxygen is sufficient and how much is too much. This concern was based on the understanding of the respiratory control centers and the fear of suppressing the hypoxic drive found in patients with chronic obstructive pulmonary disease. However, the chance of this happening in the short time that a paramedic is with a patient is remote. This depression in respiratory drive is more likely to be seen in patients on oxygen for longer periods of time. It is far more dangerous to withhold oxygen from a patient who needs it.
TYPES OF DELIVERY SYSTEMS

NASAL PRONGS

Nasal prongs (cannulae) are effective in patients who do not require greater than 30-40% oxygen. This device utilizes the patient’s own anatomical reservoir of oxygen (found in the nasopharynx, oropharynx, and hypopharynx) which mixes with the room air entrained with each breath. Oxygen flow rates should never exceed 6 L/min as this would cause rapid drying and dehydration of the nasal mucosa. Flow rates higher than 6 L/min will not cause a significant increase in FIO2 and therefore result in a waste of oxygen. Patients who will not tolerate a mask may accept nasal cannulae, which may prove to be your only alternative even though a high FIO2 cannot be obtained.

FIGURE 8: NASAL PRONGS

![Nasal Prongs Image]

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>ESTIMATED NASAL CANNULA FLOW RATES*</th>
</tr>
</thead>
<tbody>
<tr>
<td>FLOW RATE (100% O2/L)</td>
<td>% OXYGEN CONCENTRATION</td>
</tr>
<tr>
<td>1</td>
<td>24%</td>
</tr>
<tr>
<td>2</td>
<td>28%</td>
</tr>
<tr>
<td>3</td>
<td>32%</td>
</tr>
<tr>
<td>4</td>
<td>36%</td>
</tr>
<tr>
<td>5</td>
<td>40%</td>
</tr>
<tr>
<td>6</td>
<td>44%</td>
</tr>
</tbody>
</table>

* Assuming normal ventilatory effort
NON-REBREATHER (RESERVOIR) MASK

In contrast to the nasal cannulae, the non-rebreather mask utilizes not only the anatomical reservoir but the mask and the reservoir bag that is attached to it. This device is intended to supply high concentrations of oxygen at FIO$_2$’s between 60-95%. Examples where this device may be utilized include serious trauma, carbon monoxide poisoning, myocardial infarction, pulmonary edema, etc.

Flow rates below 6 L/min should not be used as it may provide insufficient oxygen to fill the reservoir.

TABLE 3

<table>
<thead>
<tr>
<th>FLOW RATE (100% O$_2$/L)</th>
<th>% OXYGEN CONCENTRATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>60%</td>
</tr>
<tr>
<td>7</td>
<td>70%</td>
</tr>
<tr>
<td>8</td>
<td>80%</td>
</tr>
<tr>
<td>9</td>
<td>90%</td>
</tr>
<tr>
<td>10</td>
<td>95+%</td>
</tr>
</tbody>
</table>

* Assuming normal ventilatory pattern
AEROSOL MASK

The aerosol mask is not something routinely used unless administering medications. The design of this mask is such that the oxygen entrained into it picks up sterile normal saline usually containing a medication, e.g. ventolin. As the oxygen passes through the nebulizer it picks up molecular and particulate saline containing medication. This medication is then transferred to the patient via the respiratory system.

At approximately 5-6 L/O₂ this device will provide an FIO₂ of about 40% oxygen.

FIGURE 10: AEROSOL MASK WITH MINI-NEBULIZER

BAG-VALVE-MASK AND OXYGEN POWERED VENTILATORS

Both of these devices can supply 100% O₂ to a patient by either positive pressure or free flow oxygen. Primarily, they are used to ventilate patients who are either not breathing or have insufficient ventilatory function.
While both will do the job, the O₂ powered ventilators do not allow the operator a feel for the patient’s lung compliance. Decreasing compliance is a significant clinical finding. The operator can also feel and hear whether the gas is being delivered to the lungs.

The O₂ powered systems deliver O₂ at a very rapid rate (around 1.6 L/second) and commonly cause an increase in airway pressures. Although most are equipped with a pressure blow off at around 60 cmH₂O gastric distention is common. This is caused by the gastric sphincter muscle in the esophagus having a release point of around 40 cmH₂O. Therefore even with proper use, dangerous and rapid gastric distention may occur causing a decreased in lung volume and an increase potential for vomiting.

The bag-valve-mask system supplies 100% O₂ to the patient when connected to a 15 L/min O₂ source and the reservoir bag is attached. Should the reservoir bag not be used, approximately 60% O₂ can be delivered.

**FIGURE 11: BAG-VALVE-MASK**
HUMIDIFICATION

Humidification is used to add moisture to the dry oxygen. It is accomplished by bubbling the air through water, thereby increasing the air’s relative humidity. Long exposure to very dry air can cause a significant drying of the mucous membranes. The water vapour can also supply warmth to otherwise cool air.

Clinical vignette

In prehospital care, when transport times are under twenty minutes, humidification of oxygen is not as necessary in most cases.

The Ontario Ministry of Health and Long Term Care issued a Directive On May 13, 2003 during the Severe Acute Respiratory Syndrome (SARS) outbreak which stated: “Oxygen should be delivered DRY avoiding nebulized humidity”.

Humidified oxygen may increase the risk of transmission of airborne or droplet infection to the health care worker. Follow local and/or provincial Directives.
CALCULATION OF TANK DURATION

To determine the duration or amount of oxygen in a gas cylinder, a formula may be used.

\[
\text{Duration of Flow (minutes)} = \frac{\text{Gauge Pressure (psi)} - \text{Safe Residual Pressure (SRP)}}{\text{Flow Rate (L/minute)}}
\]

<table>
<thead>
<tr>
<th>CONSTANT FACTOR</th>
<th>TANK CAPACITY</th>
<th>GAUGE PRESSURE (FULL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>D cylinder</td>
<td>0.16</td>
<td>350 Litres</td>
</tr>
<tr>
<td>E cylinder</td>
<td>0.28</td>
<td>625 Litres</td>
</tr>
<tr>
<td>M cylinder</td>
<td>1.56</td>
<td>3000 Litres</td>
</tr>
</tbody>
</table>

The safe residual pressure for all oxygen tanks is 200 psi.

EXAMPLE 3:

What is the duration of tank M, when using a flow rate of 10 L/minute?

\[
\text{Duration of Flow} = \frac{2000 - 200 \times 1.56}{10}
\]

\[
= \frac{2808}{10}
\]

\[
= 281 \text{ minutes}
\]

(4 hours, 41 minutes)

Clinical Note

A HYPOXIC PATIENT SHOULD NOT HAVE OXYGEN WITHHELD FOR ANY REASON
ADVANCED LIFE SUPPORT
PRE COURSE
OXYGEN DELIVERY

SELF-ASSESSMENT

Marks

[1] 1. a) What is meant by “partial pressure” of a gas?

b) The total pressure of the three gases (A, B, C) in the boxes are equal to 760 mmHg. What is the partial pressure of each gas?

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>B</td>
<td>B</td>
<td>A</td>
<td>C</td>
</tr>
<tr>
<td>B</td>
<td>C</td>
<td>B</td>
<td>C</td>
<td>A</td>
</tr>
</tbody>
</table>

GAS A: ___________________________ mmHg
GAS B: ___________________________ mmHg
GAS B: ___________________________ mmHg

[2] 2. Given an adequate supply of oxygen within the alveoli, identify the factors which determine the quantity of oxygen delivered to the tissues.

[1] 3. Oxygen moves out of the alveoli into the circulation by the process of

(a) ________________________________

[2] It is transported in the circulation primarily by (b) __________________, and minimally by (c) __________________.

[1] Oxygen release to the tissues occurs when (d) ____________________.

[3] Factors which effect oxygen release to tissues are (e) ____________________

[3] 4. List, in order of quantity (largest to smallest), the means by which carbon dioxide is transported in the body.

[1] 5. a) The major types of hypoxia are:

__________________________________________

__________________________________________

__________________________________________
[1] b) Cardiogenic shock is an example of which type of hypoxia?

[1] 6. The most reliable indication of hypoxia is cyanosis. (True or False)


7. a) Using the chart below, identify the chemical factors which affect ventilation, the site affected, and the effect on ventilation.

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>SITE AFFECTED</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

RESULT OF INCREASE IN FACTOR ON VENTILATION (↑ OR ↓)

[1] b) The two factors which act together in the control of ventilation are: ____________________________.

[1] c) The least control is exerted by the level of ________________ in the blood.

[1] 8. a) Assuming a normal ventilatory pattern, the nasal cannula will provide ________________% oxygen concentration at a flow rate of 3 L/min.

[1] b) Room air is ______________% oxygen.
9. An otherwise healthy young man is having an acute attack of asthma. The physician has ordered an inhalation treatment using the aerosol mask and mini nebulizer. The medication in the chamber is now gone. The patient appears slightly better. Your ETA to hospital is 15 minutes.

In the absence of a direct order from the physician re: oxygen administration following the inhalation, choose the best option below.

Justify your answer explaining briefly why you did not choose the other options.

a) Leave the aerosol mask in place, with O₂ running.
b) Switch to nasal prongs at 6 L/min.
c) Fill the nebulizer chamber with water and allow the patient to breathe humidified oxygen.
d) Switch to non-rebreather mask at 8-10 L/min.

10. You are called to transfer a patient from hospital A to hospital B. The transport time will be 60 minutes. The doctor orders oxygen at 4 L/min via nasal cannulae during transport. You have a full (2000 psi) D tank ready for the journey. Calculate the number of minutes this tank will last.

33 TOTAL
1. a) The weight, force or tension exerted by a gas within a mixture.

b) The partial pressure of each gas is calculated as a percentage of the whole.
   GAS A: 30% of 760 = 228 mmHg
   GAS B: 40% of 760 = 304 mmHg
   GAS B: 30% of 760 = 228 mmHg

2. Blood flow (perfusion)
The concentration of hemoglobin and the affinity of oxygen for it (oxygenation).

3. a) diffusion
b) combining with hemoglobin
c) dissolving in plasma
d) oxygen concentration in tissues is lower than in the blood
e) pH, temperature, PCO₂

4. as bicarbonate – largest hypoxia
   combined with hemoglobin (carbaminohemoglobin)
   dissolved in plasma - smallest

5. a) hypoxemia; tissue hypoxia
b) tissue hypoxia due to a lack of perfusion

6. False. Cyanosis will be reduced or absent in patients with carbon monoxide poisoning, and those with decreased hemoglobin levels.

   Other factors such as cold temperatures can reduce peripheral perfusion resulting in cyanosis. There is no reliable way to quantify cyanosis clinically. Therefore, as an isolated clinical finding it has little relevance to the patient’s PO₂.
7. a) 1/3 mark each

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>SITE AFFECTED</th>
<th>RESULT OF INCREASE IN FACTOR ON VENTILATION (↑ OR ↓)</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ CO₂</td>
<td>Central chemoreceptors (medulla)</td>
<td>↑</td>
</tr>
<tr>
<td>↓ pH</td>
<td>Central chemoreceptors (medulla)</td>
<td>↑</td>
</tr>
<tr>
<td>↑ (H⁺)</td>
<td>Pheripheral chemoreceptors (cartoid plus aortic)</td>
<td>↑</td>
</tr>
<tr>
<td>↓ O₂</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

b) CO₂ + pH (H⁺)
c) O₂

8. a) 32%
b) 21%

9. (d)
Rationale: There is no good reason not to give this patient the highest concentration of oxygen available. The non-rebreather mask at 8-10 L/min delivers 80-95% oxygen concentration. The nasal cannulae deliver about 44% at 6 L/min., and the aerosol mask about 40% oxygen concentration. Humidification in a trip lasting 15 minutes is not necessary, especially when the O₂ concentration delivered is only 40%.

10. \[
    \frac{2000 - 200 \times 0.16}{4} = \frac{288}{4} = 72 \text{ minutes of oxygen}
\]
ADVANCED LIFE SUPPORT
PRE COURSE
OXYGEN DELIVERY

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.
   Reading __________ hours
   Self assessment __________ hours
   Total time __________ hours

2. Were the objectives of the module clearly stated?
   [ ] yes [ ] no
   If no, please comment.

3. Did you see any of the resource materials?
   [ ] yes [ ] no
   If yes, which items
   _______________________________________________________________________
   _______________________________________________________________________
   _______________________________________________________________________
   Were they helpful? _______________________________________________________________________

4. Were the reference notes adequate?
   [ ] yes [ ] no
   If no, please comment.

5. Were the reference notes easy to follow?
6. Were the examples provided satisfactory?

[   ] yes  [   ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[   ] yes  [   ] no
If yes, please specify.

1. Was the level of the module satisfactory for your program of study?

[   ] yes  [   ] no
If no, please comment.

Base Hospital

9. General comments or suggested improvements.
Paramedic Resource Manual

CARDIOVASCULAR SYSTEM
SECTION THREE

2005 Update by
Ontario Base Hospital Group Education Subcommittee

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OBJECTIVES: CARDIOVASCULAR SYSTEM

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. discuss the features of cardiac muscle and relate these features to the function of cardiac muscle.
2. discuss the electrophysiology of cardiac muscle which differentiates it from other muscle tissue.
3. describe the conductive pathways in the heart and discuss the process of impulse conduction through these pathways.
4. define cardiac output and discuss the factors that affect its control.
5. describe the structure and function of arteries and veins.
6. discuss the role of the autonomic nervous system in relation to the cardiovascular system.
7. discuss the factors affecting the control of blood pressure.
8. discuss the effects of the exercise on the cardiovascular system.
9. relate to above objectives to common clinical situations.

If you have studied this subject previously, you may test your ability using the self-assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.
### GLOSSARY

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BLOOD FLOW</strong></td>
<td>The quantity of blood (in millilitres or litres) that passes a given point in a period of time (minute).</td>
</tr>
<tr>
<td><strong>BLOOD PRESSURE</strong></td>
<td>Force exerted by the blood against any unit area of the vessel wall.</td>
</tr>
<tr>
<td><strong>CARDIAC CYCLE</strong></td>
<td>A complete heartbeat, consisting of the contraction and relaxation of both atria, and the contraction and relaxation of both ventricles.</td>
</tr>
<tr>
<td><strong>CARDIAC OUTPUT</strong></td>
<td>The amount of blood pumped by the ventricles in one minute. Normally approximately 5 litres (70 mL/kg). Cardiac output = stroke volume x heart rate.</td>
</tr>
<tr>
<td><strong>CONDUCTION SYSTEM OF THE HEART</strong></td>
<td>Consists of the SA node, the internodal pathways, the AV node, the bundle of His, bundle branches and the Purkinjie fibers.</td>
</tr>
<tr>
<td><strong>DIASTOLIC PRESSURE</strong></td>
<td>The blood pressure during diastole. Diastolic pressure reflects the state of constriction of the blood vessels.</td>
</tr>
<tr>
<td><strong>ECG</strong></td>
<td>A graphic record of the electrical activity of the heart.</td>
</tr>
<tr>
<td><strong>FRANK STARLING’S LAW OF THE HEART</strong></td>
<td>Within limits, the longer the heart muscle fibers are stretched at the beginning of contraction, the stronger is their contraction.</td>
</tr>
<tr>
<td><strong>PERIPHERAL RESISTANCE</strong></td>
<td>Resistance to blood flow imposed by the force of friction between blood and the walls of the vessels – depends on the viscosity (thickness) of the blood and the diameter of the vessel. Also called afterload.</td>
</tr>
<tr>
<td></td>
<td>Mathematically, resistance depends on:</td>
</tr>
<tr>
<td></td>
<td>$\text{viscosity} \times \text{length of vessel} \div \text{radius}^4$</td>
</tr>
<tr>
<td><strong>PULSE</strong></td>
<td>The alternate expansion and recoil of an artery.</td>
</tr>
<tr>
<td><strong>PULSE PRESSURE</strong></td>
<td>Systolic pressure minus diastolic pressure.</td>
</tr>
<tr>
<td><strong>STROKE VOLUME</strong></td>
<td>The amount of blood ejected from the ventricle with a single contraction of the ventricles</td>
</tr>
<tr>
<td><strong>SYSTOLIC PRESSURE</strong></td>
<td>The peak in blood pressure during systole. Systolic pressure is a reflection of blood volume.</td>
</tr>
</tbody>
</table>
INTRODUCTION

The cardiovascular system, consisting of a pump (the heart), and a distribution and collection system (arteries and veins), performs a number of important functions in the body including:

1. Transportation of oxygen and other nutrients to the cells.
2. The removal of carbon dioxide and other waste products from the tissues.
3. Distribution of hormones and other substances required for physiological control.
4. Control of heat transfer.

Because the system must perform effectively under a wide variety of circumstances, it must have mechanisms to adapt to the changes that occur. Tissue needs vary with activity, and the cardiovascular system adjusts to these needs in a variety of ways.

A basic knowledge of the anatomy and physiology of the cardiovascular system is not just of theoretical interest. It is essential in dealing with clinical situation, such as:

- interpretation of vital signs
- recognition of shock states
- management of the patient with cardiovascular disease
- interpretation of electrocardiograms
- understanding of the role and effects of medications.

The first step in treating a patient is recognition of the abnormalities that are present. To do this effectively, one must first understand normal function and adaptive mechanisms.
THE HEART AND ITS CONDUCTION SYSTEM

The heart is a four-chambered, muscular organ, approximately the size of a closed fist, which lies in the thorax at the level of the fifth to eighth thoracic vertebrae. There are three layers to the heart:

- endocardium (lining)
- myocardium (muscle)
- epicardium (outer layer).

The heart consists of two atria and two ventricles. Functionally the ventricles are the main contractile force of the pump; the atria act primarily as entrances to the ventricles, although they also pump weakly to move the blood into the ventricles. As 70% of the blood passes into the ventricles before atrial contraction, this role is a minor one.

The Atrio-ventricular valves (AV valves), the mitral valve and tricuspid valve, have guide-wires called chordae tendinae attached to the edges of the valve leaflets and anchored to the papillary muscles which attach to the ventricular walls. These attachments prevent the valves from being forced back into the atria (regurgitation) during ventricular contraction.

Clinical vignette
Occasionally in acute myocardial infarction (AMI) a papillary muscle may infarct and rupture suddenly, leading to inability of an AV valve to close properly during ventricular contraction. Consequently, blood leaks back into the atria (regurgitation), and when this occurs with the mitral valve, regurgitation combined with impaired left ventricular contraction in the setting of AMI may lead to blood backing up into the lungs which may lead to acute pulmonary edema (APE).

Enclosing the heart is the pericardium which also consists of three layers.

Clinical vignette
In situations such as inflammation or trauma, the normally thin layer of serous fluid in the pericardial space may increase. Because of the inelastic fibrous nature of the outer layer of the pericardial sac, stretching of the pericardium to accommodate fluid build up is limited and may begin to interfere with the heart’s ability to pump. This is called pericardial tamponade.

Chronically, as much as 1-2 liters may accumulate in the pericardial sac. Acutely however, a sudden increase in pericardial fluid by as little as 150 ml may be fatal.
The right atrium receives blood from the superior and inferior vena cavae, which forms most of its upper and lower walls. It also receives the blood from the coronary veins. From the right atrium, blood passes into the right ventricle through the tricuspid valve, and from there it is pumped a short distance through the pulmonary valve into the pulmonary artery to the lungs. Blood then passes through the lungs for oxygenation.
Clinical vignette

Because the right ventricle pumps blood only a short distance to the lungs, it has a much thinner wall than the left ventricle. Consequently, the right ventricle’s force of contractility is very dependant on changes in preload (Starling’s Law – i.e. within physiological limits, the greater the heart muscle is stretched, the greater its force of contractility.

FIGURE 2: FLOW OF BLOOD THROUGH THE HEART

The four valves of the heart function passively, dependent on pressure gradients for their opening and closure. The mitral valve has two leaflets, the others three. The aorta and pulmonary arteries are dilated just distal to the valves (forming out-pouchings called sinuses), to prevent the leaflets from sticking to the walls of the vessels.

The normal heart sounds results from the vibrations of the closure of the valves. The first heart sound is from the closure of the atrio-ventricular valves and the second heart sound is from the closure of the aortic and pulmonary valves.
NEUROMUSCULAR ELECTROPHYSIOLOGY

The heart consists of three types of muscle – atrial, ventricular and specialized conductive tissue. The muscles of the atria and ventricles are like skeletal muscle in most ways, but distinguishable by the presence of intercalated disks between the cells. These disks allow impulse to travel from cell to cell faster than in other parts of the body. The specialized conductive tissue contracts minimally and transmits impulses at a faster rate.

Electrical potential exist across all cell membranes. Some cells, such as those in nerve and muscle, can transmit impulses along their membranes, i.e. they are excitable.

All cell membranes are surrounded by electrolyte solutions, inside and outside, containing 155 mEq/L of anions (negatively charged ions) and cations (positively charged ions). Excesses of anions over cations immediately inside the cell membrane and of cations over anions immediately outside the cell membrane, generate the membrane potential. This can be electrolyte solutions in a car battery. The excesses of anions and cations occur as a result of active transport or diffusion.

An example of active transport is the sodium pump, which actively transports sodium from the inside of the cell to the outside of the cell. Because sodium is a cation, the transport leads to a positive charge on the outside of the cell and a negative charge on the inside of the cell.
Diffusion depends on the permeability of the membrane (ability of a substance to pass through the membrane) and the concentrations of various ions on either side of the membrane.

At rest, by means of the sodium and potassium pumps and diffusion, the resting membrane potential is approximately -85 mV (millivolts), with a higher concentration of potassium inside the cell and a higher concentration of sodium outside the cell.

Factors that can create such a change include:

- electrical stimulation
- chemicals
- mechanical damage to the cell
- heat, cold.

At the time of the action potential, sodium permeability changes suddenly. Sodium enters the cell, leading to the inside of the cell becoming positive. Sodium is then pumped back out of the cell, returning the cell membrane to its resting state.

Sodium, potassium, and the other electrolytes diffuse through pores in the cell membrane called channels. The change in the permeability to sodium, as occurs with an action potential, is related to the opening of gates that are sensitive to changes in electrical current. These gates or channels are normally closed. Calcium is felt to play a role in closing the gates which control access to the sodium channels. Channels also exist for calcium and magnesium, a familiar concept because of the recent development and use of calcium channel blockers in cardiac patients, e.g. Diltiazem.

In excitable tissue, an action potential at any one point on the cell membrane leads to the adjacent portion of the membrane becoming excited as well. Thus, the action potential along the cell is transmitted, which leads to the creation of an impulse. Once an impulse has been generated, it will travel along the entire membrane. If, however, the fiber is in an abnormal state at the time the impulse reaches it, the action potential may not generate sufficient voltage to stimulate the adjacent area – e.g. as seen in heart block dysrhythmias.

The repolarization process involves the sodium and potassium pumps and is thus an energy requiring process.

Some excitable tissue does not repolarize immediately, but remain on a plateau for a few milliseconds before repolarization occurs. This is the situation with cardiac muscle resulting in contraction of the heart muscle for the duration of the plateau.

All excitable tissues can discharge repeatedly if the threshold for stimulation is reduced to a low enough level. All muscles...
do this when the calcium concentration is low enough. Repetitive discharges normally occur within smooth muscle and the heart.

The membrane, in the resting state of a pacemaker cell is constantly permeable to sodium. Sodium slowly enters the cell, lessening the negativity within the cell. A point is reached where the current inside the cell is sufficiently positive (reaches threshold) to generate an action potential (depolarize).
Pacemaker cells, such as the sino-atrial (SA) Node, have a tendency to generate action potentials rhythmically thus resulting in rhythmic contraction of the heart.

Cardiac muscle has a refractory period which is a period immediately after the action potential when the muscle cannot respond to another impulse. The refractory period for ventricular muscle is longer than that for atrial muscle.

Clinical vignette

Because of the difference in refractory time, the atria can beat faster than the ventricles. The Atrioventricular (AV) Node also has a longer than usual refractory period and acts as a gatekeeper between the atria and ventricles. With a very rapid atrial rhythm, the ventricles may only respond to an impulse being transmitted from the atria every few beats. e.g. atrial tachycardia with 2:1 or 3:1 block or atrial fibrillation.

FIGURE 4: ACTION POTENTIAL FOR CARDIAC MUSCLE

Refractory periods can be functional (absolute) meaning that the muscle cannot respond to any stimulus, or relative, meaning that the muscle may respond to a strong impulse (e.g. premature ventricular complex).

The period of time from one heart contraction to the next (or one P wave to the next) is call the cardiac cycle. The contraction phase is called systole, and the relaxation phase is called
diastole (Figure 5). Systole is actually comprised of atrial and ventricular contraction. Since ventricular contraction is responsible for the pulse, systole usually refers to the ventricular contraction.

The cardiac impulse originates in the sino-atrial node (SA node), the first in the network of specialized conduction tissue, which is located in the posterior portion of the right atrium just beneath, and medial to, the entrance of the superior vena cava. Most cardiac fibers are capable of automaticity, an ability to generate action potentials spontaneously, but the SA node has a higher intrinsic rate than the other tissue and, for this reason, functions as a normal pacemaker of the heart.

The SA node fibers are continuous with the atrial fibers, so that any action potential generated in the SA node spreads to the remainder of atrial tissue. In addition to this generalized spread of the action potential, there are also internodal pathways between the SA node and the atrio-ventricular node (AV node) with faster conduction than normal atrial tissue.
The impulse is delayed at the AV node, resulting in slower conduction through these fibers. This delay in conduction between the atria and the ventricles allows the atria to contract before the ventricles.

From the AV node, the impulse travels down the Bundle of HIS which bifurcates into the right and left bundle branches. The impulse then reaches the into the Purkinje network in the apex of the heart. The Purkinje fibers form a network of small fibers which spread through the ventricular chambers (Figure 6).

**Clinical vignette**

The Electrocardiogram (ECG) is a graphic representation of the heart’s electrical activity. It records the electrical voltage generated by the heart and transcribes it. The P wave represents depolarization of the atria, the QRS represents depolarization of the ventricles and the T wave represents repolarization of the ventricles.

Although it is expected that if the atria and ventricles depolarize (as seen on the ECG) that they will contract, one cannot assume that contraction has taken place unless a corresponding pulse is palpated.
The physiological delay at the AV node is represented by the PR interval. The QRS complex of the ECG is the depolarization of the ventricles, and occurs immediately before ventricular contraction. The T wave is the pattern of repolarization of the ventricles, and occurs just prior to the end of contraction.

The cardiac cycles is thus represented on the ECG as the period between two P waves.

**CONTROL OF CARDIAC FUNCTION**

The cardiac function is controlled by both intrinsic and extrinsic factors.

The major intrinsic factor is venous return. In cardiac muscle, the greater the stretch, the greater the force of contractility. What this means is that within a physiological range, the heart will pump whatever blood it receives (the venous return). This is the Frank Starling Law of the Heart, which can also be stated as “the greater the diastolic filling (the filling of the ventricles during diastole), the greater the cardiac output”.

The extrinsic factors governing cardiac function relate primarily to the autonomic nervous system, which controls not only the rate, but also the degree of contractility of the heart.

The atria are extensively innervated by both sympathetic and parasympathetic (vagal) fibers. The ventricles contain predominately sympathetic fibers, with few parasympathetic fibers.
In general:

- **Sympathetic** stimulation increases both the rate and the strength of contraction.
- **Parasympathetic** stimulation decreases both the rate and the strength of contraction.

The autonomic nervous system involvement in cardiac control originates in the vasomotor centre of the brain. The sympathetic and parasympathetic nervous systems work in a check-and-balance manner to increase or decrease heart rate and force of contractility based on the body’s metabolic demands at the moment. The autonomic nervous system has been compared to the two footed driver – i.e.

- ♥ you can go faster by applying more pressure to the gas pedal (sympathetic), or
- ♥ by simply taking you foot off the brake pedal (parasympathetic inhibition)
- ♥ you can go slower by taking your foot off the gas pedal (sympathetic inhibition), or
- ♥ by applying more pressure to the brake pedal.

Other external factors which govern cardiac function include but are not exclusive to electrolytes and temperature. Electrolytes in the circulation affect heart rate and cardiac contractility because of the role they play in the generation of action potential and the contractions of muscle. High levels of potassium lead to a dilated, flaccid heart with a slower heart rate and delayed transmission through the AV node. High levels of calcium lead to spastic contraction of the heart. Elevated sodium depresses cardiac function. Temperature increase leads to an increase in heart rate and contractility.

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INTRINSIC:</strong></td>
<td></td>
</tr>
<tr>
<td>Venous Return</td>
<td>Greater the venous return</td>
</tr>
<tr>
<td></td>
<td>Greater the cardiac output</td>
</tr>
<tr>
<td><strong>EXTRINSIC:</strong></td>
<td></td>
</tr>
<tr>
<td>Sympathetic</td>
<td>Increases heart rate</td>
</tr>
<tr>
<td>Stimulation</td>
<td>Increases contractility</td>
</tr>
<tr>
<td></td>
<td>Increases conduction velocity</td>
</tr>
<tr>
<td>Parasympathetic</td>
<td>Decreases heart rate</td>
</tr>
<tr>
<td></td>
<td>Decreases contractility</td>
</tr>
<tr>
<td></td>
<td>Decreases conduction</td>
</tr>
<tr>
<td>High Potassium</td>
<td>Decreases heart rate</td>
</tr>
<tr>
<td></td>
<td>Decreases contractility</td>
</tr>
<tr>
<td>High Calcium</td>
<td>Increases contractility</td>
</tr>
<tr>
<td>High Sodium</td>
<td>Decreases contractility</td>
</tr>
<tr>
<td>High Temperature</td>
<td>Increases contractility</td>
</tr>
<tr>
<td></td>
<td>Increases heart rate</td>
</tr>
</tbody>
</table>
ARTERIES AND VEINS

The arteries function primarily as a distribution system for the supply of nutrients to tissues. With repeated branching, arteries become progressively smaller, from the aorta (with a diameter of 2.5 cm) to the arterioles (with a diameter of 0.1 mm). In the majority of organs the branching occurs in a fashion to provide anastomotic channels which may function as alternate routes of supply in the event of obstruction of one of the vessels. End arteries are those where a single vessel provides the flow to a structure with few or no alternate routes.

Clinical vignette

An example of an "End Artery", and its significance, is the central retinal artery of the eye. Obstruction of the central retinal artery by either embolus or thrombus, deprives the retina of nutrients and leads to visual loss in the eye involved.

FIGURE 8: ARTERIES AND VEINS

Arteries are elastic muscular tubules whose walls consist of three layers:

- Intima (the smooth inner lining of endothelial cells)
- Media (the thick middle layer of muscle and elastic fibers)
- Adventitia (the outer layer of fibrous, collagenous tissue).

The arterioles are the last branches of the arterial system, and act as control valves for releasing blood to the capillaries.
The veins have a major role as a storage system (capacitance system), as well as serving as a collecting system to return blood to the heart. Like arteries, their walls are three layers thick. However, being a lower pressure system there is far less muscular and elastic tissue within their walls, and the entire medial layer is far less pronounced.

In contrast to arteries, veins have valves which consist of cup-shaped projections of epithelial tissue, resembling the semi-lunar valves of the heart. These are especially prominent in the long extremity veins. The valves serve to promote unidirectional blood flow. When the muscles contract, the veins are compressed and the blood is pushed in a forward direction.

The smooth muscle in both arteries and veins is under the control of the autonomic nervous system.

The exchange of nutrients takes place at a capillary level. Capillaries are supplied by arterioles and drained by venules. They are small vessels with walls consisting of only endothelial cells on a basement membrane. The diameter of capillaries is measured in micrometres (a micrometer is 1/1,000 of a millimeter). Their structure allows the ready exchange of nutrients and waste materials through the vessel walls. There are no smooth muscle cells in the capillaries, and they are not under the control of the autonomic nervous system.

PRACTICAL ANATOMY

No attempt will be made to review the entire anatomy of the cardiovascular system. Rather, certain practical points will be mentioned.

In the course of practising Paramedicine, it is worthwhile to have access to an anatomy and physiology book, as it is surprising how often something will arise which can be explained by an understanding of the anatomy and physiology. It is far easier to remember anatomy when it has acquired relevance through a case, and it is worthwhile taking advantage of this sort of opportunity.
CORONARY ARTERIES

It is of value to know the circulation of the coronary arteries in order to understand some of the problems that can arise with occlusion of one of the arteries, e.g. myocardial infarction. The two main arteries are the right coronary artery (RCA) and the left coronary artery. The left divides, near its origin, into the left anterior descending (LAD) and the left circumflex (CX).

As mentioned previously, these arise from the sinuses just distal to the aortic valve and travel along the surface of the heart. Cardiac muscle is supplied entirely by coronary vessels. There is no nutrient or gas exchange across the endocardial surface within the heart’s chambers. There are normally few or no anastomoses between the coronary vessels, making many of them End Arteries in the terms of occlusion (Figure 10).

The coronary arteries also supply the SA and AV nodes and the conduction system of the heart, so occlusion of these vessels can lead to ischemia of the nodes or conduction system, and subsequent arrhythmias.
PUSLE POINTS

Points where arteries are readily palpable are important in detecting a pulse in a situation where the blood pressure is low enough to prevent effective use of a sphygometer and where a fracture, crush injury or embolus may have obstructed flow to a vessel or make use of a cuff impossible. Common pulse points (Figure 11) are:

- Carotid
- Brachial
- Radial and ulnar
- Posterior tibial
- Dorsalis pedis
- Femoral.

The popliteal pulse (posterior leg behind the knee) is also palpable, but can be difficult to feel owing to the anatomy of the area.
INTERCOSTAL VESSELS

Should it be necessary to insert a needle into the chest of a patient with a tension pneumothorax, then it is useful to know that the nerve, artery and vein of the intercostal space travel on the under surface of the ribs. For this reason, a needle (or a chest tube in the hospital setting) should be inserted at the top of the rib, to avoid damaging the artery and causing intrathoracic bleeding. A needle thoracostomy is performed in the 2nd intercostal space at the mid-clavicular line.

AORTIC BRANCHES

The major branches of the aorta are useful to know to assist in the detection of a thoracic aortic dissection in the upper portion of the aorta. In this condition, the blood pressure in one arm may be lower compared to the other as blood may dissect between the intimal and medial layers along the aorta and into one of the subclavian arteries—e.g. It will be lower on the left side if blood dissects into the left subclavian artery. A blood pressure difference of ≥ 15 mmHg systolic in one arm compared to the other is considered significant. This is commonly a major clue to the diagnosis of thoracic aortic dissection and is one reason why understanding of the anatomy is important.

FIGURE 12: MAJOR BRANCHES ASCENDING AORTA

![Diagram of Major Branches Ascending Aorta](image)
JUGULAR VEIN

Recognition of jugular venous distention is important in a number of clinical situations, such as heart failure and pericardial tamponade. Therefore, it is important to know the location of the jugular vein in the neck (Figure 13).

Clinical caveat

In 3rd degree AV block and some instances of ventricular tachycardia, you may see irregular jugular venous distension (Cannon A waves) as the atria and ventricles contract at different rates (asynchronously). This means that at times the atria will contract against closed AV valves and the blood will back up into the jugular veins. Look at the neck veins in these dysrhythmias.

FIGURE 13: EXTERNAL JUGULAR VEIN

It can be worthwhile to review the relationship of some of the major arteries to bony structures to understand how a person can fracture a clavicle and lose a radial pulse, or injure the popliteal artery with a dislocation of the knee.

By relating the anatomy to practical situations, it is not only easier to recall information, but also allows you to anticipate what structures might have been injured in a particular situation.
PHYSIOLOGY OF THE CIRCULATION

REGULATION OF BLOOD FLOW

Blood flow is regulated by three sets of factors:

- Local
- Nervous
- Humoral.

LOCAL CONTROL

The prime local determinant of blood flow in most tissues is the partial pressure of oxygen. When the oxygen concentration is low, due to increased metabolism and increased oxygen consumption, the pre-capillary sphincters open, leading to increased flow in the tissues. The opposite occurs in the pulmonary circulation where if a group of alveoli are obstructed, the surrounding vessels will constrict and blood will be shunted to other areas of the lungs for gas exchange.

In a situation of high local concentration of oxygen, the pre-capillary sphincters close. This may relate to the fact that the smooth muscle contraction is an energy requiring process. Thus, when the oxygen is low, the necessary energy to cause contraction and closure of the sphincter is lacking, and the muscle relaxes.

Two special situations exist where other factors are prime determinants of sphincter smooth muscle activity:

1. In the kidney, the flow increases in response to an elevated level of sodium in the serum, or to an elevated level of end products of protein metabolism.

2. In the brain, flow increases in the presence of an elevated level of carbon dioxide (PaCO₂) or hydrogen ions.

Clinical vignette

Although PaCO₂ is a potent cerebral vasodilator and may contribute to the increase in intracranial pressure (ICP) in the setting of a head injury, hyperventilation is not indicated in the prehospital setting unless hypercarbia is suspected, and even then, it should be done briefly. Ideally, ETCO₂ monitoring should be available to ensure CO₂ is kept to the low end of normal – i.e. approximately 33 mmHg.

Hyperventilation of the head injured patient has gone out of vogue in the prehospital setting because of the risk of the "watershed effect" the vessels within the injured area are damaged constricting the vessels surrounding the damaged area from hyperventilation results in blood flow into the damaged area resulting in worsened edema and further secondary brain damage
NERVOUS FACTORS

Two major features dominate the nervous system control of blood flow in tissue. First, the control has a rapid onset (within 1 second), and second, nervous system control overrides local factors, affecting larger areas of the body.

The sympathetic nervous system exerts the most important influence over the peripheral vascular system. Vasomotor fibers of the sympathetic system leave the spinal cord through all thoracic as well as the upper two lumbar vertebrae. These fibers pass into the sympathetic chain, and are then distributed to the vessels through the peripheral sympathetic nerve fibers and the spinal nerves. Sympathetic stimulation alters peripheral vascular resistance and therefore alters flow. As the system also controls the venous system, it is capable of changing the volume in the capacitance system.

The vasomotor centre of the brain acts as the control centre for this system. The upper and lateral portions of the centre, which lie in the reticular substance of the lower brainstem, are tonically active, constantly sending nerve impulses at the basal rate through the vasoconstrictor fibers. This is called sympathetic vasoconstrictor tone and maintains all vessels in a constant state of partial contraction called vasomotor tone. Loss of this tone, such as would occur with a transection of the spinal cord, leads to a significant fall in blood pressure.

Clinical vignette

Patients with a high thoracic or cervical cord injury may be hypotensive, but will not exhibit the typical reflex tachycardia seen in shock. The patient will have a normal pulse and be warm to touch. The normal pulse and warm skin results from the disruption of the sympathetic outflow from the spinal cord and loss of vasomotor tone. Patients are warm and have a low blood pressure because the vessels are dilated. Hemorrhagic shock can be present in these patients as well, however the usual signs of shock will be absent due to the loss of sympathetic tone from the cord injury.

The vasomotor centre controls vasoconstriction and cardiac activity.

Many areas of the brain influence the vasomotor centre, including the cortex of the brain. Fear is an example of the cortex stimulating the vasomotor centre to increase sympathetic outflow. With fear, our heart rate increases, our peripheral vessels constrict and we sweat – all triggered by the sympathetic nervous system. In the same way when we think of exercising, the vasomotor centre receives this message, and our heart and vessels are preparing for this before we actually start the exercise.

HUMORAL FACTORS

Norepinephrine is the substance secreted at the endings of the vasoconstrictor fibers facilitating smooth muscle contraction in vessels. If vasoconstrictor impulses are sent to the adrenal medulla, this leads to the release of norepinephrine and epinephrine into the bloodstream from the medulla, as circulating catecholamines.
The sympathetics to skeletal muscle carry dilator as well as constrictor fibers. Stimulation of the motor cortex leads to the release of these vasodilators through the hypothalamus.

Norepinephrine and epinephrine from the adrenal medulla are the major humoral factors involved in flow control. Norepinephrine is a vasoconstrictor and epinephrine dilates vessels to skeletal muscle and cardiac muscle, while causing vasoconstriction in other areas.

The factors controlling blood flow are summarized in Table 2.

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local:</td>
<td></td>
</tr>
<tr>
<td>Increased oxygen concentration</td>
<td>Vasoconstriction</td>
</tr>
<tr>
<td>Decreased oxygen concentration</td>
<td>Vasodilation</td>
</tr>
<tr>
<td>Nerves:</td>
<td></td>
</tr>
<tr>
<td>Sympathetic system</td>
<td>Vasoconstriction</td>
</tr>
<tr>
<td></td>
<td>Vasodilation (skeletal muscle)</td>
</tr>
<tr>
<td>Humoral – Adrenal Medulla:</td>
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</tr>
<tr>
<td>Norepinephrine</td>
<td>Vasoconstriction</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Vasodilation (except skeletal and cardiac</td>
</tr>
<tr>
<td>muscle where vasodilates)</td>
<td></td>
</tr>
<tr>
<td>Angiotensin</td>
<td>Vasoconstriction</td>
</tr>
</tbody>
</table>

**BLOOD PRESSURE AND ITS CONTROL**

Normally our bodies control our mean arterial blood pressure within the range of 80-120 mm mercury. With age, the mean arterial pressure increases – the systolic component increasing more than the diastolic component. This increase is attributed to atherosclerosis, with loss of the normal elasticity of the vessels.

The control of blood pressure operates on a feedback system, similar to the thermostat in your home which provides a feedback system to the furnace to regulate the heat.

The arterial pressure depends on the cardiac output and the mean arterial resistance, which, from the definitions given in the glossary, is highly dependent on the radius of the vessel – the smaller radius, the higher the resistance. Most of the resistance in the cardiovascular system is at the level of the arterioles.

The means of controlling blood pressure are either:

- Rapid – autonomic nervous system circulating catecholamines
- Intermediate
- Slow – the renal system
There is normally a degree of sympathetic stimulation with vasoconstriction in the peripheral circulation keeping the tone of the vessels above baseline in the same fashion that there is a degree of sympathetic stimulation of the heart.

**RAPID SYSTEMS**

Baroreceptors, or pressure receptors, are found in most of the major arteries of the thorax. They are most abundant in the walls of the internal carotids just above the bifurcation of the common carotids and the walls of the aorta.

Stimulation of these receptors by stretch secondary to higher blood pressure sends a message to the vasomotor centre of the medulla (carotid baroreceptors) or up the vagus nerve to the same centre (aortic baroreceptor).

This stimulation leads to inhibition of the vasoconstrictor centre and excitation of the vagal nerve. The end result is a fall in blood pressure due to decrease in heart rate, decrease in contractility and dilation of the peripheral vessels. With a persistent elevation of blood pressure, this system adapts to accept the new level as normal.

**FIGURE 14: BARORECEPTORS**

![Diagram of Baroreceptors](image)

Pressure ↑ sensed in AORTIC and CAROTID BARORECEPTORS

Via Afferent Nerves

Medulla

Inhibit Vasoconstrictor Centre
Excite Vagal Centre

Vasodilation
↓ Heart Rate
↓ Contractility

↓ Pressure
**Chemoreceptors** (located in the carotid and aortic bodies) are sensitive to a fall in oxygen concentration, a rise in hydrogen ion concentration, or a rise in carbon dioxide. These respond best when blood pressure is low. When stimulated, messages regarding the situation are taken to the vasomotor centre and lead to excitation of the vasomotor centre with the corresponding rise in blood pressure (vasoconstriction, increased heart rate, increased contractility).

The **venous system** is also stimulated in all of these situations. The response of constriction in this system is to decrease the capacitance of the veins. From Starling’s Law of the Heart, it is known that the heart will pump (within limits) what it receives, so it follows that increasing the venous return increases the cardiac output, and in turn, the blood pressure. With lack of stimulation, more blood is stored in the venous system, and venous return to the heart is less.

The arterial resistance is called the afterload of the heart, and the venous return is called the preload. These terms can be very useful in understanding abnormalities which occur in the cardiovascular system.

Sympathetic stimulation, as mentioned above, also leads the stimulation of the adrenal medulla to release epinephrine and norepinephrine into the circulation. These circulating catecholamines excite the heart, constrict most arterioles, and constrict veins.

**INTERMEDIATE SYSTEMS**

The intermediate systems are those of renin-angiotensin, and vasopressin.

**SLOW SYSTEMS**

The primary long term system for the control of the blood pressure is the renal system which controls the circulating volume. In general, high blood pressure leads to increased output of water and electrolytes by the kidneys which depletes the extracellular fluid volume, decreases venous return (preload) and cardiac output, and thus lowers the blood pressure. The reverse is true of low blood pressure.

Unlike the other systems, which can adapt to a new level of blood pressure over time, this system remains active.
TABLE 3
FACTORS CONTROLLING BLOOD PRESSURE

<table>
<thead>
<tr>
<th>FAST ACTING SYSTEMS</th>
<th>INTERMEDIATE</th>
<th>SLOW ACTING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baroreceptors</td>
<td>Renin – Angiotensin</td>
<td>Renal – body fluid system</td>
</tr>
<tr>
<td>Chemoreceptors</td>
<td>Vasopressin</td>
<td></td>
</tr>
<tr>
<td>Circulating Catecholamines</td>
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</tbody>
</table>

CARDIAC OUTPUT

The cardiac output is the quantity of blood pumped from the left ventricle into the aorta per minute. In normal resting conditions in an average adult, cardiac output averages 5 litres/minute (70 mL/kg). In the athlete during exercise, it can increase to over 20 litres/minutes.

Since this quantity of blood depends on the quantity pumped with each beat of the ventricle (stroke volume), and the number of beats per minute (heart rate).

\[
\text{CARDIAC OUTPUT} = \text{STROKE VOLUME} \times \text{HEART RATE}
\]

From this it can be seen that the factors controlling the cardiac output will be those controlling the stroke volume and the heart rate. It can also be seen that over a period of time,

\[
\text{CARDIAC OUTPUT} = \text{VENOUS RETURN}
\]

The heart will normally pump the amount of blood entering the right atrium (within physiological limits). Under normal conditions the venous return (preload) is a prime determinant of cardiac output. When the heart becomes that limiting factor (it can no longer pump the amount of blood it receives), the heart is said to be failing.

Factors that increase the permissive level of the heart to pump what it receives are increased heart rate and increase contractility. As these occur with sympathetic will increase the cardiac output.

Factor affecting venous return (preload) are:

- Increased preload with:
  - Increased blood volume
  - Increased sympathetic stimulation
  - Contraction of skeletal muscle
Clinical vignette

Every time we take a breath, intrapleural pressure changes. With inspiration, our intrapleural pressure is more negative; with expiration, more positive. In situations such as asthma, where expiration is compromised by marked narrowing of the airways with expiration, the pressure in the intrapleural space can become considerably more positive and actually impair venous return.

The veins are a low pressure system. Positive pressure can easily collapse them. In the asthmatic with a high intrapleural pressure, the veins can collapse, so that venous return falls much more than the norm with expiration. This can be detected in a fall in the patient’s blood pressure with expiration, a situation called pulsus paradoxus. Why it has been given this name is confusing, as it is not different from normal, but rather and exaggeration of a normal response which all of us have every time we breathe.

With a normal heart, the heart can pump what blood it receives. If some factor lowers the heart’s ability to pump, then there will be a backup in the venous system, (heart failure). This leads to elevation of the jugular veins in the neck and can lead to engorgement of the liver, and leakage of fluid into the pulmonary circulation, leading to pulmonary edema.

Factors that affect the heart’s ability to pump are:

- Increased with:
  - Sympathetic stimulation
  - Hypertrophy (more muscle, as in an athlete)
  - Low systemic arterial pressure
  - Inhibition of the parasympathetics.

- Decreased with:
  - MI
  - Parasympathetic stimulation
  - Inhibition of sympathetic stimulation
  - Myocarditis
  - Cardiac hypoxia
  - Congenital heart disease.
While at rest, the amount of oxygen might well be very adequate - but what happens if this patient exercises?

Exercise will require increased cardiac output to supply the needs of the skeletal muscles of the body. To prepare for this, the body stimulates the vasomotor centre and the sympathetic nervous system constricts the peripheral blood vessels, increases the heart rate and increases the contractility of the heart.

The afterload against which the heart must pump is increased, and the preload is increased. The heart must pump more blood against greater resistance to deal with the situation. This increases the work that the heart must do increasing its need for oxygen.

As the heart's arteries are incapable of meeting these needs, the muscle of the heart become ischemic and the patient develops angina – chest pain. If the ability of the heart is greatly impaired, the patient may actually go into pulmonary edema.

Now the patient takes a nitroglycerine tablet before the exercise. The nitroglycerine tablet has the effect of increasing the venous capacitance, lessening the preload of the heart, and reducing the systemic vascular resistance lessening the afterload on the heart. Since the heart has less work to do, with the decrease in preload and afterload, the patient can exercise without experiencing chest pain. Naturally we are not talking about our cardiac patient running a marathon with nitroglycerine, but he is able to do more than he would be able to do without it.
ADVANCED LIFE SUPPORT  
PRECURSE  
CARDIOVASCULAR SYSTEM  

SELF-ASSESSMENT

Marks

[10] 1. What should you examine in a patient who has fallen on his shoulder and has swelling and tenderness over the clavicle? Give reasons for your examination.

[7] 2. How are arteries and veins different in structure and function?

3. An individual has sustained a major GI bleed, losing a significant amount of his blood volume. Identify the mechanism that will come into play in an attempt to maintain his blood pressure. Describe the physical findings of the examination of the patient that reveal that these are in force.

Complete these tasks using the outline below, filling in the blanks.

[4] a) The body attempts to maintain tissue flow at an acceptable level to deal with the needs. If the patient bled, his ________ would fall, and therefore his ________ would fall. This would lead to a fall in _______________ and ultimately a fall in _______________.

[7] b) The ________ would detect this fall leading to _______________ stimulation. This stimulation leads to an increase in _______________ and ________ of the heart. The peripheral cardiovascular system compensates with increased _______________ and _______________ tone.

[4] c) The vital signs would include _______________ and the patient would have _______________ skin because of _______________. The _______________ might still be normal if these methods were effective.

d) Over time, the _______________ would attempt to correct the situation by retaining fluid to replace the lost volume.

[5] e) Since the cardiac output = _______________ x _______________, the heart rate would increase initially; then a point would be reached where ________ filling was not complete because of the rapid rate. This would lead to a fall in the stroke volume, and eventually a fall in the ________ (because of the rapid heart rate). At this stage, if volume continues to be lost, the __________________________ will fall in spite of the increase in heart rate.
As blood provides oxygen carrying capacity, the fall in blood volume will lead to less oxygen being carried to the tissues, and metabolism in some tissues where vasoconstriction is intense. The respiratory rate will _____________ to keep the acid base balance normal.

The patient will have these findings as a result of the compensation mechanisms. He may become confused secondary to the lack of _____________ and will demonstrate a _____________ in pressure when sat up (as the normal compensation mechanisms will already be in force with the patient supine).

Why could a patient who has sustained a heart attack have a fall in blood pressure during transport? (In a heart attack, a branch of one of the coronary arteries is blocked.)

What are the factors and their effects involved in increasing cardiac output to deal with exercise?

The autonomic nervous system involves which aspects of the cardiovascular control?

Outline the ANS affects (sympathetic and parasympathetic) on each of the above aspects.

Explain the two special features which differentiates cardiac muscle from other muscle.

Why is the SA nodes the “pacemaker” of the heart?

Sympathetic outflow for peripheral vascular control is at the spinal cord level of _____________ to _____________.

Sympathetic control is exerted over which of the following: (more than one answer may be correct)

- Arteries
- Veins
- Capillaries.

66 TOTAL
1. Key concepts are in bold.

Since subclavian artery runs underneath the clavicle, if the patient has fractured his clavicle there could be damage to the subclavicle artery. This artery supplies the arm, so it would be important to check the pulses distal to the fracture, namely the branchial pulse on the medial molar side of the elbow, and the radial and ulnar at the wrist.

One might also check:
- The blood pressure in both arms to see if they were the same
- The temperature of the injured arm compared to the other (if the blood supply was less it would be cooler)
- Function of the muscles in the distal arm
- Sensation in that area.

2. Key concepts are in bold.

- Arteries carry blood under more pressure than veins
- Arterial walls have thicker muscular coats and more elastic tissue than veins
- Veins have valves to assist in the one way flow, arteries do not
- Veins have a storage function for blood (one of their major roles), which is different from arteries
- Arteries have a major role in providing resistance to flow by dilation and constriction. The resistance in veins is much less
- Arteries carry blood away from the heart, veins carry blood to the heart.

3. a) The body attempts to maintain tissue flow at an acceptable level to deal with the needs. If the patient bled, his blood volume would fall; and therefore his venous return would fall. This would lead to a fall in cardiac output, and ultimately a fall in blood pressure.

b) The baroreceptors would detect this fall leading to sympathetic stimulation. This stimulation leads to an increase in rate and contractility of the heart. The peripheral cardiovascular system compensates with increased arterial and venous tone.

The substance with mediate and regulate blood flow control at this time are collectively called catecholamines.
c) The vital signs would include increased heart rate, and the patient would have cool skin fuse of peripheral vasoconstriction. The blood pressure might still be normal if these methods were effective.

d) Over time, the kidneys would attempt to correct the situation by retaining fluid to replace the lost volume.

e) Since the cardiac output = stroke volume x heart rate the heart rate would increase initially; then a point would be reached where ventricular filling was not complete because of the rapid rate. This would lead to a fall in the stroke volume, and eventually a fall in the cardiac output (because of the rapid heart rate). At this stage, if volume continues to be lost, the blood pressure will fall in spite of the increase in heart rate.

f) As blood provides oxygen carrying capacity, the fall in blood volume will lead to less oxygen being carried to the tissues, and anaerobic metabolism in some tissues where vasoconstriction is intense. The respiratory rate will increase to keep the acid base balance normal.

g) The patient will have these findings as a result of the compensation mechanisms. He may become confused secondary to the lack of adequate circulation to the brain, and will demonstrate a — postural drop in pressure when sat up (as the normal compensation mechanisms will already be in force with the patient supine).

4. The coronary arteries supply the muscle of the heart, and they lack anastomotic channels. For this reason if one of the coronary arteries is blocked, the area supplied by that artery is without oxygen and becomes non-functional.

Depending on the vessels, this could lead to:

○ Damage to muscle, therefore less effective pumping of the heart, resulting in less stroke volume and a fall in blood pressure.

○ Damage to the conducting system of the heart, therefore development of an arrhythmia with a fall in the heart rate, and/or less co-ordinated pumping of the heart, leading to a fall in blood pressure.

○ Rupture of the heart through the dead muscle, leading to fluid collecting in the pericardium, less effective pumping from the increased pressure on the muscle, and less effective filling due to pressure on the atria, with the end result being a fall in blood pressure.

5. With exercise the cardiac output increases to cope with the increased demands for oxygen and other substrates, and the increased need to remove waste materials.
The thought of exercise leads to an increase in cardiac output. The autonomic nervous system causes increased heart rate and contractility, and increased venous tone. These lead to an increase in cardiac output.

At the onset of exercise, the motor cortex is stimulated leading to sympathetic vasodilation to muscles, increased muscle blood flow, and increased cardiac output (less resistance to flow to muscles). This stimulation also leads to increased heart activity and blood pressure.

The use of the skeletal muscles leads to increased venous return.

The increased muscle metabolism, leads to a fall in the oxygen concentration at the capillary level of the muscles, with this local factor leading to vasodilation there.

6. The autonomic nervous system is involved in the control of both cardiac and vascular function.

7. (½ mark for each effect – given in bold)

Cardiac
  - Sympathetic stimulation leads to
    - Increased heart rate
    - Increased contractility
    - Increased conduction speed.
  - Parasympathetic stimulation leads to
    - decreased heart rate
    - decreased contractility
    - decreased conduction speed.

Vascular
  - Sympathetic stimulation leads to
    - vasoconstriction of arteries
    - vasoconstriction of veins

8. Plateau on the action potential which causes contraction for the duration of the plateau.

   Repetitive discharges, which lead to a tendency for rhythmic contraction.

9. The SA node has a higher intrinsic rate of discharge than other sites.

10. a) T1 to L2
    b) Arteries and veins
ADVANCED LIFE SUPPORT
PRE COURSE
CARDIOVASCULAR SYSTEM

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.
   
   Reading ___________ hours
   Self assessment ___________ hours
   Total time ___________ hours

2. Were the objectives of the module clearly stated?

   [ ] yes  [ ] no
   If no, please comment.

3. Did you see any of the resource materials?

   [ ] yes  [ ] no
   If yes, which items
   __________________________________________________________
   __________________________________________________________
   __________________________________________________________
   Were they helpful?
   __________________________________________________________

4. Were the reference notes adequate?

   [ ] yes  [ ] no
   If no, please comment.

5. Were the reference notes easy to follow?

   [ ] yes  [ ] no
   If no, please comment.
6. Were the examples provided satisfactory?

[ ] yes  [ ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[ ] yes  [ ] no
If yes, please specify.

1. Was the level of the module satisfactory for your program of study?

[ ] yes  [ ] no
If no, please comment.

Base Hospital

9. General comments or suggested improvements.
OBJECTIVES: SHOCK

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. define shock.
2. classify the types of shock.
3. briefly describe the pathophysiology and distinguishing clinical features of each type of shock.
4. for each type of shock, state prehospital situations in which the provider should anticipate the development of shock.

If you have studied this subject previously, you may test your ability using the self-assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.
INTRODUCTION

The approach to and management of the patient in shock requires a systematic, organized process based on the underlying pathophysiology and an understanding of the fundamental principles of resuscitation. This module will focus on:

1. A useful definition of shock.
2. A number of important points with respect to the diagnosis of shock.
3. A brief explanation of the pathophysiology at a cellular level.
4. A classification of the types of shock.
5. An overview of the patient’s compensation to reduced tissue perfusion.
6. A grading system for the severity of hemorrhagic shock.
7. The principles of assessment and management in the prehospital setting.

DEFINITION

Shock is a clinical state in which there is a widespread reduction of tissue perfusion resulting in:

- inadequate oxygenation at a cellular level
- inadequate removal of toxic metabolic by-products which, if prolonged, leads to a generalized impairment of cellular function and ultimately cellular death.

The key words in defining shock are perfusion and oxygenation.

IMPORTANT POINTS

The words hypotension and shock are not synonymous. Many individuals will be encountered in the prehospital environment with blood pressures of 90/50, which is “normal” for that particular patient. This particular individual remains warm, well perfused, and well oxygenated at a cellular level. In contrast are patients with a blood pressure of 120/70, who may in fact be in shock. This individual may normally be markedly hypertensive, and now clinically is vasoconstricted, peripherally cyanosed, and poorly perfused. Therefore, not only are hypotension and shock not synonymous, but a normal blood pressure does not ensure adequate cellular oxygenation. As will be emphasized in this module, it is the entire clinical picture of the patient which determines the adequacy of tissue perfusion.

THE CELLULAR LEVEL

Prior to explaining the disruption which occurs at a cellular level, it is important to understand the principles of cellular metabolism. Cells (which in large numbers combine to make up tissues; tissues combine in large numbers to make up organs) can utilize three principal sources for the energy necessary for routine cellular functions, i.e. Carbohydrates, proteins, lipids. These sources are necessary for producing cellular constituents, maintaining a functioning cellular membrane, performing special functions depending on the specific organ.
CARBOHYDRATES
Under most circumstances, cells will preferentially utilize carbohydrates, in the form of glucose. When glucose is metabolized by cells, energy is stored in the form of adenosine triphosphate (ATP), an energy rich molecular compound.

Two processes are available for metabolism of glucose:

- The first is inefficient, expensive for the cell and occurs in the absence of oxygen, i.e. an anaerobic environment. This process is known as glycolysis which produces 2-3 ATP and lactic acid as a by-product.
- The second process is efficient and occurs in the presence of oxygen, i.e. an aerobic environment. This is known as the Krebs Citric Acid Cycle and runs in combination with the Electron Transport Chain producing 36-37 ATP and CO₂ as a by-product which is easy to eliminate.

Under most circumstances most cells in the body utilize the efficient pathways in an aerobic (oxygen rich) environment.

When cells suffer from a generalized decrease in perfusion, there is a reduction in the delivery of oxygen to cells. The rapidity with which this occurs and the host’s ability to compensate (outlined below) will determine the ultimate outcome.
If the reduction in oxygen delivery to the cell persists, anaerobic or inefficient metabolism occurs. The process of glycolysis yields pyruvate as an end product which is subsequently metabolized to lactic acid. The accumulation of lactic acid results in a metabolic acidosis within and outside the cell.

Necessary cellular functions (determined by the organ in which the cell operates) ceases because of both decreased energy and the accumulation of toxic by-products of anaerobic metabolism. The cell soon becomes unable to perform its necessary homeostatic processes. Intracellular structures called lysosomes (bags of toxic enzymes) rupture and digest the cellular contents. The cell membrane becomes incompetent, and with increasing time, ruptures. This releases the toxic intracellular enzymes and metabolic by-products into the circulation. The delivery of these toxic substances to other cells results in further cellular dysfunction and damage. Tissues and subsequently organs, can no longer function. The organism finally dies if the process is not reversed (irreversible shock).

The goal of resuscitating the patient in shock is to improve cellular perfusion and oxygenation.
CLASSIFICATIONS OF SHOCK

Not all shock is caused by hypovolemia. This is important not only for the purpose of diagnosis, but also for management and resuscitation.

The individual who is in shock because of a tension pneumothorax, will not respond to crystalloid resuscitation (volume infusions of I.V. fluids such as normal saline). An individual who is in anaphylactic shock, will require the administration of epinephrine, to control the process of mediator release and subsequent vasodilation and capillary leak. A simple classification of the causes of shock is listed in Table 1.

<table>
<thead>
<tr>
<th>TYPES</th>
<th>CAUSES</th>
</tr>
</thead>
<tbody>
<tr>
<td>HYPOVOLEMIC</td>
<td></td>
</tr>
<tr>
<td>Hemorrhagic</td>
<td>o Internal loss, e.g. GI bleed</td>
</tr>
<tr>
<td></td>
<td>o External loss, e.g. compound fracture</td>
</tr>
<tr>
<td></td>
<td>o GI losses, e.g. prolonged severe diarrhea in infants</td>
</tr>
<tr>
<td></td>
<td>o Renal losses, e.g. excessive use of diuretics</td>
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<tr>
<td></td>
<td>o Cutaneous losses, e.g. heat exhaustion, burns.</td>
</tr>
<tr>
<td>Non-Hemorrhagic</td>
<td></td>
</tr>
<tr>
<td>MECHANICAL/ OBSTRUCTIVE</td>
<td>o Tension pneumothorax</td>
</tr>
<tr>
<td>(Mechanical interference</td>
<td>o Cardiac tamponade</td>
</tr>
<tr>
<td>with blood flow)</td>
<td>o Massive pulmonary embolus</td>
</tr>
<tr>
<td></td>
<td>o Dissecting aortic aneurysm</td>
</tr>
<tr>
<td>CARDIOGENIC</td>
<td>o Myocardial infarction/contusion</td>
</tr>
<tr>
<td>(Impaired function of the</td>
<td>o Arrhythmia</td>
</tr>
<tr>
<td>heart as a pump)</td>
<td></td>
</tr>
<tr>
<td>NEUROGENIC</td>
<td>o Spinal shock</td>
</tr>
<tr>
<td>(Block of the sympathetic</td>
<td>o Severe head injury or intracranial vascular event</td>
</tr>
<tr>
<td>out flow resulting in</td>
<td>o Overdose</td>
</tr>
<tr>
<td>peripheral vasodilation)</td>
<td></td>
</tr>
<tr>
<td>SEPTIC AND ANAPHYLACTIC</td>
<td>o Vasodilation caused by humoral or toxic substances acting on blood</td>
</tr>
<tr>
<td></td>
<td>vessels.</td>
</tr>
<tr>
<td>OTHERS (Rare)</td>
<td>o Addison’s disease (adrenal gland failure)</td>
</tr>
<tr>
<td></td>
<td>o Myxedema coma (thyroid gland failure)</td>
</tr>
</tbody>
</table>
A simple mnemonic which stresses the important causes of shock is outlined below:

S  Septic, Spinal (Neurogenic)
H  Hypovolemic (+/- Hemorrhagic)
O  Obstructive (Mechanical)
C  Cardiogenic
K  Anaphylactic “k”

A similar and acceptable approach is to think of shock as secondary to dysfunction of one of three major factors.

FIGURE 2: AN APPROACH TO SHOCK

2A. FACTORS MAINTAINING TISSUE PERFUSION

Pump

Vessels ———> Volume

2B. DYSFUNCTIONS IN FACTORS MAINTAINING TISSUE PERFUSION

Inadequate pumping action of the heart

Example

Cardiogenic shock due to acute myocardial infarction

Inadequate peripheral resistance  Inadequate circulating volume

Example

Vasogenic Shock due to Anaphylaxis  Hypovolemic Shock due to Hemorrhage
TYPES OF SHOCK

MECHANICAL/ OBSTRUCTIVE SHOCK

It is important to note that the mechanical obstruction of blood flow may result in shock. A trauma patient with a tension pneumothorax must be diagnosed rapidly as therapy is specific (release of air from within the pleural space) and dramatic.

With a tension pneumothorax, the raised intrathoracic pressure impedes venous return, and the shift of the mediastinum may contribute to impaired cardiac function. A massive pulmonary embolus impedes blood flow to the lungs and subsequently to the left ventricle. Cardiac tamponade results in impaired diastolic filling since fluid/blood in the pericardial sac compresses the heart. A dissecting aortic aneurysm can obstruct blood flow distal to the left ventricle and result in widespread tissue hypoperfusion and hypoxia.

An important physical finding with both mechanical shock and cardiogenic shock is distended neck veins indicating raised central venous pressure. Raised central venous pressure rules out hypovolemia as the cause of the shock state (at least in isolation) and should raise suspicion of a mechanical or cardiac etiology.

SEPTIC SHOCK

Septic shock is precipitated by the release of endotoxins by microorganisms (usually gram negative bacteria) into the bloodstream. This results in decreased vascular resistance, peripheral pooling of blood and ultimately capillary leak with fluid extravasation. In sepsis, the effective blood volume is low relative to the size of the “tank” and the end result is a decreased blood supply to major organs and ultimately organ failure. Most commonly seen in the elderly with underlying medical illnesses such as diabetes or cancer as well as in the very young. The signs and symptoms are variable in the early and last stages of sepsis as outlined in Table 2. Clinical management is based on use of inotropes (drugs that increase cardiac contractility) or vasopressors and/or fluid volume resuscitation with normal saline of Ringers Lactate. Hospital or critical care treatment would include the use of antibiotics, steroids and general supportive measures. Mortality is approximately 45%1.
CLINICAL VIGNETTE

Suspect septic shock with the following: Recent known infection, urinary catheterization, pneumonia, infected surgical wound, fever or unexplained hypothermia, hypotension (<90 mmHg systolic) that may not respond to fluid resuscitation, tachycardia, tachypnea.

CARDIOGENIC SHOCK

Cardiogenic shock is the most severe manifestation of decreased left ventricular pumping function. Cardiogenic shock develops because the left ventricle is unable to maintain the cardiac output at a level necessary for adequate tissue perfusion. The majority of patients with cardiogenic shock will have had a massive left ventricular infarction. Other potential causes are a right ventricular contusion (occurring with deceleration injuries) in a trauma patient resulting in damage to one of the AV valves, or a dysrhythmia (e.g. unstable tachydysrhythmias such as ventricular tachycardia, atrial fibrillation, etc).

FIGURE 3: CARDIOGENIC SHOCK IN THE SETTING OF MYOCARDIAL INFARCTION

- **EXTENSIVE LEFT VENTRICULAR DAMAGE**
- **ADDITIONAL MYOCARDIAL DAMAGE**
- **DECREASED CARDIAC OUTPUT**
- **INADEQUATE TISSUE PERFUSION**
- **FALL IN ARTERIAL BLOOD PRESSURE**
- **GENERALIZED VASOCONSTRITION** (resulting in increased afterload)
- **EVENTUALLY COMPENSATION FAILS** (leading to profound vasodilation)

CLINICAL MANIFESTATIONS OF CARDIOGENIC SHOCK

Hypotension, altered mental status, cold moist skin, metabolic acidosis.
In the setting of acute myocardial infarction (AMI), most patients who die of cardiogenic shock usually have more than 40% of the myocardium destroyed (not necessarily from the initial infarction). The infarcted area continues to enlarge during the course of cardiogenic shock and since blood flow through the coronary arteries decreases during shock, the myocardium is further deprived of oxygen. This further impairs myocardial contractility of the uninjured ventricular segment and at the same time promotes additional tissue destruction. A vicious cycle is set up which explains the high mortality (>80%) from cardiogenic shock in the setting of AMI.

**ACUTE RIGHT VENTRICULAR INFARCTION - SHOCK**

Right ventricular (RV) infarction occurs in approximately 25-30% of patients who experience an acute inferior left ventricular infarction. Most often this results from a proximal occlusion of the right coronary artery (RCA) which feeds the SA node, AV node, right ventricle and the inferior left ventricle in 90% of the population. Consequently, the right ventricle is not able to contract effectively and the result is hypotension and cardiogenic shock. However, unlike the cardiogenic shock that most of us envision, the clinical presentation of RV infarction has all of the typical elements of shock such as hypotension, altered mental status, shortness of breath, but pulmonary edema is absent, the neck veins are generally distended and tachycardia may be absent, and in fact the patient may be bradycardic. This occurs because the SA node may be ischemic or excess vagal tone may blunt the reflex tachycardia typically seen in shock.

---

**Clinical vignette**

Acute Right Ventricular Infarct: Signs & symptoms consistent with cardiac ischemia/AMI, hypotension, bradycardia or normal heart rate, SOB, **clear chest**, distended neck veins.

12 Lead ECG: look for ST elevation in the inferior Leads (II, III, aVF) and ST elevation in RV4.

Note: Primary Care Paramedics are encouraged to learn 12 Lead ECG interpretation as ECG changes may be seen in the prehospital setting that may be of value for hospital staff.

* Adapted from Denny, M.P.: Septic Shock, JEN 3:19, Jan-Feb 1977.
** Adapted from “Therapeutic Considerations in Critical Care Medicine, Hemodynamic and Respiratory Aspects of Shock”, Kalamazoo, Michigan, 1976, The Upjohn Co.
ANAPHYLACTIC SHOCK

Anaphylaxis is an acute, severe, systemic allergic reaction caused by the release of chemical mediators (histamine, prostaglandins, leukotrienes and kinin) after an interaction with IgE antibodies on the surface of mast cells and basophils. These chemical mediators result in widespread vasodilation (predominant cause of shock), capillary leak with decreased blood volume and tissue swelling, bronchospasm, increased mucus production and shock. The most common causative agents are medications (especially after parenteral administration), foods and insect stings/bites. Simple allergic reactions may take minutes to hours, however anaphylaxis typically occurs within minutes. A high index of suspicion is therefore necessary and careful history may reveal an allergy history.

Anaphylaxis can cause significant organ compromise and death within minutes.

The primary systems involved in anaphylactic shock are:

1. Respiratory
   - Upper airway obstruction secondary to edema
   - Bronchospasm secondary to bronchoconstriction.

2. Integumentary
   - Urticaria (hives)
   - Local swelling

3. Gastrointestinal
   - Nausea, vomiting and diarrhea secondary to chemical mediator release and sympathetic nervous system stimulation.

4. Circulatory
   - Widespread vasodilation leading to hypotension resulting in circulatory collapse.

The mainstay of treatment for anaphylactic shock is the administration of epinephrine to counteract the widespread vasodilation, reverse the mediator response and decrease airway swelling and bronchospasm. Fluid resuscitation and the use of antihistamines and steroids may also be appropriate.

Note: Further treatment with salbutamol for wheezing may be appropriate if bronchospasm does not respond adequately to epinephrine.

HEMORRHAGIC SHOCK

As emphasized previously, not all shock is hemorrhagic. A frequent mistake is to assume that the trauma patient is in shock from hypovolemia (as opposed to a tension pneumothorax) or
that an elderly confused, alcoholic, male is in shock from a GI bleed (as opposed to septic shock). It is important to rule out other potential causes for shock which may have specific treatment. The module on hypovolemia and its management will focus on this problem.

Conversely, if a patient with a severe head injury is in shock, it is much more likely due to hypovolemia than the head injury and should be treated as such.

Table 3 is useful in that it correlates physical findings with the approximate blood loss. Calculations are based on a 70 kg male with a blood volume of 70 mL/kg. The physical findings may overlap between different classes of shock, and the principles are more important than specific numbers.

<table>
<thead>
<tr>
<th>TABLE 3</th>
<th>SEVERITY OF HEMORRHAGIC SHOCK*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CLASS I</td>
</tr>
<tr>
<td>Blood loss in mL</td>
<td>Up to 750 mL</td>
</tr>
<tr>
<td>Blood loss in %</td>
<td>Up to 15%</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>72-84</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>118/82</td>
</tr>
<tr>
<td>Capillary blanch test</td>
<td>Normal</td>
</tr>
<tr>
<td>Respiratory Rate</td>
<td>14-20</td>
</tr>
<tr>
<td>CNS – mental status</td>
<td>Slightly anxious</td>
</tr>
<tr>
<td>Fluid replacement</td>
<td>Crystalloid</td>
</tr>
</tbody>
</table>

(Use 3:1 rule for fluid resuscitation)

Clinical vignette

OXIMETRY: Remember that the SpO₂ in a hypovolemic patient may be normal. However, because of blood loss (loss of hemoglobin), the body’s oxygen carrying capacity will be reduced. Therefore, supplemental oxygen is critical in the setting of hypovolemia to increase the amount of dissolved oxygen in blood plasma.

* Adapted from the American College of Surgeons. Classification of Hemorrhagic Shock, Advanced Trauma Life Support Course.
COMPENSATORY MECHANISMS IN SHOCK

DEFINITION

When a patient is in shock, the body will attempt to maintain an adequate blood flow to vital organs (brain, heart, kidneys), to maintain cardiac output and tissue perfusion.

There are three major mechanisms employed to achieve this:

- Nervous
- Chemical
- Hormonal

If vital organ perfusion is adequate the shock is said to be compensated. This is, however, an unstable hemodynamic situation which can deteriorate with time or additional stress on the system, i.e. further blood loss, myocardial dysfunction secondary to prolonged ischemia.

COMPENSATORY MECHANISMS EMPLOYED

1. **Neurogenic compensation** is the most rapid and is known as the fight or flight response. It consists primarily of arterial and venous vasoconstriction (in an attempt to maintain an adequate perfusion pressure) and an increased heart rate (in an attempt to maintain cardiac output). This means that there is preferential perfusion of the brain and heart.

2. **Chemical compensation** occurs within thirty minutes. The decreased cardiac output and increased oxygen extraction by the tissues leads to a decreased arterial PaO₂ causing the chemoreceptors in the aorta and carotid arteries to stimulate the respiratory centre. The respiratory centre responds with a respiratory alkalosis. Unfortunately this leads to vasoconstriction of cerebral vessels, cerebral ischemia and changes in level of consciousness.

3. **Hormonal compensation** can occur when impulses arrive via the sympathetic nervous system. Three major types of hormonal compensation can result:

   a) The adrenal medulla is stimulated to release its hormones (epinephrine/norepinephrine – potent vasoconstrictors).

   b) Decreased blood flow to the kidneys leads to the activation of the Renin-Angiotensin system. Renin converts angiotensinogen (protein) into angiotensin I. Increased angiotensin I passes through the lungs where an enzyme called angiotensin converting enzyme (ACE) converts angiotensin I to angiotensin II. The hormone angiotensin II is a powerful vasoconstrictor and it also stimulates the adrenal cortex to release aldosterone. Aldosterone causes the kidneys to
retain sodium. The retained sodium results in an increased intravascular volume, thereby increasing systemic perfusion.

c) The hypothalamus stimulates the anterior pituitary to secrete adrenocorticotrophic hormone (ACTH) which in turn stimulates the release of adrenal cortical hormones (glucocorticoids). These hormones influence the metabolism of carbohydrates, proteins and fats, and decrease the permeability of capillary walls. This helps to limit the loss of intravascular fluid.

It should be noted that these compensatory mechanisms are time-limited, and although attempting to improve vital organ perfusion, result in a further widespread reduction in tissue perfusion secondary to generalized vasoconstriction. These compensatory mechanisms are less efficient in infants and toddlers, with advancing age or with underlying concurrent illnesses.

SUMMARY

As noted in the discussion above, shock may be considered as a spectrum from early reversible shock to late irreversible shock. Each patient must be considered as an individual and the ultimate outcome will depend on the past health, age, pre-existing illnesses, etc.

Assessment of the patient will focus not only on diagnosing the presence of shock but also on elucidating the cause(s) so that appropriate therapeutic interventions may be undertaken. As noted above, certain specific treatments, e.g. pericardiocentesis, may only be carried out in a hospital setting and rapid transport may be the most appropriateprehospital management.

The principles of assessment, and management (oxygenation and management of airway, hypovolemia, selected emergencies) will be covered in other modules.

The major principles of resuscitation for the patient in shock are to:

1. Improve cellular perfusion
2. Improve cellular oxygenation.

REFERENCES

ADVANCED LIFE SUPPORT
PRE COURSE
SHOCK

SELF-ASSESSMENT

Marks
[2] 1. a) The clinical aim of shock therapy is to restore two processes at the cellular level. What are they?
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________

[2]  b) What is the relationship between these processes?
___________________________________________________________________________

[4½ ] 2. For each of the three etiologies of shock shown below (Pump, Vessels, Volume), list three possible causes for shock. (1/2 mark for each correct answer).

PUMP:___________________________________________________________________________

VESSELS:___________________________________________________________________________

VOLUME:___________________________________________________________________________

[3]  3. The cell prefers to utilize a) ______________________ and b) _______________ to make c) _________________________________.

OBHG Education Subcommittee
4. Briefly explain the etiology of the metabolic acidosis seen with shock.

5. Explain the statement “hypotension and shock are NOT synonymous”.

6. What is the approximate total blood volume of a healthy 100 kg man?

7. Slight tachycardia and mild anxiety may be your only clues to early detection of an occult bleed. Why?

8. a) Which four primary body systems are affected in anaphylactic shock?

   b) Name one clinical manifestation for each system affected.
Marks

[1] 9. a) One physical finding will rule out hypovolemia as a cause of shock. This is _________________.

[2] b) This finding is associated with ________________ or ______________ causes of shock.

[1] 10. Clinical findings particular to cardiogenic shock are:

___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________

[4] 11. The patient’s ability to compensate for shock is influenced by ________________,
    ________________, ________________, and _________________.

31 ½ TOTAL
ADVANCED LIFE SUPPORT
PRE COURSE
SHOCK

SELF-ASSESSMENT ANSWERS

1. a) Oxygenation and perfusion.
   b) Oxygenation is dependent upon delivery of oxygen to the blood via the respiratory system and the ability of the hemoglobin to transport it to the cell.

2. Pump: a) MI.
   b) Myocardial contusion
   c) Dysrhythmia

Vessels: a) Sepsis
   b) C-spine injury
   c) Anaphylaxis

Volume: a) GI losses
   b) Renal losses
   c) Hemorrhage

3. a) O₂
   b) Glucose
   c) ATP or energy

4. Subtract one mark for each key concept missed to zero.

*Decreased oxygen delivery* to cells results in *metabolism without oxygen: glycolysis* leads to *pyruvate* production, which is metabolized to *lactic acid*, creating metabolic acidosis.

5. What is of note is whether the total clinical picture of the patient indicates that his cells are oxygenated and perfused.

6. 7 litres (7,000 mL)

7. Neurogenic (SNS) compensation or the “fight or flight” response is responsible for the vasoconstriction, tachycardia and anxiety.
8. (1/2 mark for each correct answer)
   
   a)  ○ Respiratory
       ○ Integumentary
       ○ Gastrointestinal
       ○ Circulatory
   
   b)  ○ Bronchospasm or edema
       ○ Hives
       ○ Nausea, vomiting, or diarrhea
       ○ Hypotension

10. a)  Jugular venous distension OR distended neck veins.

       b)  Cardiogenic or mechanical (obstructive).

11. (1/2 mark for each correct answer)

       Pulmonary edema, JVD

12. Duration of shock
    Severity of shock
    Age
    Concurrent illness
ADVANCED LIFE SUPPORT
PRECOURSE
SHOCK

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.
   Reading ________ hours
   Self assessment ________ hours
   Total time ________ hours

2. Were the objectives of the module clearly stated?
   [    ] yes [    ] no
   If no, please comment.

3. Did you see any of the resource materials?
   [    ] yes [    ] no
   If yes, which items
   _____________________________________________________________
   _____________________________________________________________
   _____________________________________________________________
   Were they helpful?
   _____________________________________________________________

4. Were the reference notes adequate?
   [    ] yes [    ] no
   If no, please comment.

5. Were the reference notes easy to follow?
   [    ] yes [    ] no
If no, please comment.

6. Were the examples provided satisfactory?

[  ] yes [  ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[  ] yes [  ] no
If yes, please specify.

8. Was the level of the module satisfactory for your program of study?

[  ] yes [  ] no
If no, please comment.

Base Hospital

9. General comments or suggested improvements.
OBJECTIVES: FLUIDS & ELECTROLYTES

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. Name and describe the body fluid compartments.

2. Describe the distribution of body fluid in the intracellular and extracellular compartments.

3. Define and state the role of the following in fluid and electrolyte movement.
   a) Diffusion
   b) Active Transport
   c) Osmosis
   d) Osmotic Pressure
   e) Oncotic Pressure (colloidal osmotic pressure)

4. State the approximate volume of body water in the normal adult.

5. State the distribution (as % of body weight) of fluid volumes in the normal adult and the pediatric patient.

6. Identify significant fluid loss (as % of body weight) in the adult and the pediatric patient.

7. Briefly explain the use of normal saline and 5% D/W in management of fluid depletion.

8. Describe briefly the role of the kidney (including the hormones involved) in the maintenance of fluid and electrolyte balance.

9. Define:
   a) Dehydration
   b) Edema
   c) Volume depletion.

10. State the physiological roles of Na+, K+, Cl-, Ca++ and HCO3-.

If you have studied this subject previously, you may test your ability using the self-assessment questions at the end of each section. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the section, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.
<table>
<thead>
<tr>
<th><strong>GLOSSARY</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>ACTIVE TRANSPORT</td>
<td>The passage of molecules or ions across a semi-permeable</td>
</tr>
<tr>
<td></td>
<td>membrane against the concentration gradient, i.e. from an area</td>
</tr>
<tr>
<td></td>
<td>of lower to higher concentration of that substance; requires</td>
</tr>
<tr>
<td></td>
<td>expenditure of energy.</td>
</tr>
<tr>
<td>ANION</td>
<td>An ion with a negative (-) charge</td>
</tr>
<tr>
<td>ANOXIA</td>
<td>Absence of oxygen in the body tissues</td>
</tr>
<tr>
<td>CATION</td>
<td>An ion with a positive (+) charge</td>
</tr>
<tr>
<td>COLLOID</td>
<td>Molecules which are invisible to the naked eye but are too large</td>
</tr>
<tr>
<td></td>
<td>to dissolve and are therefore suspended in solution. In the</td>
</tr>
<tr>
<td></td>
<td>blood, this refers to large protein molecules.</td>
</tr>
<tr>
<td>CONCENTRATION GRADIENT</td>
<td>The tendency for substances to move from an area of higher to</td>
</tr>
<tr>
<td></td>
<td>lower concentration with two solutions of different concentrations are separated by a semi-permeable membrane.</td>
</tr>
<tr>
<td>DEHYDRATION</td>
<td>A condition in which there is a net loss of water from the fluid</td>
</tr>
<tr>
<td></td>
<td>compartments of the body.</td>
</tr>
<tr>
<td>DIFFUSION</td>
<td>The movement of molecules of ions from an area of higher to</td>
</tr>
<tr>
<td></td>
<td>lower concentration of that substance, i.e. with the</td>
</tr>
<tr>
<td></td>
<td>concentration gradient; a passive transport system.</td>
</tr>
<tr>
<td>EDEMA</td>
<td>A condition in which the interstitial (tissue) spaces contain</td>
</tr>
<tr>
<td></td>
<td>an excessive amount of extracellular fluid; characterized by</td>
</tr>
<tr>
<td></td>
<td>swelling and puffiness of the tissues.</td>
</tr>
<tr>
<td>ELECTROLYTE</td>
<td>A substance capable of dissociating into ions and which in</td>
</tr>
<tr>
<td></td>
<td>solution will conduct an electrical current.</td>
</tr>
<tr>
<td>HOMEOSTASIS</td>
<td>The maintenance of a steady state in the body, and a relatively</td>
</tr>
<tr>
<td></td>
<td>constant concentration of ions, pH, and osmotic pressure in the</td>
</tr>
<tr>
<td></td>
<td>various body fluids.</td>
</tr>
<tr>
<td>EXTRACELLULAR FLUID (ECF)</td>
<td>A solution, primarily saline (NaCl), which occupies the areas</td>
</tr>
<tr>
<td></td>
<td>outside the cells. (&quot;extra&quot; = outside; &quot;cellular&quot; = the cells)</td>
</tr>
<tr>
<td>INTRACELLULAR FLUID (ICF)</td>
<td>A solution of water, electrolytes, and proteins which circulates</td>
</tr>
<tr>
<td></td>
<td>within the cells (&quot;intra&quot; = inside; &quot;cellular&quot; = the cells)</td>
</tr>
</tbody>
</table>
**GLOSSARY**

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>INTERSTITIAL FLUID (ISF)</td>
<td>Extracellular fluid composed of water and electrolytes which circulates between and around the cells. (“inter” = between; “stitial” = spaces)</td>
</tr>
<tr>
<td>INTRAVASCULAR FLUID (IVF)</td>
<td>Extracellular fluid composed of water, electrolytes and proteins which is contained within the blood vessels. (“intra” = inside; “vascular” = blood vessels)</td>
</tr>
<tr>
<td>METABOLIC ACIDOSIS</td>
<td>A disturbance of the acid-base balance in which the body pH decreases (becomes more acidic) due to decreased blood levels of bicarbonate ions.</td>
</tr>
<tr>
<td>METABOLIC ALKALOSIS</td>
<td>A disturbance of the acid-base balance in which the body pH increases (becomes more alkaline) due to increased blood levels of bicarbonate ions.</td>
</tr>
<tr>
<td>ONCOTIC PRESSURE</td>
<td>Osmotic pressure due to the presence of plasma colloids.</td>
</tr>
<tr>
<td>OSMOSIS</td>
<td>The passage of water (solvent) across a semi-permeable membrane from a more dilute solution (lower solute concentration, higher water concentrations) to a more concentrated solution (higher solute concentration, lower water concentration), i.e. with the concentration gradient; a passive transport system.</td>
</tr>
<tr>
<td>OSMOTIC PRESSURE</td>
<td>The amount of pressure that would be required to prevent the movement of water (osmosis) across semi-permeable membrane when two solutions of different concentrations are separated by that membrane.</td>
</tr>
<tr>
<td>PASSIVE TRANSPORT</td>
<td>The movement of ions or molecules in the direction of the concentration gradient, thereby requiring no energy expenditure.</td>
</tr>
<tr>
<td>SEMI-PERMEABLE MEMBRANE</td>
<td>A membrane that only permits the passage of certain molecules and not others.</td>
</tr>
<tr>
<td>SKIN TURGOR</td>
<td>The degree of tension of the skin.</td>
</tr>
<tr>
<td>SOLUTE</td>
<td>The substance/compound that is dissolved in a solution.</td>
</tr>
<tr>
<td>SOLVENT</td>
<td>The liquid in which another substance (the solute) is dissolved to form a solution.</td>
</tr>
</tbody>
</table>
FLUID AND ELECTROLYTE BALANCE

Body cells must have fluids and electrolytes available to them in order to function normally. All of the body's processes (respiration, metabolism, digestion, excretion, etc.) are affected by the volume of body fluid present as well as its specific composition, i.e. the concentrations of the various electrolytes and other solutes. Disturbances in the fluid or the electrolyte balance may lead to cellular dysfunction and can seriously jeopardize a patient's life. These imbalances occur during many illnesses, usually due to the loss of fluids and/or electrolytes, e.g. by vomiting, diarrhea, fistulas, excessive urination, etc. Although the fluid or electrolyte imbalance itself is rarely a direct cause of death, it certainly contributes to the seriousness of an illness. It is therefore important to understand how water and electrolytes are distributed in the body, as well as the mechanisms involved in maintaining the body's normal water and electrolyte balance.

DISTRIBUTION OF BODY FLUID

Body fluid (mainly water) accounts for about 60% of the total body weight of an adult, but this varies with fat content and with age. A fat person has proportionally less body water than a thin person (Figure 1). In infants, body fluid comprises about 80% of total body weight, while in the elderly, there is a decrease in total body water (Figure 2).

The amount of body fluid in an adult varies from 45%-75% depending on the fat content of the body. Fat tissue is essentially water-free. An obese person has the same volume of body water as a leaner person, therefore it represents a lower percentage of his total body weight. Thus, there is an inverse relationship between body water (as % of body weight) and fat content.
The thin man pictured in Figure 1 contains less fat and therefore more water in proportion to his total body weight. The fat man contains proportionally less water as a percentage of his body weight because of the large amount of adipose (fat) tissue present.

In the newborn, body fluid comprises about 80% of the total body weight, with more than one half of this as extracellular fluid. As the child grows, proportions and total volume gradually approximate the adult fluid distribution.

Water is found both inside and outside the cells, and is usually considered to be divided into two main "compartments" or spaces. Of the total body water, approximately 2/3 of the water is contained inside the cells themselves. This water is collectively referred to as the INTRACELLULAR FLUID (ICF), and is said to be found in the intracellular compartment. The remaining 1/3 of total body water is distributed throughout the body as the EXTRACELLULAR FLUID (ECF). This refers to all the fluid found outside the cells and is said to be located in the extracellular compartment. The extracellular fluid is further divided into two main categories. The INTRAVASCULAR FLUID (IVF) is that part of the extracellular fluid located within the blood vessels as blood plasma. The remainder of the extracellular fluid, referred to as the INTERSTITIAL FLUID (ISF), is found between the cells and the blood vessels and in tissue spaces.
It is important to understand that the term "compartment" does not refer to one specific contained space, like an organ, but rather it is a convenient abstraction used to describe where fluid is found throughout the body.

The relative distribution of body fluid is summarized as follows:

- The average adult has approximately 42 liters of body water, which represents about 60% of his/her total body weight.  
- Of this 60%, 40% (28 L) is present as intracellular fluid (ICF) and 20% (14 L) is present as extracellular fluid (ECF).  
- The extracellular fluid can be further divided as intravascular fluid or plasma 5%, (3.5 L) and interstitial fluid, 15% (10.5 L).
MOVEMENT OF FLUIDS AND ELECTROLYTES

The various fluid "compartments" of the body are separated by semi-permeable (or selectively-permeable) membranes. These membranes allow some molecules to pass through freely, while restricting or preventing the passage of other molecules. In health, the total volume and composition of the fluid in each compartment remains remarkably stable in spite of the fact that the water and solute molecules are in constant motion, moving from one compartment to another. There are several mechanisms by which this movement may occur.

DIFFUSION

Many solute molecules move between fluid compartments by means of simple diffusion. This term refers to the natural tendency of all substances to move about in a solution in an effort to distribute themselves evenly throughout the solution. If a membrane is permeable to a certain substance, i.e. if it allows free passage of that substance across it, then those molecules will move through the membrane in an effort to equalize their concentration on either side of the membrane.

![Diffusion Diagram](image)

The direction of movement in diffusion is said to be "with the concentration gradient". This means that molecules follow the natural tendency to equalize concentration by moving from an area of higher concentration to an area of lower concentration of that substance. Because it follows the "natural" direction of flow, no energy is required for simple diffusion to take place. Therefore, diffusion is referred to as a passive transport system.

Diffusion, then, is a general term referring to the passive movement of any substance from an area of higher to lower concentration of that substance. When discussing biological systems, the term diffusion is usually used loosely in reference to the movement of solute molecules. Of course, solvent molecules diffuse as well. In fact, in the human body, the majority of molecules diffusing across semi-permeable membranes are solvent molecules, e.g.
water. Because the diffusion of water molecules is such an important process, it is given the special name of osmosis.

**OSMOSIS**

In osmosis, water moves across a semi-permeable membrane from the more dilute solution (lower solute concentration) to the more concentrated solution (higher solute concentration). It may appear at first glance that this is a contradiction of the earlier statement that diffusion is movement from an area of higher to lower concentration, i.e. with the concentration gradient. However, remember that the definition refers to the concentration of the substance that is doing the diffusing.

The process of osmosis does occur in the direction of the concentration gradient since water is moving from an area of higher concentration of water (more dilute solution; lower solute concentration) to an area of lower concentration of water (more concentrated solution; higher solute concentration). Because movement is with the concentration gradient, osmosis is also a passive transport system, i.e. no energy required. Therefore, osmosis refers specifically to the movement of water (solvent) molecules across a semi-permeable membrane from an area of higher to lower concentration of water (lower to higher concentration of solute). Osmosis is illustrated in Figures 5 and 6.

In Figure 5, solution “A” has fewer solute particles per unit volume than solution “B”. Therefore, solvent molecules move from solution A to solution B. Osmosis occurs until the number of solute particles per unit volume is the same on both sides of the membrane.

![Figure 5: Osmosis](image)

The movement of solvent results in diluting the number of solute particles per unit volume in solution B. When the number of solute particles per unit volume is the same on both sides of the membrane, the movement of solvent molecules occurs equally in both directions, i.e. a dynamic equilibrium exists (Figure 6). The two solutions are said to be isotonic to each other.
Osmosis and diffusion occur simultaneously. The processes work together in an attempt to equalize the concentrations and balance the osmotic pressures of the two solutions by moving solute out of the more concentrated solution and water out of the more dilute solution.

When two solutions of differing concentrations are separated by a semi-permeable membrane there is a 'pulling force' which draws water through to the more concentrated side, i.e. higher solute concentration. The amount of pressure that would be required to prevent this movement of water is referred to as the **osmotic pressure** of a solution.

The osmotic pressure is determined by the number of particles of solute on the more concentrated side, relative to the side with the lower concentration. The greater the number of particles in the concentrated solution, the more 'pull' there will be to draw the water through the membrane and therefore, the greater the pressure required to prevent that movement.

In Figure 7, solution A has fewer solute particles per unit volume (lower osmotic pressure) than solution B. Solvent will move from solution A to solution B, unless pressure is applied to solution B to prevent osmosis.
The osmotic pressure due to plasma colloids (protein molecules mainly) is specifically referred to as the **COLLOIDAL OSMOTIC PRESSURE** or **ONCOTIC PRESSURE**.

**ACTIVE TRANSPORT**

The processes of diffusion and osmosis follow the natural tendency of molecules or ions to move from areas of higher to areas of lower concentrations in an attempt to equalize concentrations. However, it is often necessary for the body to move substances in the opposite direction, against the concentration gradient, in order to maintain a higher concentration of a substance on one side of a membrane. The process which moves substances against the concentration gradient is referred to as **ACTIVE TRANSPORT**.

In order to move substances from an area of lower concentration to an area of higher concentration, a carrier substance* is often needed (Figure 8). For example, insulin acts as a carrier substance to transport glucose across the cell membrane, from the blood (ECF) into the cell (ICF).

Energy, in the form of adenosine triphosphate (ATP), must be expended to facilitate the transport (hence the term "active transport").
Therefore, active transport refers to the movement of molecules or ions from an area of lower concentration to an area of higher concentration of that substance, against the concentration gradient. Some important substances actively transported in the body include ions of sodium, potassium, chloride, hydrogen, calcium and iron, amino acids, and some sugars.

All of the processes discussed are responsible for carrying the molecules of water, foods, gases, wastes, and many kinds of ions between compartments, in and out of the body's cells. Together, they act to maintain a proper chemical and osmotic balance between the intracellular and extracellular fluids.

* The carrier molecule undergoes a structural change as it picks up the substance to be transported. This change requires energy.

**FLUID LOSS**

In order to maintain fluid balance in the body, the daily intake of fluids must match the daily output. The kidneys excrete the largest quantity of fluid, but fluid also leaves the body through the lungs, skin and gastrointestinal tract. Fluid is replenished in the body by ingestion of liquids and by digestion of foods.

There is a basic minimum daily requirement for fluid of **approximately 2500 mL**. In the healthy individual the total intake by all routes is equal to the total output by all routes.

Injury or disease can cause abnormal increases in water loss and seriously upset the fluid and electrolyte balance of the body. Water loss via the lungs and skin is increased in conditions causing fever or an increased respiratory rate, in hot or dry environments and in skin injuries, e.g. burns. Water loss via the kidneys is increased in conditions involving increased solute excretion such as diabetes mellitus and in conditions in which there is a decrease in antidiuretic hormone (ADH) levels. Serious water and electrolyte loss can also occur from the G.I. tract in the case of severe vomiting or diarrhea.
As previously mentioned, the total fluid volume of the average adult is approximately 42 liters - 28 liters as intracellular fluid (ICF) and 14 liters as extracellular fluid (ECF). Any change in the amount of composition of these fluids may cause serious problems. In the adult, a water loss of 5% by body weight (≈ 2 L) is considered unfavourable, a loss of 10% (≈ 4 L) is considered serious, and a loss of 20% (≈ 8 L) is usually fatal.

Infants and small children need a proportionately larger fluid intake and output in relation to adults since they have a greater body surface area in proportion to mass and an increased metabolic rate. Also, infants have immature kidneys which require proportionately more water to excrete metabolic wastes. The younger the child, the smaller his fluid reserve, and therefore the greater his vulnerability to water deficit. Volume depletion in infants and small children can also be estimated based on body weight loss. A water loss of 2-4% by body weight is considered mild, a loss of 5-9% is considered moderate, and a water loss of over 10% is considered to be severe.

### Clinical vignette

1. Normal saline is used to fill the intravascular space for patients who are hypovolemic or hypotensive. Because normal saline is isotonic, it will equilibrate in both the intravascular and extravascular space. Hypovolemic patients have first lost fluid from the intravascular space. Fluid is then shifted from the extravascular space into the intravascular space in an attempt to maintain blood pressure. If necessary, intracellular fluid will shift into the extracellular space as well. In fluid resuscitation, only one third of normal saline will remain in the vascular space.

2. 5% dextrose in water (D5W) is a hypotonic solution and therefore is not held within the intravascular space. Rather it diffuses into the extravascular space where the sugar is metabolized by the cells. It is used as a vehicle for administering certain drugs and should not be used in patients who require fluid resuscitation.
ROLE OF THE KIDNEY IN FLUID MOVEMENT

More than any other organ in the body, the kidneys play a major role in regulating fluid and electrolyte balance. This balance or ‘homeostasis’ is maintained and restored by adjusting the output of these substances according to their intake.

When the extracellular fluid (ECF) volume is too low (due to increased loss or inadequate intake) the kidneys respond by retaining more fluid (excreting less urine). When the extracellular fluid volume is too high, the kidneys respond by excreting more urine. Exactly how this occurs can be understood by examining the effect of ADH and aldosterone on the kidney tubules, (Figures 9 and 10).

**FIGURE 9: ACTION OF ADH ON THE KIDNEY**

If the body is dehydrated (Figure 9A), the blood osmotic pressure increases (blood becomes more concentrated). The pituitary gland responds to high osmotic pressure by increasing its production of ADH. The more ADH that is secreted, the more water is retained.

If the body is overhydrated (Figure 9B), the blood osmotic pressure decreases (blood becomes more dilute). The pituitary gland responds to low osmotic pressure by decreasing its production of ADH. The less ADH that is secreted, the more water is lost.
If the body is dehydrated, there is a resultant decrease in blood volume and blood flow. This decrease in blood volume and flow is sensed by specialized cells in the kidney and results in the increased secretion of aldosterone from the adrenal glands. Aldosterone causes the kidneys to retain sodium (and with it, water) to correct the volume deficit. The reverse process occurs in the case of overhydration, resulting in the loss of sodium and water by the kidney.
CONDITIIONS OF FLUID IMBALANCE

VOLUME DEPLETION

Volume depletion is the loss of water from all fluid compartments of the body. As the degree of volume depletion increases, fluid is lost first from the interstitial fluid (ISF), then the intravascular fluid (IVF; plasma), and finally from the intracellular fluid (ICF). Volume depletion usually results from either an inadequate fluid intake or an excessive fluid loss. Some of these conditions are listed in Table 1.

<table>
<thead>
<tr>
<th>INADEQUATE FLUID INTAKE</th>
<th>EXCESSIVE FLUID LOSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>o  inability to swallow</td>
<td>o  chronic vomiting</td>
</tr>
<tr>
<td>o  coma</td>
<td>o  severe diarrhea</td>
</tr>
<tr>
<td>o  unavailability of water</td>
<td>o  diabetes mellitus</td>
</tr>
<tr>
<td>o  extreme debilitation and illness</td>
<td>o  diabetes insipidus</td>
</tr>
<tr>
<td>o  mechanical devices and intubation</td>
<td>o  kidney failure</td>
</tr>
<tr>
<td></td>
<td>o  fever</td>
</tr>
<tr>
<td></td>
<td>o  hemorrhage</td>
</tr>
<tr>
<td></td>
<td>o  hyperventilation</td>
</tr>
<tr>
<td></td>
<td>o  drainage from wounds and suctioning</td>
</tr>
<tr>
<td></td>
<td>o  burns</td>
</tr>
</tbody>
</table>

Two entirely different disorders can occur when one is assessing a patient with a fluid and/or electrolyte disorder:

o One is a disorder of total volume, e.g. volume overload, volume depletion
o The other is a disorder of water balance, e.g. water relative to salt, as in hyponatremia (↓Na+) or hypernatremia (↑Na+).

The diagnosis of volume status is made clinically, while the diagnosis of water balance disorders is made in a laboratory.

Dehydration is a vague term but in its strictest sense refers to a loss of water from the body.

When assessing a patient clinically we are concerned about his volume status and a patient is therefore more accurately described as being volume depleted rather than dehydrated (water depleted).
Volume depletion can be recognized by watching for changes in body temperature (increased), postural vital signs (decreased blood pressure, increased pulse rate), skin turgor, and by observing the appearance of the skin and the mucous membranes of the mouth for dryness.

Signs and symptoms of volume depletion to watch for include:

- thirst
- dry skin
- dry mucous membranes
- sunken eyes (especially in infants)
- sunken fontanel (infants)
- low grade fever
- increased pulse rate
- poor skin turgor (turgor in the skin of the forehead may be a better gauge of fluid status than the back of the hand in the elderly)
- hypotension
- altered mental status

**Clinical vignette**

The most reliable way of confirming volume depletion is by testing postural or orthostatic vital signs. The pulse and blood pressure are tested first with the patient in a supine position and then in an upright position. If volume depletion is present, the pulse will increase and the blood pressure will decrease when the patient is put in an upright position.

Especially susceptible to volume depletion are individuals with a relatively low proportion of total body water, such as infants, the elderly, and the obese. Volume depletion is potentially serious clinically because it involves not only a change in water balance, but a change in electrolyte balance as well (most importantly - sodium, potassium, chloride and bicarbonate).

**VOLUME OVERLOAD**

Edema refers to the presence of excess extracellular fluid (from plasma) in the interstitial spaces or fluid compartment. This abnormal accumulation of fluids produces noticeable swelling or "puffiness" in some tissues, particularly in the lower extremities or dependant areas of the body. By the time edema is noticeable, the adult patient will have accumulated about 4.5 kg (≈ 4.5 L) of extra fluid. Any condition which results in excessive retention of salt and water or in a decrease in plasma proteins (especially albumin) can lead to edema. In each case, excess water moves from the plasma into the tissues in an attempt to equalize the osmotic pressure. Some conditions leading to edema are listed in Table 2.
TABLE 2
CONDITIONS LEADING TO EDEMA

<table>
<thead>
<tr>
<th>LOW PROTEIN LEVELS</th>
<th>EXCESSIVE FLUID/SALT RETENTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>o malnutrition (↓ intake)</td>
<td>o heart disease e.g. congestive heart failure</td>
</tr>
<tr>
<td>o liver disease (↓ synthesis)</td>
<td>o kidney disease</td>
</tr>
<tr>
<td>o kidney disease (loss in urine) e.g. nephrosis</td>
<td>o pregnancy</td>
</tr>
<tr>
<td>o severe burns</td>
<td>o anti-diuretic medications</td>
</tr>
</tbody>
</table>

PHYSIOLOGICAL ROLES OF ELECTROLYTES

Body fluids contain two types of dissolved substances:

- those that dissociate or ionize in solution, called electrolytes, e.g. NaCl-
- those that do not dissociate, called non-electrolytes, e.g. glucose

The term electrolyte refers specifically to the fact that solutions of these substances will conduct an electric current.

Each of the fluid compartments of the body has a particular composition by electrolytes which are unique to that fluid. The principal extracellular electrolytes are ions of sodium, chloride, calcium, and bicarbonate, while the principal intracellular electrolytes are ions of potassium, magnesium, and phosphates and ionized proteins. In the extracellular fluid (plasma and ISF), the main cation is sodium and the main anion is chloride. Inside the cell (ICF) the main cation is potassium and the main anion is monohydrogen phosphate. The distribution of these electrolytes is summarized in Table 3.
TABLE 3
RELATIVE DISTRIBUTION OF ELECTROLYTES

<table>
<thead>
<tr>
<th>EXTRACELLULAR FLUID CATIONS (+)</th>
<th>ANIONS (-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium *Na&lt;sup&gt;+&lt;/sup&gt;</td>
<td>Chloride *Cl&lt;sup&gt;-&lt;/sup&gt;</td>
</tr>
<tr>
<td>Calcium Ca&lt;sup&gt;++&lt;/sup&gt;</td>
<td>Bicarbonate HCO&lt;sub&gt;3&lt;/sub&gt;-</td>
</tr>
<tr>
<td>Potassium K&lt;sup&gt;+&lt;/sup&gt;</td>
<td>Protein-</td>
</tr>
<tr>
<td>Magnesium Mg&lt;sup&gt;++&lt;/sup&gt;</td>
<td>Biphosphate PO&lt;sub&gt;4&lt;/sub&gt;&lt;sup&gt;2-&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Sulphate SO&lt;sub&gt;4&lt;/sub&gt;&lt;sup&gt;2-&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Organic acids-</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>INTRACELLULAR FLUID CATIONS (+)</th>
<th>ANIONS (-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium *K&lt;sup&gt;+&lt;/sup&gt;</td>
<td>Biphosphate *HPO&lt;sub&gt;4&lt;/sub&gt;&lt;sup&gt;2-&lt;/sup&gt;</td>
</tr>
<tr>
<td>Magnesium Mg&lt;sup&gt;2+&lt;/sup&gt;</td>
<td>Protein-</td>
</tr>
<tr>
<td>Sodium Na&lt;sup&gt;+&lt;/sup&gt;</td>
<td>Sulphate SO&lt;sub&gt;4&lt;/sub&gt;&lt;sup&gt;2-&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Bicarbonate HCO&lt;sub&gt;3&lt;/sub&gt;-</td>
</tr>
<tr>
<td></td>
<td>Chloride Cl&lt;sup&gt;-&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

* Major or dominant anions and cations in each fluid.

In clinical usage, the term electrolyte is used to refer to the four ions in plasma that most greatly affect water and acid-base balance (Na<sup>+</sup>, K<sup>+</sup>, Cl<sup>-</sup> and HCO<sub>3</sub>-), and increasingly, Ca<sup>++</sup>. These electrolytes profoundly influence water distribution, osmotic pressure, acid-base balance and neuromuscular irritability. Each has its own special functions in the body and although some play larger roles than others, all are necessary for the maintenance of health.

Sodium (Na<sup>+</sup>) is the major extracellular cation and therefore plays a major role in determining extracellular fluid volume and osmotic pressure. Because it represents about 90% of all the extracellular cations, it is especially important in the transmission of electrical impulses in nerve and muscle fibers. Since sodium ions are exchanged for hydrogen ions in the acidification of the urine, sodium has a role in pH regulation. It is also involved to some degree in regulating cell membrane permeability. Excessive levels of sodium ions in the blood (hypernatremia) produce symptoms of extreme muscle irritability, dry, sticky mucous membranes, flushed skin and intense thirst; while symptoms of low blood sodium levels (hyponatremia) include lethargy, muscle weakness, edema, decreased urinary output, and mental confusion leading to coma.

Potassium (K<sup>+</sup>) is the major intracellular cation, and it plays a major role in the regulation of muscle irritability. Potassium ions are crucial to the normal functioning of the heart muscle by allowing the muscle to contract properly and to rest properly in the diastolic phase (between contractions). K<sup>+</sup> is also somewhat associated with pH balance in that it is freely exchanged
with hydrogen ions when the body is responding to an acid-base disturbance. Both high and low blood potassium levels are potentially threatening because of their effect on cardiac muscle. High levels of potassium in the blood (hyperkalemia) cause heart arrhythmias (progression: peaked T \rightarrow flattening of the P waves \rightarrow QRS widening \rightarrow slowing of the heart rate, heart blocks, etc), weakening of cardiac contractility and eventually heart failure. Low blood potassium levels (hypokalemia) produce symptoms of cardiac excitability including tachycardia and ectopy (e.g. premature ventricular complexes), improper heart contractions, poor circulation, muscle cramps and eventually weakness and loss of muscle tone. Hypokalemia may also lead to cardiac arrest (usually due to anoxia created by paralysis of the respiratory muscles).

Chloride (Cl\(^{-}\)) is the major extracellular anion, and like sodium, plays an important role in the maintenance of extracellular fluid volume and osmotic pressure. The excretion and reabsorption of chloride ions is also related to acid-base regulation.

Calcium (Ca\(^{++}\)) is well-known for its important role in the formation of bones and teeth. It is a crucial factor in blood coagulation and in the activation of some enzymes. It also assists in the transmission of nerve impulses and the proper contraction of muscle fibers. It acts to decrease neuromuscular activity.

Bicarbonate (HCO\(_3\)^{-}\)) acts as a buffer-base in the bicarbonate/carbonic acid buffer system. This is the most important buffer in the blood and plays the key role in maintaining the body's acid-base balance. Bicarbonate ions (HCO\(_3\)^{-}\)) react with free hydrogen ions (H\(^{+}\)) in the body fluids to form undisassociated carbonic acid (H\(_2\)CO\(_3\)^{-}\)). In this way, acid (H\(^{+}\)) can be neutralized and carried to the kidneys for excretion. High levels of bicarbonate in the blood lead to a condition of metabolic alkalosis and symptoms of nausea, vomiting, diarrhea, confusion, irritability and agitation leading to coma. Low blood levels of bicarbonate lead to a condition of metabolic acidosis and the accompanying symptoms of headache, drowsiness, nausea, vomiting, diarrhea, stupor, and eventually, coma.
MARKS

1. Differentiate between:

[2] a) intracellular fluid (ICF) and extracellular fluid (ECF)

[2] b) intravascular fluid (IVF) and interstitial fluid (ISF)

2. Define the terms:

[1] a) osmotic pressure

[1] b) oncotic pressure.
3. Complete the following chart:

<table>
<thead>
<tr>
<th>Type of substance moving</th>
<th>Diffusion</th>
<th>Osmosis</th>
<th>Active Transport</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direction of movement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy requirements</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4. Fill in the blanks:

Body fluid accounts for about \((a)\) \% of the total body weight of an adult. Expressed in liters, the total fluid volume of the average adult is approximately \((b)\) \(L\). The amount of body fluid as a percentage of total body weight varies from person to person with \((c)\) and \((d)\). In infants, body fluid comprises about \((e)\) \% of total body water.

In disease conditions, water loss can be estimated based on percentage of body weight lost. In an adult, a fluid loss of \((f)\)% is considered serious, and a loss of 20% is usually fatal. Infants and small children are especially susceptible to volume depletion due to their smaller fluid reserve. In an infant, a fluid loss of \((g)\)% is considered moderate, while a loss of \((h)\)% is considered to be severe.

5. Name the two main hormones which act on the kidney to regulate fluid and electrolyte balance.
6. Define the terms:
   
a) dehydration

   
b) edema

   
c) volume depletion.

7. Complete the following chart:

<table>
<thead>
<tr>
<th></th>
<th>EXTRACELLULAR FLUID</th>
<th>INTRACELLULAR FLUID</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Cation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major Anion</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

8. State two physiological functions for each of the following:
   
a) sodium

   
b) potassium
c) chloride


d) calcium


30 Total
ADVANCED LIFE SUPPORT  
PRE COURSE  
FLUIDS AND ELECTROLYTES

SELF-ASSESSMENT ANSWERS

1. a) Intracellular fluid: The fluid which circulates inside the body’s cells.
Extracellular fluid: The fluid which circulates outside the body's cells.

b) Intravascular fluid: The portion of extracellular fluid located within the blood vessels (blood plasma).
Intestinal fluid: The portion of extracellular fluid found between the cells and blood vessels and in the tissue spaces.

2. a) Osmotic Pressure: The amount of pressure that would be required to prevent the movement of water (osmosis) across a semi-permeable membrane when two solutions of different concentrations are separated by that membrane.

b) Oncotic Pressure: The osmotic pressure due specifically to the presence of plasma colloids, e.g. proteins. Also referred to as "Colloidal Osmotic Pressure".

1. (1 mark for each column correct).

<table>
<thead>
<tr>
<th>Type of substance moving</th>
<th>Diffusion</th>
<th>Osmosis</th>
<th>Active Transport</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecules &amp; ions (usually solute)</td>
<td>Water (solvent)</td>
<td>Molecules &amp; ions (solute)</td>
<td></td>
</tr>
<tr>
<td>With the concentration gradient; from area of higher to lower conc. Of that substance.</td>
<td>With the concentration gradient; from area of higher to lower conc. of water (i.e. from more dilute to more concentrated solution)</td>
<td>Against the concentration gradient; from area of lower to higher conc. Of that substance.</td>
<td></td>
</tr>
<tr>
<td>Passive process (no energy required)</td>
<td>Passive process (no energy required)</td>
<td>Active process (energy is required)</td>
<td></td>
</tr>
</tbody>
</table>

4. a) 60%
b) 42 L
c) age (or fat content)
d) fat content (or age)
e) 80%
f) 10%
g) 5-9%
h) over 10%
5. antidiuretic hormone (ADH) and aldosterone.

6. a) Dehydration: A condition in which there is a net loss of water from the fluid compartments of the body.

   b) Edema: A condition in which the interstitial (tissue) spaces contain an excessive amount of extracellular fluid; characterized by swelling and puffiness of the tissues.

   c) Volume depletion: A clinical term, referring to findings which indicate that circulating volume is diminished.

7.

<table>
<thead>
<tr>
<th>Major Cation</th>
<th>EXTRACELLULAR FLUID</th>
<th>INTRACELLULAR FLUID</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td></td>
<td>K⁺</td>
</tr>
<tr>
<td>Major Anion</td>
<td></td>
<td>HPO₄⁻</td>
</tr>
</tbody>
</table>

8. a) Sodium (any two of):
   - maintenance of osmotic pressure and ECF volume ` transmission of nerve and muscle impulses
   - pH regulation (acidification of urine)
   - regulation of cell membrane permeability

b) Potassium:
   - regulation of muscle irritability (especially cardiac muscle)
   - pH regulation (acidification of urine)

c) Chloride:
   - maintenance of osmotic pressure and ECF volume ` acid-base regulation (excretion with NH₄⁺ ions and reabsorption alternately to HCO₃⁻ ions)

d) Calcium (any two of)
   - formation of bones and teeth
   - blood coagulation
   - enzyme activation
   - transmission of nerve impulses
   - contraction of muscle fibers
   - control of neuromuscular activity
9. Bicarbonate - part of the major blood buffer system, (i.e. the bicarbonate/carbonic acid buffer system)

10. More normal saline than 5% D/W stays in the intravascular space, since normal saline is isotonic and 5% D/W becomes hypotonic once the dextrose component is taken up by the cells. Fluid remaining in the intravascular space then increases the circulating volume.
ADVANCED LIFE SUPPORT
PRE COURSE
FLUIDS AND ELECTROLYTES

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.

   Reading ___________________ hours
   Self assessment ____________ hours
   Total time ________________ hours

2. Were the objectives of the module clearly stated?

   [ ] yes [ ] no
   If no, please comment.

3. Did you see any of the resource materials?

   [ ] yes [ ] no
   If yes, which items
   ______________________________
   ______________________________
   ______________________________
   Were they helpful?
   ______________________________

4. Were the reference notes adequate?

   [ ] yes [ ] no
   If no, please comment.

5. Were the reference notes easy to follow?

   [ ] yes [ ] no
   If no, please comment.
6. Were the examples provided satisfactory?

[ ] yes [ ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[ ] yes [ ] no
If yes, please specify.

1. Was the level of the module satisfactory for your program of study?

[ ] yes [ ] no
If no, please comment.

Base Hospital

9. General comments or suggested improvements.
Paramedic Resource Manual

ACID-BASE BALANCE
SECTION SIX

2005 Update by
Ontario Base Hospital Group Education Subcommittee

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OBJECTIVES: ACID-BASE BALANCE

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. explain the importance of acid-base balance.
2. explain the role of hydrogen ion excretion.
3. define pH and state the normal pH range of blood.
4. name the four physiological buffering systems and state the role of the most important of these in the maintenance of acid-base balance.
5. explain the role of the complementary mechanisms of the blood, lungs and kidneys in maintaining homeostasis.
6. define and state the prehospital, clinical manifestations of:
   a) Metabolic acidosis
   b) Metabolic alkalosis
   c) Respiratory acidosis
   d) Respiratory alkalosis.

If you have studied this subject previously, you may test your ability using the self-assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.

PREREQUISITE KNOWLEDGE

This module includes the information necessary to meet the objectives. However, in order to successfully complete this module, a prerequisite background knowledge in the following topics is required:

1. A basic knowledge of the concepts of pH, acidity and alkalinity, and of the relationship between pH and hydrogen ion (H⁺) concentration. (This information is reviewed in the Glossary of this module.)
2. A familiarity with the theory of buffers.
GLOSSARY

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACIDOSIS</td>
<td>A disturbance of the acid-base balance of the body resulting in a blood pH below 7.35, i.e. acidic compared to normal pH.</td>
</tr>
<tr>
<td>ALKALOSIS</td>
<td>A disturbance of the acid-base balance of the body resulting in a blood pH above 7.45, i.e. alkaline compared to normal pH.</td>
</tr>
<tr>
<td>BUFFER</td>
<td>A chemical solution which reacts with, and resists changes in pH, when acid or base is added to the solution. In the body, buffers act as a transport system to move excess H⁺ ions to the lungs and kidneys for excretion.</td>
</tr>
<tr>
<td>COPD</td>
<td>Abbreviation for “Chronic Obstructive Pulmonary Disease”.</td>
</tr>
<tr>
<td>DEOXYHEMOGLOBIN</td>
<td>The non-oxygenated or reduced form of (reduced Hb) Hemoglobin is symbolized: Hb</td>
</tr>
<tr>
<td>EXTRACELLULAR FLUID (ECF)</td>
<td>The body fluid which occupies the area outside the cells. This includes the blood plasma and fluid in the tissue spaces.</td>
</tr>
<tr>
<td>FIXED ACIDS</td>
<td>Also called “non-volatile acids”. Those acids which cannot be “breathed off” as gases by the lungs, but must be excreted in the urine.</td>
</tr>
<tr>
<td>HOMEOSTASIS</td>
<td>The state of equilibrium in the body with respect to various functions and to the chemical compositions of the fluids and tissues. The processes through which such equilibrium is maintained.</td>
</tr>
<tr>
<td>HYPERVENTILATION</td>
<td>Ventilations which are increased in depth and/or rate, resulting in a loss of CO₂ from the body.</td>
</tr>
<tr>
<td>HYPOVENTILATION</td>
<td>Ventilations which are decreased in rate and/or depth, resulting in a retention of CO₂ in the body.</td>
</tr>
<tr>
<td>INTERSTITIAL FLUID (ISF)</td>
<td>That part of the Extracellular fluid that circulates between and around the cells.</td>
</tr>
<tr>
<td>INTRACELLULAR FLUID (ICF)</td>
<td>A solution of water and electrolytes which circulates within the cells of the body.</td>
</tr>
<tr>
<td>Term</td>
<td>Description</td>
</tr>
<tr>
<td>------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>METABOLIC ACIDOSIS</td>
<td>A condition of decreased blood pH (&lt;7.35) resulting from a decrease in HCO$_3^-$ concentration (due to excessive loss of HCO$_3^-$ or retention/production of acid).</td>
</tr>
<tr>
<td>METABOLIC ALKALOSIS</td>
<td>A condition of increased blood pH (&gt;7.45) resulting from an increase in HCO$_3^-$ concentration (due to retention of HCO$_3^-$ or excessive loss of acid).</td>
</tr>
<tr>
<td>MIXED ACID-BASE DISTURBANCE</td>
<td>A condition in which more than one type of acid-base disturbance exists simultaneously.</td>
</tr>
<tr>
<td>NON-VOLATILE ACIDS</td>
<td>Also called “fixed acids”. Those acids which cannot be “breathed off” as gases by the lungs, but must be excreted in the urine.</td>
</tr>
<tr>
<td>OXYHEMOGLOBIN</td>
<td>The oxygenated (oxygen-bound) form of hemoglobin (symbolized: HbO$_2$).</td>
</tr>
<tr>
<td>pH</td>
<td>Symbol used to express the logarithm of the reciprocal of the Hydrogen ion concentration (used to express the degree of acidity or alkalinity.</td>
</tr>
<tr>
<td>RBCs</td>
<td>Abbreviation for “red blood cells”.</td>
</tr>
<tr>
<td>RESPIRATORY ACIDOSIS</td>
<td>A condition of decreased blood pH (&lt;7.35) due to retention of CO$_2$ which results in an increase in H$_2$CO$_3$ concentration.</td>
</tr>
<tr>
<td>RESPIRATORY ALKALOSIS</td>
<td>A condition of increased blood pH (&gt;7.45) due to an excessive loss of CO2 which results in a decrease in H$_2$CO$_3$ concentration.</td>
</tr>
<tr>
<td>TETANY</td>
<td>A hyperirritability of the muscles leading to tremors and spasms (occurs when the body is in a state of alkalosis).</td>
</tr>
<tr>
<td>VOLATILE ACIDS</td>
<td>Those acids which can be excreted from the body as gases, i.e. “breathed off” by the lungs.</td>
</tr>
</tbody>
</table>
**pH, ACIDITY AND ALKALINITY**

The degree of acidity or alkalinity of a solution is determined by its hydrogen ion \((H^+)\) concentration. An increase in hydrogen ion \((H^+)\) concentration makes a solution more acidic, while a decrease makes it more alkaline. The concentration of free hydrogen ions \((H^+)\) in the body fluids is extremely small, approximately 0.0000001 mol/L, which can be expressed mathematically as \(10^{-7}\) mol/L. Because these small numbers are so cumbersome to work with, the concept of pH was developed to express hydrogen ion \((H^+)\) concentration more compactly. For convenience, acidity and alkalinity is expressed as the negative logarithm of the hydrogen ion \((H^+)\) concentration, which is referred to as pH.

\[
pH = -\log [H^+]
\]

Hydrogen Ion Concentration

in mol/L

To express a hydrogen ion concentration of \(10^{-7}\) mol/L as a pH, the negative sign is dropped and this concentration \((10^{-7})\) of \(H^+\) is indicated as pH 7. Thus, a range of hydrogen ion concentrations of \(10^{-1}\) to \(10^{-14}\) (0.1 to \(0.00000000000001\)) mol/L can be represented as a range of pH from 1 to 14.

<table>
<thead>
<tr>
<th>[H+] mol/L</th>
<th>pH units</th>
</tr>
</thead>
<tbody>
<tr>
<td>(10^{-1})</td>
<td>1</td>
</tr>
<tr>
<td>(10^{-7})</td>
<td>7</td>
</tr>
<tr>
<td>(10^{-14})</td>
<td>14</td>
</tr>
</tbody>
</table>

ACIDIC ↔ NEUTRAL → ALKALINE

There is an inverse relationship between pH and the hydrogen ion \((H^+)\) concentration. That is, as the hydrogen ion concentration increases, the pH decreases, and vice versa. Thus, an acidic solution has a low pH and a high hydrogen ion concentration, while an alkaline solution has a high pH and low hydrogen ion concentration.

**IMPORTANCE OF ACID-BASE BALANCE**

Normal metabolic function can occur only if the composition of the body cells and their surrounding environment are kept relatively constant. Therefore, one of the most important functions of the body is the careful regulation of both fluid and electrolyte balance, and acid-base balance.*
Disturbances in the acid-base balance of the body lead to cellular dysfunction and can seriously jeopardize a patient’s life. Necessary metabolic activities can proceed only if the balance between acidic and basic substances in body fluids is kept within proper limits. The activity of virtually all the thousands of enzymes within the cells are to some extent pH-dependent.

Even more important, however, is the pH-dependence of the overall functioning of the body, e.g. membrane transport processes, and ionic states of all substances. In an acidic or basic environment, some chemical reactions are accelerated while others are slowed down and can even be stopped completely. It is, therefore, important to understand the mechanisms involved in maintaining the body’s normal acid-base balance, and the consequences of acid-base disturbances.

HYDROGEN ION PRODUCTION AND EXCRETION

Acid-base balance at its simplest level refers to the homeostasis (or balance) of the hydrogen ion (H+) concentration in body fluids. Since many end-products of the body’s metabolic reactions are acidic, the body produces a large excess of acid under normal physiological conditions (Figure 1).

If this acid were allowed to accumulate, the effect on the blood, tissue fluid and intracellular fluid pH’s would be dramatic and lethal. Thus, in the face of continual production of acids, the body must have mechanisms by which it can minimize the pH changes in the body fluids. In addition, since the amount of acid produced continually varies, these mechanisms must be able to respond to pH variations and adapt accordingly.

---

* Water and electrolyte balance and acid-base balance are closely interrelated, and in relation to their disturbance in disease, they must be considered together. However, for convenience, these two topics are presented separately, in different instructional modules.

** The concept of pH and its relationship to acidity and alkalinity is reviewed in the Glossary of this module.
MECHANISMS OF pH REGULATION AND HYDROGEN ION EXCRETION

In counteracting the accumulation of excess acid (H\(^+\) ions), the body faces two distinct tasks:

- elimination of the excess hydrogen ions (H\(^+\)) from the body
- prevention of pH changes in the blood while transporting these hydrogen ions to the organs where they will be excreted.

The transportation and elimination of excess hydrogen ions (acid) from the body is accomplished by means of the respiratory system, the renal system and the chemical buffer systems. The respiratory and renal systems are physiological mechanisms by which hydrogen ions (H\(^+\)) are excreted from the body, while the chemical buffer systems rely on their physiochemical action to minimize pH changes in the body fluids during hydrogen ion transport to the lungs and kidneys. Each of these mechanisms (Figure 2) shares the responsibility of maintaining the hydrogen ion concentration (and therefore the blood pH) within its normal, narrow limits.
The body has three main mechanisms by which it regulates blood pH. The chemical buffer systems act more or less immediately in response to an upset in the balance of hydrogen ions in the body fluids. These buffers are responsible for maintaining a normal blood pH (7.35 – 7.45) during hydrogen ion transport to the lungs and kidneys. The lungs and kidneys are responsible for the elimination of excess hydrogen ions from the body. The respiratory response is almost immediate, while the renal response is slowest to act.

THE PHYSIOLOGICAL BUFFER SYSTEMS

An acid-base buffer is a chemical solution which prevents excessive change in pH (and H+ concentration) when either acid or base is added to the solution. Specifically, a buffer is a mixture of: either a weak acid and its alkali salt, or a weak base and its acid salt. In the body, the buffers of physiological importance are mixtures of weak acids and their alkali salts.

If excess base is added to the solution, the weak acid part of the buffer reacts to neutralize it. Likewise if excess acid is added to the solution, the alkali salt part of the buffer reacts to neutralize it. In this way, the body’s buffers can be regarded as chemical sponges, soaking up surplus hydrogen ions (H+) or releasing them as required.

All of the base that is available for immediate neutralization of acids produced by cell metabolism is in the form of buffer salts. Thus, it is only by the chemical action of these buffers that hydrogen ions (H+) can be transported in the blood to the lungs and kidneys for excretion without the blood pH dropping drastically. The chemical action of the buffers
occurs within a fraction of a second to prevent excessive changes in hydrogen ion (H⁺) concentration and pH. Although there are many buffer systems working within body fluids, four main systems exist.

- The bicarbonate/carbonic acid buffer system
- The phosphate buffer system
- The protein buffer system
- The hemoglobin buffer system.

The bicarbonate/carbonic acid buffer system is the major buffer system for fixed acids in the blood. (It buffers ~ 0.7 (70%) of the fixed acids in the plasma and ~ 0.3 (30%) of the fixed acids in the RBCs). It is quantitatively the largest buffer system in the body, and is therefore the most important overall in regulating pH. Part of its importance derives from the fact that each of the components of this buffer system can be regulated via the lungs and kidneys:

- carbonic acid (H₂CO₃) can be retained or exhaled as carbon dioxide (CO₂)
- bicarbonate (HCO₃⁻) can be retained or excreted by the kidney tubules as required by the body.

In the blood, the normal ratio of bicarbonate/carbonic acid is 20/1, so this system is heavily weighted towards buffering against excess acid production.

Both components of this important buffer system can be regulated via the lungs and the kidneys (Figure 3). The weak acid component, carbonic acid (H₂CO₃), can be retained or exhaled as carbon dioxide (CO₂) via the lungs, while the salt component, bicarbonate (HCO₃⁻) can be retained or excreted by the kidney tubules according to the body’s needs.
The other buffer systems are of less clinical significance than the bicarbonate/carbonic acid buffer system and will not be discussed here.

THE RESPIRATORY SYSTEM

The lungs are responsible for the regulation of the levels of volatile acids in the body fluids. The term “volatile” indicates that these compounds can be “breathed off”, i.e. excreted as gases.

Carbon dioxide (CO₂), the major end-product of metabolism, is being formed continuously inside the cells. It diffuses out of the cells, through the interstitial fluid, into the bloodstream. Here it forms carbonic acid (H₂CO₃) and dissolves. It is then buffered and transported to the lungs. In the lung tissue, CO₂ is reformed, diffuses into the alveoli and is exhaled (Figure 4).
In the lungs, acid is excreted as carbon dioxide (CO₂), while the water (H₂O) produced by the reaction is dissipated into the general water pool in the body. Note that these equilibrium reactions can be shifted to the left to produce more acid (H⁺) or to the right to eliminate acid (as CO₂) as required by the body.

Whenever an imbalance in the hydrogen ion (H⁺) concentration exists, the respiratory centre is stimulated. If the body needs to eliminate excess acid (H⁺ ions), the lungs respond by increasing the rate of respiration (called HYPERVENTILATION) so as to eliminate more acid as CO₂ (Figure 5). If the body needs to retain acid (H⁺ ions) to counteract a pH which is too alkaline, the lungs can decrease the rate of respiration (called HYPOVENTILATION) so as to retain more CO₂ (Figure 5).
In the lungs, the equilibrium reaction can be shifted to the left to retain acid by decreasing the role of ventilation (HYPOVENTILATION), or can be shifted to the right to eliminate excess acid by increasing the ventilation rate (HYPERVENTILATION).

The respiratory response to changes in hydrogen ion (H⁺) concentration and blood pH is very rapid. Working alone, the lungs can readjust the H⁺ concentration within seconds after a
sudden change has occurred. However, the respiratory mechanism has only a 50%-75% efficiency rate, i.e. the lungs alone cannot return the pH to its normal level of approximately 7.40. This is because, as the hydrogen ion concentration approaches normal, the stimulus to the respiratory centre is lost. For example, if the blood pH suddenly drops from 7.40 to 7.00, the respiratory system can return the pH to about 7.20-7.30 within a minute. Beyond that, the kidneys and the buffering systems must act to restore the balance. In a case such as this, medical treatment may include attempting to mimic the body's own respiratory response to the acidic pH. For example, the intentional hyperventilation of patients who have been in a state of cardio-respiratory arrest for some time prior to your arrival.

THE RENAL SYSTEM

The kidneys are responsible for the regulation of the levels of non-volatile or fixed acids, acid which cannot be “breathed off”, in the body. These include lactic acid, ketone bodies, phosphoric acid and sulfuric acid. All these acids dissociate in the body fluids to produce free hydrogen ions (H+) which must then be excreted by the kidneys.

Whenever an imbalance in the hydrogen ion (H+) concentration occurs, the kidney tubule cells are stimulated to adjust the excretion or reabsorption (retention) of acid (as H+ ions) or buffer base (as HCO3− ions) as required. If the body needs to eliminate excess acid (H+ ions) when the blood pH is too low, the kidneys respond by excreting more H+ ions (acid) and retaining more HCO3− ions (buffer base to neutralize excess acid). If the blood pH becomes too alkaline, the kidneys respond by retaining H+ ions (acid) and excreting HCO3− ions (buffer base).

The renal response to changes in hydrogen ion (H+) concentration and blood pH is quite slow in comparison to that of the respiratory system. The kidneys, working alone, would require from several hours to a day or more to readjust the H+ concentration after a sudden change. However, due to their versatility, the kidneys are the most powerful of the control mechanisms, and have a 100% efficiency rate. The kidneys, by themselves, are able to return the pH completely to normal given adequate time.
The interaction between the lungs, kidneys and chemical buffer systems are summarized in Figure 6.

In the blood, the chemical buffers act like a reservoir to “even out” the flow of hydrogen ions (H\(^+\)) into the respiratory and renal mechanisms. Excess acid can be eliminated via the kidneys (as H\(^+\)) or the lungs (as CO\(_2\)).

### ACID-BASE DISTURBANCES

In disease, the pH of the blood and other fluids may move outside of normal limits due to an upset in the balance between acids and bases. This condition poses a serious threat to life unless it is remedied without delay. The organs which act to regulate acid-base homeostasis will attempt to restore the balance:

The lungs, by excreting or retaining acid (as CO\(_2\))
The kidneys, by excreting or retaining acid (as H\(^+\)) or base (as HCO\(_3\)) as required.
Because of the importance of bicarbonate ions (HCO$_3^-$) and carbon dioxide (CO$_2$) in the regulation of acid or base excesses or deficits, the pH of the blood is directly dependent on bicarbonate/carbonic acid ratio in the blood. The pH of the blood and other body fluids is directly proportional to the ratio of bicarbonate (HCO$_3^-$) to carbonic acid (H$_2$CO$_3$) (Figure 7).

In health, the normal blood pH is 7.40 and the bicarbonate/carbonic acid ratio is 20/1. When the ratio is disturbed, the pH of the blood and body fluids may move outside the normal limits (pH 7.35-7.45). The resulting condition will be either ACIDOSIS, in which the pH is below the lower limit, or ALKALOSIS, when the pH is above the upper limit.

Acidosis can result from the body’s failure to excrete excess acid (respiratory disease, kidney failure), the body’s overproduction of acid metabolites (diabetes mellitus), or a loss of the body’s alkaline reserves (severe diarrhea). Alkalosis can result from excessive loss of acid (hyperventilation, severe vomiting), or the addition of large amounts of alkali (excessive antacid ingestion).
When the pH goes below 7.35, there is an excess of hydrogen ions (acid) and a state of ACIDOSIS exists. When the pH goes above 7.45, there is a depletion of hydrogen ions (acid) and a state of ALKALOSIS exists. The extreme limits that are compatible with life are a blood pH of approximately 6.80-7.90. However, as the pH deviates from the norm of 7.35-7.45, there is a progressive deterioration in enzyme systems and cellular function. The patient's prognosis at such values is very poor and in either extreme, the imbalance may result in death if not corrected. As a point of interest, the body tends to tolerate acidosis much better than alkalosis.

In summary:

\[
\begin{align*}
\text{Normal blood pH} &= 7.35 \text{ – } 7.45 \\
\text{pH < 7.35} & \text{is} \quad \text{ACIDOSIS} \\
\text{pH > 7.45} & \text{is} \quad \text{ALKALOSIS}
\end{align*}
\]

Since an acidosis or an alkalosis can result from an abnormality in either the bicarbonate or the carbonic acid concentration, acid-base disturbances can be further classified as either “metabolic” or “respiratory”. The concentration of bicarbonate ions (HCO\(_3^-\)) is affected by the metabolic production of acids and bases and by the activities of the kidneys. Because of this, bicarbonate (HCO\(_3^-\)) is referred to as the METABOLIC component of the ratio (or sometimes the “NON-RESPIRATORY” component).

If there is an excess of non-volatile acids or a loss of base, the blood bicarbonate (HCO\(_3^-\)) concentration decreases, causing a drop in pH to an acidic level. Therefore, any acid-base disturbance involving a loss of bicarbonate (HCO\(_3^-\)) is classified as – a METABOLIC ACIDOSIS. If there is a decreased buffering use of bicarbonate (due to a decrease in non-volatile acids) or other increase in the base concentration, the blood bicarbonate (HCO\(_3^-\)) concentration increases, causing a rise in pH to an alkaline level. Therefore, any acid-base disturbance involving an increase in bicarbonate (HCO\(_3^-\)) concentration is classified as a METABOLIC ALKALOSIS (Figure 9).
The concentration of carbonic acid (\(H_2CO_3\)) is determined by the carbon dioxide (CO\(_2\)) concentration, which is in turn controlled by the lungs. Because of this, carbonic acid (\(H_2CO_3\)) is referred to as the RESPIRATORY component of the ratio.

If the lungs cannot remove CO\(_2\) (by decreased lung activity), the blood carbonic acid (\(H_2CO_3\)) concentration increases, causing a drop in pH to an acidic level. Therefore, any acid-base disturbance associated with an increase in carbonic acid (\((H_2CO_3)\)) concentration (as CO\(_2\) retention) is classified as a RESPIRATORY ACIDOSIS. If the lungs remove too much CO\(_2\) (by increased lung activity), the blood carbonic acid (\(H_2CO_3\)) concentration decreases, causing a rise in pH to an alkaline level. Therefore, any acid-base disturbance associated with a decrease in carbonic acid (\(H_2CO_3\)) concentration (as CO\(_2\) loss) is classified as a RESPIRATORY ALKALOSIS.
These, then, are the four main classifications of acid-base disturbances. In each case, the loss from, or addition to the body of acid or base, causes a shift in blood pH outside the normal range. At this point each disturbance is said to be **UNCOMPENSATED**.

In respiratory disturbances, the lungs are usually compromised, and so the kidneys will attempt to compensate, i.e. restore the acid-base balance, by retaining or excreting H⁺ ions (acid) or HCO₃⁻ ions (base) as needed.

In metabolic disturbances, the lungs will attempt to compensate by retaining or eliminating CO₂ (acid) as required. If the kidneys are not directly involved in the metabolic disturbances, they will also help to restore the acid-base balance.

In addition to the body’s own compensatory mechanisms, administration of chemical treatment by a physician may be required in order to restore the acid-base balance. When the pH is brought back within the normal range, the disturbance is said to be **FULLY COMPENSATED**. In most cases however, the action of compensatory mechanisms and medical treatment result in only **PARTIAL COMPENSATION**, i.e. pH improves but remains outside the normal range, until the primary cause of the disturbance can be corrected.
In seriously ill patients, it is not uncommon to have two types of acid-base disturbance present simultaneously – so-called **MIXED DISTURBANCES**. For example, in a case of salicylate (aspirin) overdose, the initial disturbance is a respiratory alkalosis. This occurs due to the stimulation of the respiratory centre by the drug which produces marked hyperventilation. Then, the metabolism of the salicylates results in an accumulation of acids in the body and a metabolic acidosis. Cases of mixed acid-base disturbances are quite complex and difficult, and treatment must be vigorous to prevent death.

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>CONDITIONS LEADING TO ACIDOsis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>METABOLIC ACIDOSIS</strong></td>
<td><strong>RESPIRATORY ACIDOSIS</strong></td>
</tr>
<tr>
<td>o Uncontrolled diabetes mellitus (+ ketoacids)</td>
<td>o Lung disease – COPD – Pneumonia (impaircd gas exchange, retention of CO₂)</td>
</tr>
<tr>
<td>o Starvation or severe carbohydrate – reduced diet (↑ ketoacids)</td>
<td>o Barbiturate overdose (depression of respiratory centre, retention of CO₂)</td>
</tr>
<tr>
<td>o Severe exercise (↑ lactic acid)</td>
<td>o Overdose of narcotics, or any sedative, tranquilizer or major depressant (same as for barbiturates)</td>
</tr>
<tr>
<td>o Severe diarrhea (G.I. loss of HCO₃⁻)</td>
<td>o Head injury (brain damage, depression of respiratory centre, retention of CO₂)</td>
</tr>
<tr>
<td>o Renal failure (failure to excrete H⁺)</td>
<td>o Any condition resulting in hypoventilation (retention of CO₂)</td>
</tr>
<tr>
<td>o Cardiac failure/arrest (↑ lactic acid)</td>
<td></td>
</tr>
<tr>
<td>o Acute alcohol intoxication (↑ fixed acids)</td>
<td></td>
</tr>
<tr>
<td>o Salicylate overdose (↑ fixed acids)</td>
<td></td>
</tr>
<tr>
<td>o Shock (↑ lactic acid)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>CONDITIONS LEADING TO ALKALOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>METABOLIC ALKALOSIS</strong></td>
<td><strong>RESPIRATORY ALKALOSIS</strong></td>
</tr>
<tr>
<td>o Excessive administration of sodium bicarbonate during treatment of cardiac arrest (↑ HCO₃⁻)</td>
<td>o Salicylate overdose – early stages (stimulation of respiratory centre, loss of CO₂)</td>
</tr>
<tr>
<td>o Prolonged volume depletion (excessive diuretic use)</td>
<td>o High fever (stimulation of respiratory centre, loss of CO₂)</td>
</tr>
<tr>
<td>o Prolonged vomiting (gastric loss of H⁺)</td>
<td>o Hysteria/voluntary overbreathing (hyperventilation, loss of CO₂)</td>
</tr>
<tr>
<td>o Gastric suction (gastric loss of H⁺)</td>
<td>o Passive overventilation by incorrectly adjusting artificial ventilator (loss of CO₂)</td>
</tr>
<tr>
<td>o Hyperaldosteronism (i.e. Cushing’s Disease) (renal loss of H⁺)</td>
<td>o Any condition resulting in hyperventilation (excretion of CO₂)</td>
</tr>
</tbody>
</table>
In respiratory acidosis there is a build up of CO₂ (usually associated with hypoxia) – the causes of which are listed in Table 1. Since the accumulation of CO₂ is not desirable you will have to provide adequate ventilation either by treating the cause and/or assisting the patient’s ventilation. This may require manually ventilating the patient and treating the cause later. Hypoxia will be corrected by oxygenating the patient.

Some of the causes of respiratory acidosis may be treated in the field promptly and adequately, e.g. narcotic overdose can be reversed with naloxone with immediate correction of the respiratory acidosis and hypoxia. Other causes such as pulmonary edema or a head injury may require assisted ventilation and/or intubation. This is used because the patient’s condition is not immediately reversible. It is necessary to lower the CO₂ and increase the oxygen levels in order to return the pH of body fluids to normal. Vital organs are compromised by increased CO₂ and decreased O₂. Increased CO₂ can cause the brain to swell, the heart can become irritable and susceptible to arrhythmias and the blood pressure can drop. Therefore adequately ventilating the patient can be vital to survival.

An example of metabolic acidosis that can be treated initially in the field is diabetic ketoacidosis. These patients are usually volume depleted and require large volumes of isotonic saline (normal saline) to correct their volume depletion. Once the patient’s volume is restored then perfusion to the tissues is improved. This decreases the build up of lactic acid thus returning the anion gap and the blood pH to normal.

Respiratory alkalosis is a condition in which the patient’s respiratory system is stimulated to a degree that the level of CO₂ is lowered below normal. While hyperventilation due to anxiety is one possibility, before you apply the paper bag for rebreathing, consider the possibility of ASA toxicity or diabetic ketoacidosis as the cause of the respiratory pattern. In these instances, rebreathing will do more harm than good.
Marks

[4] 1. Fill in the blanks:

The overall functioning of the body relies heavily on the maintenance of proper acid-base balance. Chemical reaction rates are very pH dependent, as is the activity of the (a) ______ which catalyze these reactions. The acid-base balance refers specifically to the balance or (b) ___________ of the hydrogen ion concentration (and therefore, pH) in body fluids.

The end-products of metabolism are mainly (c) ____________ in nature. Therefore, the body must act to prevent lethal changes in the pH of body fluids. The excess hydrogen ions are excreted via the (d) _____________ and the (e) ________________ and are "neutralized" during transport in the blood by (f) _____________. The lungs excrete the (g) _________________ acids, while the kidneys excrete the (h) _________________ acids. These are the mechanisms by which the body maintains acid-base balance.

[2] 2. Complete the following chart, comparing the response of the lungs vs. the kidneys to acid-base disturbances.

<table>
<thead>
<tr>
<th>Response to:</th>
<th>LUNGS</th>
<th>KIDNEYS</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Speed of response (fast or slow)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b) Restores normal pH (partially or completely)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

[4] 3. Complete the following chart, stating the response of the lungs and the kidneys to low blood pH (excess acid) and high blood pH (excess base).

<table>
<thead>
<tr>
<th>Response to:</th>
<th>LUNGS</th>
<th>KIDNEYS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low blood pH (excess acid)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High blood pH (excess base)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
4. a) NaHCO$_2$ is the chemical formula for _________________.
   b) Prepared as a drug for intravenous administration it would be used to (raise/lower) the pH.
   c) The effect of hyperventilation of a patient in cardio-respiratory arrest is (raise/lower) the pH.
   d) The untreated cardiac arrest patient will have an acid-base disorder of which type? _________________________________.

Why?

5. a) State the normal range of blood pH.
   b) Name the main buffer pair which determines blood pH.
6. Complete the following chart by stating:

- The effect on blood pH (increased or decreased)
- The type of acid-base disturbance resulting from each imbalance.

(1/2 mark each for effect; 1 mark each for naming disturbance)

<table>
<thead>
<tr>
<th>IMBALANCE</th>
<th>EFFECT ON pH</th>
<th>TYPE OF ACID-BASE DISTURBANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCO₃⁻ ↑↑</td>
<td>H₂CO₃ ↓</td>
<td></td>
</tr>
<tr>
<td>H₂CO₃</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HCO₃⁻ ↓↓</td>
<td>H₂CO₃ ↑</td>
<td></td>
</tr>
<tr>
<td>H₂CO₃</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>H₂CO₃ ↑↑</td>
<td></td>
</tr>
<tr>
<td>H₂CO₃</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

7. a) State two clinical situations which may result in metabolic acidosis, indicating the source of the acidosis.

__________________________________________________________________________
__________________________________________________________________________
__________________________________________________________________________
__________________________________________________________________________
__________________________________________________________________________

33 TOTAL
SELF-ASSESSMENT ANSWERS

1. a) enzymes
   b) homeostasis
   c) acidic
   d) lungs
   e) kidneys
   f) (chemical) buffers
   g) volatile
   h) non-volatile or fixed

2. | LUNGS       | KIDNEYS     |
   |-------------|-------------|
   a) Speed of response (fast or slow) | fast | slow |
   b) Restores normal pH (partially or completely) | partially | completely |

3. | Response to:               | LUNGS                   | KIDNEYS                          |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Low blood pH (excess acid)</td>
<td>Hyperventilation</td>
<td>Excrete H⁺ ions (acid)</td>
</tr>
<tr>
<td></td>
<td>(exhale excess acid as CO₂)</td>
<td>Retain HCO₃⁻ ions (base)</td>
</tr>
<tr>
<td>High blood pH (excess base)</td>
<td>Hypoventilation</td>
<td>Excrete HCO₃⁻ ions (base)</td>
</tr>
<tr>
<td></td>
<td>(retain acid as CO₂)</td>
<td>Retain H⁺ ions (acid)</td>
</tr>
</tbody>
</table>

4. a) sodium bicarbonate
   b) raise
   c) raise
   d) mixed acidosis (i.e. both respiratory and metabolic) Why?
      (1 mark each)
      o lack of ventilation causes CO₂ accumulation (respiratory component).
      o Hypoxia and lack of perfusion lead to anaerobic metabolism at the cellular level.
      o Lactic acid production results from anaerobic metabolism. This is the metabolism component.
   e) ventilations that are increased in either rate, depth or both, resulting in loss of CO₂.
   f) ASA toxicity; head injury; diabetic ketoacidosis.
5. a) normal range of blood pH = 7.35 – 7.45  
b) Main buffer pair which determines blood pH: bicarbonate/carbonic acid or HCO₃⁻/H₂CO₃

6.

<table>
<thead>
<tr>
<th>IMBALANCE</th>
<th>EFFECT ON pH</th>
<th>TYPE OF ACID-BASE DISTURBANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCO₃⁻ ↑↑ H₂CO₃</td>
<td>↑ (increase)</td>
<td>Metabolic Alkalosis</td>
</tr>
<tr>
<td>HCO₃⁻ ↓↓ H₂CO₃</td>
<td>↓ (decrease)</td>
<td>Metabolic Acidosis</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>↓ (decrease)</td>
<td>Respiratory Acidosis</td>
</tr>
<tr>
<td>H₂CO₃ ↑↑</td>
<td>↑ (increase)</td>
<td>Respiratory Alkalosis</td>
</tr>
</tbody>
</table>

7. Any two of:
   - diabetic ketoracidosis (↑ ketoacids)
   - starvation (↑ ketoacids)
   - severe exercise (↑ lactic acid)
   - severe diarrhea (loss of HCO₃⁻)
   - renal failure (↑ H⁺)
   - cardiac arrest (↑ lactic acid)
   - acute alcohol intoxication (↑ fixed acids)
   - ASA overdose (↑ fixed acids)
   - Shock (↑ lactic acid)
ADVANCED LIFE SUPPORT
PRE COURSE
ACID-BASE BALANCE

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.

   Reading  ___________ hours
   Self assessment  ___________ hours
   Total time  ___________ hours

2. Were the objectives of the module clearly stated?

   [ ] yes  [ ] no
   If no, please comment.

3. Did you see any of the resource materials?

   [ ] yes  [ ] no
   If yes, which items
   __________________________________________________________
   __________________________________________________________
   __________________________________________________________
   Were they helpful?
   __________________________________________________________

4. Were the reference notes adequate?

   [ ] yes  [ ] no
   If no, please comment.

5. Were the reference notes easy to follow?
[ ] yes  [ ] no
If no, please comment.

6. Were the examples provided satisfactory?

[ ] yes  [ ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[ ] yes  [ ] no
If yes, please specify.

1. Was the level of the module satisfactory for your program of study?

[ ] yes  [ ] no
If no, please comment.

Base Hospital

9. General comments or suggested improvements.
Paramedic Resource Manual

NERVOUS SYSTEM
SECTION SEVEN

2005 Update by
Ontario Base Hospital Group Education Subcommittee

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OBJECTIVES: NERVOUS SYSTEM

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. Locate and describe the functions of the components of the central nervous system (the spinal cord and major parts of the brain).

2. Locate and briefly describe the protective/nutritive aspects of the central nervous system:
   a) Skull
   b) Meninges
   c) Vertebrae
   d) CSF
   e) Circulation.

3. Briefly describe the somatic and autonomic nervous systems in terms of:
   a) Neuronal components
   b) Sympathetic and autonomic components
   c) Control mechanisms
   d) Effects on the major organs and body systems.

4. Briefly describe the neuron and the mechanism by which impulse transmission occurs.

5. Identify and locate the common neurotransmitters.

6. State and differentiate between the two major types of pain.

7. State the functions of pain.

8. Identify the components of the nervous system and factors responsible for influencing:
   a) Consciousness, wakefulness and cognitive function
   b) Decreased levels of consciousness and coma.

9. Identify the components of the accepted assessment tool for evaluating brain function.

10. Define and briefly describe reflex action, identifying the reflexes tested by prehospital personnel.

11. Identify the most vulnerable areas of the central nervous system in terms of illness and injury.

12. Describe and explain the mechanics of increased intracranial pressure.
13. Describe briefly the pathology associated with seizures, TIA’s, CVA’s, the major intracranial bleeds, head injury, spinal cord injury.

If you have studied this subject previously, you may test your ability using the self-assessment questions at the end of each section. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the section, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.
GLOSSARY

ACTION POTENTIAL synonym for nerve impulse

AFFERENT conducting toward a central site of reference

ATROPHY wasting

AXON nerve cell appendage along which impulses travel away from the nerve cell body

CLEFT a fissure or longitudinal opening (as in synaptic cleft)

CONDUCTIVITY the capacity to convey energy, e.g. impulse

CONSTRICT to narrow or compress

CONVULSION a series of involuntary contractions of the voluntary muscles

CVA cerebral vascular accident, i.e. problem pertaining to the blood vessels of the brain

DENDRITE tree-like processes composing most of the receptive surface of a neuron

DILATE to stretch a structure or orifice

EDEMA an abnormal accumulation of fluid in the interstitial spaces of the body

EFFECTOR conducting or progressing away from a central site

EMBOLUS a clot or other plug brought by the blood from another vessel and forced into a smaller one leading to the obstruction of circulation

EXCITABILITY readiness to respond to a stimulus – irritability

HEMATOMA a localized collection of extravasated blood, usually clotted, in an organ, space or tissue

HEMIPARESIS slight or incomplete paralysis affecting one side of the body

HEMIPLEGIA paralysis of one side of the body
<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEMISPHERE</td>
<td>half of a spherical or roughly spherical structure or organ</td>
</tr>
<tr>
<td>HOMEOSTASIS</td>
<td>a tendency of biological systems to maintain stability while continually adjusting to conditions that are optimal for survival</td>
</tr>
<tr>
<td>ICP</td>
<td>- intracranial pressure&lt;br&gt;- measured as the pressure of the fluid in the subarachnoid space</td>
</tr>
<tr>
<td>IMPULSE (NERVE)</td>
<td>the electrochemical process propagated along nerve fibres</td>
</tr>
<tr>
<td>INTRACEREBRAL</td>
<td>within the cerebrum</td>
</tr>
<tr>
<td>INTRACRANIAL</td>
<td>within the cranium (skull)</td>
</tr>
<tr>
<td>INVOLUNTARY</td>
<td>performed independently of conscious will</td>
</tr>
<tr>
<td>ISCHEMIA</td>
<td>the deficiency of blood in a part, due to a total or partial obstruction of a blood vessel</td>
</tr>
<tr>
<td>LESION</td>
<td>a broad term referring to any pathological or traumatic discontinuity of tissue or loss of function of a part</td>
</tr>
<tr>
<td>MYELIN</td>
<td>a lipid substance forming a sheath around the axons of certain nerve fibres</td>
</tr>
<tr>
<td>NEURON</td>
<td>nerve cell</td>
</tr>
<tr>
<td>NEUROTRANSMITTER</td>
<td>a substance released into the synaptic cleft between an axon and the target cell can either excite or inhibit the target cell</td>
</tr>
<tr>
<td>PERIPHERY</td>
<td>the portion of a system outside the central area</td>
</tr>
<tr>
<td>PERMEABLE</td>
<td>permitting passage of a substance</td>
</tr>
<tr>
<td>PRODROMAL</td>
<td>symptom indicating the onset of a disease</td>
</tr>
<tr>
<td>PSYCHOMOTOR</td>
<td>pertaining to motor effects of cerebral activity</td>
</tr>
<tr>
<td>RECEPTOR</td>
<td>a site on the surface or within a cell that recognizes and binds with specific molecules, or a sensory nerve ending that responds to various stimuli</td>
</tr>
<tr>
<td>REFLEX</td>
<td>a particular automatic response mediated by the nervous system abnormally increased muscle tone causing a continuous resistance to stretching</td>
</tr>
<tr>
<td>SPASTICITY</td>
<td>a particular automatic response mediated by the nervous system abnormally increased muscle tone causing a continuous resistance to stretching</td>
</tr>
</tbody>
</table>

OBHG Education Subcommittee  

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<table>
<thead>
<tr>
<th>THROMBUS</th>
<th>an aggregation of blood factors frequently causing vascular obstruction at the site of its formation</th>
</tr>
</thead>
<tbody>
<tr>
<td>TIA</td>
<td>transient ischemic attack</td>
</tr>
<tr>
<td>VISCERA</td>
<td>large interior organs in any of the great body cavities</td>
</tr>
<tr>
<td>VOLUNTARY</td>
<td>accomplished in accordance with will</td>
</tr>
</tbody>
</table>
INTRODUCTION

Although nervous system tissue accounts for only 3% of total body weight, it functions to activate, control and integrate all of the systems in the body. It receives information regarding changes in the internal and external environment and initiates and regulates the appropriate response to the stimulus. Thus the nervous system plays an important role in maintaining homeostasis.

The nervous system can be classified by either location or by method of control. Anatomically it is divided into the Central Nervous System (CNS) and the Peripheral Nervous System (PNS). Functionally, the peripheral nervous system is divided into the Somatic Nervous System (SNS), which is under voluntary control, and the Autonomic Nervous System (ANS), which is involuntary.
SUMMARY

CLASSIFICATION OF NERVOUS TISSUE

CENTRAL NERVOUS SYSTEM
- Brain
- Spinal Cord

PERIPHERAL NERVOUS SYSTEM
- Somatic (voluntary)
  - Spinal Nerves
  - Cranial Nerves
- Autonomic
  - Parasympathetic
  - Sympathetic

(Singular Neuron System)  (Multiple Neuron System)
CENTRAL NERVOUS SYSTEM

The Central Nervous System (CNS) consists of the brain and spinal cord. These areas are of major importance in the nervous system as a whole. As you progress through this material keep the following facts in mind:

- The effects of CNS injuries depend on the location and extent of the damage sustained.
- It may be possible to deduce the nature of an injury by observing what abilities are missing or compromised.
- Functional losses may be permanent when the CNS is injured, since CNS tissue is unable to regenerate.
- Both the brain and spinal cord are surrounded by bone and protective membranes (the meninges).

THE BRAIN

The brain is the largest and most complex mass of nervous tissue. It is housed within a bony structure, the skull, which is a relatively “closed-box”. The brain does not achieve its final size (3-4 kg) until approximately the twentieth year. With age, the brain loses size and weight as it undergoes various degrees of atrophy.

The divisions of the brain that we shall consider are the cerebrum, cerebellum, and the brain stem. In addition, other structures of note are the thalamus, hypothalamus, pituitary gland, ventricles, skull and meninges.

CEREBRUM

The cerebrum is the largest part of the brain and is divided into right and left hemispheres. One hemisphere is dominant over the other in intellectual and motor functions. These cerebral hemispheres are connected and communicate through a structure call the corpus callosum.

The cerebrum has another layer of gray matter, composed of nervous tissue cell bodies and dendrites. This covering is called the cerebral cortex. Beneath the cortex is the cerebral medulla, more commonly referred to as white matter. This is an area of myelinated axons that interconnect neurons both within the nervous system and with other body parts.

The surface of the cerebral cortex is marked by ridges and grooves (gyri) and is divided into lobes by spaces called sulci. There are four distinct lobes in the cerebrum which correspond in name to the bones of the skull located adjacent to these areas – frontal, parietal, temporal and occipital.

The functions of the cerebrum are complicated, numerous and not fully understood as yet. It is known to be involved in such intellectual activities as interpretation of sensory impulses, control of voluntary muscles, storage of memory, thought and reasoning.
The **anterior aspect** of the frontal lobe deals with the elaboration of thought and correlates different types of information into a coherent sequence of action, e.g. decision making. The motor ability for speech, i.e. muscle control, is also centered in the area.

The **posterior portion of the frontal lobe** controls the initiation of voluntary movement. Dysfunction in this area may result in hemiplegia, spasticity or hemiparesis of the **opposite side** of the body and/or convulsive seizures.

Just posterior to the frontal lobe lies that part of the **parietal lobe** which represents the somatic sensory area. It is here that basic stimuli are felt and then redirected into surrounding sensory areas for more explicit interpretation. Lesions in this lobe result in disturbances in the sensation of touch.

The **temporal lobe** functions as the primary hearing centre and for the storage of memory patterns. Injury or illness to this area may result in receptive dysphasia (inability to understand the significance of the spoken word), visual hallucinations, visual defects and/or psychomotor seizures.

The posterior aspect of the occipital lobe is concerned with sight. Dysfunction within this lobe may manifest as blindness of the nasal half of one eye and the temporal half of the other and/or visual hallucination (with or without generalized convulsions).
THALAMUS

Situated below the inferior central portion of the cerebrum is the thalamus. The thalamus acts as a relay station which receives sensory impressions from lower regions in the body and projects them onto the cerebral cortex for interpretation. It is possible that certain types of pain may reach consciousness in this area as well.
Clinical vignette

Pain is a protective function and works by apprising the brain of almost any type of damaging process. This causes the appropriate stimuli. Pain receptors are activated only when body tissues are being stimulated – not only due to the initial damage but also from swelling, release of chemicals and other stimuli.

Damaged tissue immediately causes the release of substance called **bradykinin**, which stimulates pain nerve endings. Bradykinin also triggers the release of histamine. Histamine increases capillary permeability, allowing fluid, white blood cells and other chemicals into the damaged area, causing swelling and more pain.

Painful stimuli are received at exposed nerve endings throughout the periphery (skin) and the viscera. The impulses are carried to the spinal cord and then to the brain and interpretation. Examples of the stimuli damaging to tissues include:

- Trauma
- Ischemia
- Intense heat
- Extreme cold
- Chemical irritation

The stimuli for and the nature of the pain produced will vary to some degree for each tissue, for example:

- Skin – cutting or burning
- Digestive tract – distension or spasm
- Skeletal muscle – ischemia
- The heart – ischemia

Peripheral pain can be appropriately localized, via the cerebral cortex, to a specific body surface area. Visceral sensations (which may present as pain, a burning sensation or pressure) may be felt on a surface area of the body rather than within the organ itself. This is called **referred pain**. It may be referred to the surface immediately above the organ, or it may be felt in an area a considerable distance away from the originating organ.

The surface area to which visceral pain is referred usually corresponds to that portion of the body where embryonic development of the organ occurred, **e.g.** the heart originates in the embryonic neck as does the arm, therefore heart pain is frequently referred to the arm.
HYPOTHALAMUS

Lying beneath the thalamus is the hypothalamus. The hypothalamus has many functions. It is the main autonomic centre for the regulation of the activities of smooth muscle, cardiac muscle and endocrine glands. It also sends fibers to subordinate centres throughout the brain which regulate various automatic functions such as digestion, excretion and respiration. It is important as a control centre for hunger and thirst.

PITUITARY GLAND

The pituitary gland, which stores and/or releases several important hormones, is attached by a stalk to the inferior surface of the hypothalamus.

CEREBELLUM

The cerebellum is concerned with the control of posture and with fine muscle control. Illness or injury to this portion of the brain may result in headaches, vomiting and/or visual disturbances. These problems may involve neighbouring cranial nerves and/or the brain stem.
BRAIN STEM

Structurally, the brain stem is made of three divisions:

- Midbrain
- Pons
- Medulla oblongata.

FIGURE 4: BRAIN STEM

The midbrain lies just below the cerebrum. Structurally as well as functionally, it serves to connect the cerebrum to the lower brain centers and the spinal cord. It is an important site of visual and auditory reflex activity, and is the origin for cranial nerves III and IV (pupillary reflexes and eye movements).

Lying inferior to the midbrain and superior to the medulla oblongata is the pons which functions as a conduct ion network between the spinal cord and brain. It contains the nuclei of cranial nerves V through VIII.

The medulla oblongata is also a conduction pathway between the spinal cord and brain. This area is formed by an enlargement of the cord as it enters the cranial cavity through the foramen magnum. The medulla oblongata contains the nuclei for the cranial nerves VIII through XII. It is a centre for the reflexes of vomiting, coughing and hiccups.
The most important function of the medulla oblongata is that it serves as the control centre for vital visceral activities including:

- Cardiac vital centre (controls the heart rate)
- Vasomotor vital centre (sends impulses to the smooth muscles in the walls of blood vessels causing them to constrict, with a subsequent rise in blood pressure)
- Respiratory vital centre (functions with the pons to regulate the rate, depth and rhythmicity of breathing).

Illness or injury affecting the medulla oblongata can result in death due to the compromise of vital control centers.

Scattered throughout the entire brain stem, but concentrated in the medulla oblongata, is a collection of large and small-interconnected neurons known as the **reticular formation**. This net-like structure of gray and white matter is believed essential for the cortical activities associated with initiating and maintaining wakefulness. As such, it is often referred to as the **Reticular Activating System (RAS)**.

Very little is known about the neural mechanisms that produce the cerebral function of consciousness. Self-awareness and awareness of one's environment may define the state of consciousness. One fact is clear; the excitation of cortical neurons by impulses conducted to them by the RAS is necessary for consciousness. The cerebral cortex cannot activate itself. Without continuous stimulation an individual will remain unconscious and will not be able to be aroused.

As well as being crucial for maintaining consciousness, the RAS is considered the arousal or alerting system for the cerebral cortex. Pain, loud verbal stimuli and/or movement can cause an arousal reaction, e.g. immediate activation of the RAS. This is the means by which sensory stimuli awaken us from sleep.

Drugs that act directly on the RAS will affect the level of consciousness, e.g. barbiturates depress the RAS while amphetamines are thought to stimulate this system.

**VENTRICLES OF THE BRAIN**

The ventricles are four fluid-filled interconnected cavities located within the cerebral hemispheres and brain stem. The fluid circulating between the ventricles and through the **subarachnoid space** of the skull and spinal column is called **cerebrospinal fluid (CSF)**. This fluid is secreted into the ventricles, circulates and is then reabsorbed. The delicately balanced volume of CSF primarily consists of water containing glucose, sodium chloride and protein. The main function of CSF is to act as a fluid cushion to protect the delicate brain and spinal cord tissue, maintaining a constant environment for these vital structures.
MENINGES

The brain and spinal cord possess three complete membranes coverings known as meninges. From outermost to innermost, these are the **dura mater**, the **arachnoid mater** and the **pia mater**.

In the cranial cavity, the avascular dura mater comprises not only the tough fibrous outermost covering for the brain, but also the lining membrane (periosreum) on the inner surface of the skull. There is normally no space between these two layers.

The **arachnoid mater** is a very thin, delicate, avascular membrane which lines and completely adheres to the inner surface of the dura mater. Between the arachnoid mater and the underlying pia mater is the **subarachnoid space**, containing **cerebrospinal fluid**.

The **pia mater** is a transparent, delicate membrane which completely adheres to the outer surface of the brain and spinal cord like a “skin”. The pia mater contains blood vessels and is therefore a nutritive layer as well.

**FIGURE 5: SCHEMATIC DIAGRAM OF THE MENINGES**
THE SKULL

Underneath the three layers comprising the scalp lies the skull, a cranial vault which is especially thin in the temporal regions. The cranium is a fusion of many smaller bones joined at suture lines.

The base of the skull is irregular and internally rough. This term actually refers to the floor of the skull, as seen from above. The base is comprised of the bones behind the nose and above the roof of the mouth, namely the ethmoid and sphenoid.

In the event of a skull fracture, the type and extent of this injury will vary with the age of the patient and the nature of the injury, i.e. mechanical agent and amount of force.

BLOOD SUPPLY TO THE BRAIN

It is impossible to over emphasize the importance of an uninterrupted blood supply to the brain. The brain receives about 16% of the total cardiac output and consumes about 20 percent of the oxygen used by the whole body. An abrupt cessation of oxygen carrying blood to the brain may lead to an unconscious state in several seconds. Brain death follows in several minutes from the initial cessation of oxygen. The amount of time this actually takes depends on predisposing medical and environmental factors. The brain stores approximately a 10
second supply of oxygen. This means that metabolic failure is almost instant with cerebral hypoxia.

Glucose is another life-sustaining component found in blood. The lack of this substance in the blood, e.g. hypoglycemia, results in diminished cellular metabolism which especially affects the brain. If hypoglycemia is prolonged, cell death slowly occurs – eventually causing brain death.

The superficial (external) and deep (internal) cerebral veins drain venous blood from the cerebrum. The cerebral veins empty into the dura sinuses, which are channels that lie between the two layers of dura mater. Venous blood drains into its respective internal jugular vein (right and left). All venous blood of the brain empties into the two internal jugular veins.

**FIGURE 7: CIRCULATION OF BLOOD IN THE BRAIN**

**THE SPINAL CORD**
The medulla oblongata continues outside the skull as the spinal cord, a rod of nervous tissue approximately 45 cm in length in the adult. The spinal cord terminates in the lower back (at L₁ in the adult) as the conus medullaris (Figure 8).
The coverings or meninges of the spinal cord are the same as those around the brain and are continuous with the meninges around the brain.

**FIGURE 8: SPINAL CORD**

[Diagram of the spinal cord with labeled parts: Medulla, Conus Madularis (Cord Ends at L1), Cauda Equina]
ADVANCED LIFE SUPPORT
PRE COURSE
THE NERVOUS SYSTEM

SELF-ASSESSMENT: CENTRAL NERVOUS SYSTEM

Marks

[1] 1. Which part of the brain extends from the cerebral hemisphere to the foramen magnum?

[7] 2. Match the brain structure in Column A with its function in Column B.

<table>
<thead>
<tr>
<th>COLUMN A</th>
<th>COLUMN B</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Thalamus</td>
<td>a) concerned with sight</td>
</tr>
<tr>
<td>2. Temporal lobe</td>
<td>b) contains cardiac vital center</td>
</tr>
<tr>
<td>3. Hypothalamus</td>
<td>c) center for static and dynamic equilibrium</td>
</tr>
<tr>
<td>4. Medulla oblongata</td>
<td>d) the main autonomic center</td>
</tr>
<tr>
<td>5. Cerebellum</td>
<td>e) relay station for sensory impressions</td>
</tr>
<tr>
<td>6. Cerebrum</td>
<td>f) the intellectual center of the brain</td>
</tr>
<tr>
<td>7. Occipital lobe</td>
<td>g) primary hearing center</td>
</tr>
</tbody>
</table>


__________________________________________________________________________
4. a) The recticular activating system is located ______________________.
   
   b) Its two functions are:

5. Which part of the brain co-ordinates muscle tone and movement?

6. The pituitary gland is located on the undersurface of the (a) ________________.
   It is protected inferiorly by (b) ________________.

7. There are (a) ____________________ cerebral hemispheres connected by a structure called (b ____________________).

8. Where is CSF found? What is its main function.

9. a) The coverings of the brain and spinal cord are collectively called ____________.

   b) From the skull proceeding to the brain, name these covering layers.

   c) Between which two layers is the subarachnoid space located?

   d) What is found in this space?
10. Name the four main lobes of each cerebral hemisphere.

________________________________________________________________________

________________________________________________________________________

11. The spinal cord extends from the (a)______________ to the lower level of the first lumbar vertebra. The termination of the spinal cord is known as the (b)___________________________.

12. All venous blood from the brain drains into the___________________________.

13. The brain receives over 50% of the total cardiac output. (True or False).

30 TOTAL
SELF-ASSESSMENT ANSWERS: CENTRAL NERVOUS SYSTEM

1. Brain stem

2. 1. e  
   2. g  
   3. d  
   4. b  
   5. c  
   6. f  
   7. a

3. The flow, as seen from above, comprised of the bones behind the nose and above the roof of the mouth.

4. a) Throughout the brain stem, but primarily in the medulla oblongata.  
   b) - control of wakefulness and consciousness  
      - responsible for the protective mechanism of the arousal reaction in response to certain stimuli.

5. Cerebellum

6. a) Hypothalamus  
    b) Bone (skull)

7. a) Two  
    b) The corpus callosum

8. CSF is found in the ventricles of the brain, in subarachnoid space around the brain and in the spinal cord. The main function of CSF is protection.

9. a) Meninges  
    b) Dura mater  
       Arachnoid mater  
       Pia mater  
    c) Arachnoid mater and pia mater  
    d) Cerebrospinal fluid (CSF)
10. Frontal, temporal, parietal, occipital

11. a) Foramen magnum
b) Conus medullaris

12. Internal jugular vein

13. False (the brain receives a large % of the cardiac output = 16% but not 50%).
PERIPHERAL NERVOUS SYSTEM

Neurons (nerve cells) are comprised of a cell body (containing the nucleus) and an extension called the axon which terminates either at a synapse (junction with another nerve fibre) or an end plate in a muscle or organ.

Peripheral nerves consist mainly of long axons running the whole length of the nerve. The cell bodies of motor nerves are located inside the spinal cord; while those of sensory nerves are just outside the spinal cord in groups called ganglia (singular-ganglion). Autonomic (involuntary) nerves are connected together by a chain of ganglia lying adjacent to the spinal column in the thoracic and abdominal cavities.

Because of differences in function, the peripheral nervous system is classified as either:

- Somatic (voluntary) or,
- Autonomic (involuntary)

SOMATIC NERVOUS SYSTEM

Many of our activities, such as walking, raising a hand or opening our mouth are actions that involve skeletal muscles and are under our conscious control. The innervation of the muscles controlling these voluntary actions is referred to as the somatic nervous system.

The somatic nerves are usually studied in two convenient anatomic groups: spinal nerves and cranial nerves.

SPINAL NERVES

Arising from the lateral aspect of the spinal cord are nerves that transmit impulses to and from skeletal muscle. There are 31 pairs of such nerves know as spinal nerves which correspond to the segments of the spinal cord. (8 cervical, 12 thoracic, 5 lumbar, 5 sacral, 1 coccygeal). These fibres however enter and exit the spinal column between L₁ and L₂.
When the spinal cord is cut transversely (across), the tissue will show two areas of different colour. There is a central H-shaped dark area and an outer white area. The dark or gray area is chiefly composed of nerve cell bodies, while the white matter is chiefly made up of nerve fibres (axons).

Attached to the dorsal aspect of the spinal cord, are the sensory roots. Nerves bring stimili to the spinal cord from skeletal muscle through the sensory or dorsal roots.

Attached to the ventral aspect of the spinal cord, are the motor roots of the spinal nerves. These nerves carry impulse from the spinal cord to the skeletal muscles.

The spinal cord provides the two-way communication system between the brain and body parts.
Dermatomes indicate areas of body innervated by specific spinal cord segments. Note that the spinal cord segments do not correspond with vertebral bodies and the spinous processes correspond with neither.

**FIGURE 10: DERMATOMES**
CRANIAL NERVES

There are 12 pairs of nerves that arise from the base (bottom) of the brain. Because these nerves arise from within the skull, they are referred to as cranial nerves.

The cranial nerves are important for such functions as sight, hearing, vision, heart action, digestion and respiration. You will note that some of these nerves more appropriately belong to the autonomic system since they either partially or exclusively control involuntary functions, e.g. III, VII, IX, X.

FIGURE 11: BASE OF BRAIN SHOWING ORIGIN CRANIAL NERVES I – XII
<table>
<thead>
<tr>
<th>NERVE</th>
<th>FUNCTION(S)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Olfactory</td>
<td>Smell</td>
</tr>
<tr>
<td>II. Optic</td>
<td>Vision</td>
</tr>
</tbody>
</table>
| III. Occulomotor | Equality and reaction of pupils.  
                        Eye movement, eyelid elevation, pupil constriction, consensual reaction |
| IV. Trochlear | Eye movement                                                                                                                              |
| V. Trigeminal | Chewing  
                        Sensations of the face (facial anaesthesia)  
                        Corneal reflex                                                                 |
| VI. Abducens  | Eye movement                                                                                                                             |
| VII. Facial   | Facial expressions (look for unequal facial  
                        Expression – e.g. stroke. Inability to close one eye)  
                        Taste                                                                 |
| VIII. Acoustic (cochlear, hearing, equilibrium vestibular) | “Ringing in the ears” (tinitis)  
                        Nystagmus                                                                 |
| XI. Glosso-pharyngeal | Swallowing  
                        Sensations of the throat and tonsils  
                        Pharyngeal/gag reflex (sensory)                                                                 |
| X. Vagus      | Autonomic functions – lungs, heart, G.I. tract, bladder  
                        Gag reflex response (motor)                                                                 |
| XI. Spinal    | Shoulder movement, head accessory rotation                                                                                               |
| XII. Hypoglossal | Tongue movement                                                                                                                          |

The 12 cranial nerves leave the brain in various locations, pass through the subarachnoid space and then through small holes in the skull to arrive at their destination sites. Prehospital evaluation of cranial nerve function has a low priority, except for pupillary response (cranial nerve III).
AUTONOMIC NERVOUS SYSTEM

There are many tissues and organs in our body that we cannot consciously or voluntarily control. Such activities as heart contractions, digestive processes, dilation and constriction of blood vessels and pupil size are all done automatically or involuntarily. The innervation of these muscles and glands is by the autonomic nervous system.

Anatomically, the parasympathetic (or craniosacral) division of the autonomic nervous system accompanies cranial nerves from the brain and from spinal nerves at the lower end of the spinal cord (Figure 12).

All sympathetic (or thoracolumbar) nerves arise with spinal nerve along the mast of the spinal cord. They are connected by a chain of ganglia running parallel to the spinal column.

In general the sympathetic and parasympathetic divisions of the autonomic nervous system have opposing actions. For example, the stimulation of the heart by the sympathetic division will increase the heart rate, while simulation by the parasympathetic division will slow the heart.

The sympathetic system, (SNS) prepares the body for stressful situations and emergencies, requiring fast action and great exertion. It increases the hear rate and respiration, dilates pupils, curtails digestion, and makes you hair “stand on end”.

The parasympathetic system, (PNS) restores the body to normal conditions when the emergency has diminished. The system promotes digestion, slows the heart rate and breathing to normal and causes the pupils to return to normal size.
FIGURE 12: INNERVATION OF ORGANS BY THE SYMPATHETIC AND PARASYMPATHETIC DIVISIONS OF THE AUTONOMIC NERVOUS SYSTEM

AUTONOMIC

Parasympathetic Outflow (with cranial nerves)

Motor & Sensory Function (Somatic Nervous System)

Sympathetic Outflow

Temperature Control

Blood Vessels

Parasympathetic Outflow

Bladder, Bowel, External Genitalia

Head
An important function of the sympathetic nervous system is the control of blood vessels. Most of these vessels are constricted by SNS stimulation of the smooth muscle layer within the vessel. However, in order to provide more blood flow under stressful conditions to the areas they serve, the coronary vessels and skeletal muscle vessels are dilated. By controlling peripheral blood vessel diameter, the SNS is capable of regulating both cardiac output and arterial pressure. (Constriction of veins and venous reservoirs increases cardiac output; constriction of arterioles increases peripheral resistance, which elevates arterial blood pressure.)

<table>
<thead>
<tr>
<th>TABLE 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AUTONOMIC EFFECTS ON VARIOUS ORGANS OF THE BODY</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ORGANS</th>
<th>EFFECT OF SNS STIMULATION</th>
<th>EFFECT OF PNS STIMULATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>EYE</td>
<td>Dilated pupil</td>
<td>Contracted pupil</td>
</tr>
<tr>
<td>SWEAT GLANDS</td>
<td>Copious sweating</td>
<td>None</td>
</tr>
<tr>
<td>HEART</td>
<td>Increased activity HR</td>
<td>Decreased activity HR</td>
</tr>
<tr>
<td></td>
<td>Vasodilated coronaries</td>
<td>Constricted coronaries</td>
</tr>
<tr>
<td>SYSTEMIC BLOOD VESSELS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal</td>
<td>Constricted</td>
<td>No action</td>
</tr>
<tr>
<td>Muscle</td>
<td>Dilated</td>
<td>No action</td>
</tr>
<tr>
<td>Skin</td>
<td>Constricted or dilated</td>
<td>No action</td>
</tr>
<tr>
<td>LUNGS</td>
<td>Dilated bronchi</td>
<td>Constricted bronchi</td>
</tr>
<tr>
<td>GUT</td>
<td>Decreased peristalsis</td>
<td>Increased peristalsis</td>
</tr>
</tbody>
</table>

Other autonomic effects from SNS stimulation where the PNS has no effect include:

- Increased glucose released to blood from liver
- Decreased urinary output from kidneys
- 50% increase in basal metabolism
- Increased adrenal cortical secretion
- Increased mental activity.
ADVANCED LIFE SUPPORT
PRE COURSE
NERVOUS SYSTEM

SELF-ASSESSMENT: PERIPHERAL NERVOUS SYSTEM

Marks

[1] 1. The innervation to the muscles involved in such activities as running and throwing a baseball is known as the ___________________________ nervous system.

[2] 2. What is the autonomic nervous system?

[1] 3. What is the effect on the heart when stimulated by the sympathetic division of the autonomic nervous system?

[5] 4. For the following body actions indicated whether the sympathetic or parasympathetic division is predominant.
   a) Dilation of pupils _____________________________
   b) Digestive processes slowed ___________________________
   c) Increased ventilation ___________________________
   d) Body is prepared for any emergency situation ___________________________
   e) Digestive processes restored to normal ___________________________
5. Name the cranial nerve that controls the pupillary reflex.

6. The main function of the spinal cord is: ________________________________.
SELF-ASSESSMENT ANSWERS: PERIPHERAL NERVOUS SYSTEM

1. Somatic

2. Autonomic nervous system is the innervation of organs and glands that function automatically.

3. Increases heart rate.

4. a) Sympathetic  
   b) Sympathetic  
   c) Sympathetic  
   d) Sympathetic  
   e) Parasympathetic.

5. Third or oculomotor.

6. Pathway for conduction of nerve impulses to and from the brain.
FUNCTIONS OF NERVOUS TISSUE

All activities that we are involved with every day of our lives are controlled by the nervous system.

We have all experienced one of the joys of life, basking in the sunlight of an early summer morning. Who would not count as one of the great things in life, the warmth of the sunlight, a gentle breeze rustling through the trees, the singing of the birds or the sight and smell of flowers as we marvel at the world around us.

From this beautiful, but simple experience, we can identify the chief functions of the nervous system.

1. Transmission of the Nerve Impulse

   The various sensations that have been briefly delineated in our early morning walk are eventually transmitted to the brain from the eyes, ears, nose, and skin. Within the brain, the information is analyzed.

2. Interpretation

   When the impulses from our various sense organs (and all other areas of the body) are received by the brain, such impulses or messages are sorted out or interpreted. Finally, information received by the brain may be stored for future reference.

3. Storage

   The various pleasant sensations associated with a warm day may be recalled and experienced again on a later cold winter day.

   Storage of information (memory) is such a complicated function of the brain, it is entirely likely there are various areas designated for this function. Events that occurred yesterday may be stored in one area of the brain, and as time passes, moved to another brain site for long term memory.

All living organisms possess the ability to transmit nerve impulses. However, the functions of interpretation and storage are characteristic of higher forms of life, as exemplified by man.
How Is The “Message” Carried?

The “message” refers to the impulses (stimuli) that are relayed (transmitted) from tissues and organs that comprise the body, to the spinal cord and brain. It is also apparent that messages are sent from the brain and spinal cord to all the components that make up the human body.

Such information is carried by neurons, the functional units of nervous tissue. Neurons are found throughout the body. It has been estimated that the number of neurons within the brain along may exceed twenty billion.

Functionally, neurons may be divided into two main types:

1. Sensory (Afferent) Neurons

   The sensory neurons are concerned with conveying impulses to the brain and spinal cord (central nervous system) from tissues and organs any place in the body, e.g. fingers, toes.

2. Motor (Efferent) Neurons

   The motor neurons are concerned with carrying impulses away from the brain and spinal cord to all structures in the body.

   There are, in addition to sensory and motor neurons, neurons that connect or join motor and sensory neurons. These are exclusive to the brain and spinal cord, and are referred to as internuncial neurons or interneurons.

Although sensory and motor neurons are functionally different, they are similar in structure. Impulses are carried by both in the same manner. The impulse travels from the dendrite end of the neuron to the motor end plate (or synapse) of the neuron.

FIGURE 13: DIRECTION OF NERVE IMPULSE WITHIN A NEURON

[Diagram of a neuron showing the direction of impulse travel from dendrites to cell body to axon to motor end plate (or synapse).]
Efferent fiber arrangement is slightly different in each of the peripheral nervous systems.

The somatic (cranial and spinal nerves) efferent pathway to the skin and skeletal muscles consists of only one neuron. In contrast, in the autonomic system, impulses to visceral structures consists of one neuron from the brainstem or spinal column to a ganglion outside the spinal cord (preganglionic neuron), and one from the ganglion to the end organ known as the visceral effector.

Naturally, this fiber is named postganglionic neuron. This concept assumes clinical importance when discussing neurotransmitters.

**EXCITABILITY AND CONDUCTIVITY**

Nervous tissue has the property of excitability, which means it has the ability to respond to a change in the environment. When stimulated, nervous tissue also has the property of conductivity, which is the ability to transmit nerve impulses to the central nervous system. “Messages” or “orders” are then conveyed back to the various effectors to make adjustments.

Nerve impulses are then initiated by receptors in tissues, in response to changes in the internal and external environments.

Nervous tissue has the ability to respond to a stimulus. This response is manifested by the transmission of the nerve impulses and is a complex action. The transmission of electrical impulses (illustrated in Figure 14) along the axon membrane can be briefly explained as follows:

- **A resting neuron** is in a non-conducting state (Figure 14A). The semi-permeable membrane is polarized through the active transport of Na+ (sodium pump). When the outside of the semi-permeable membrane is more positive, relative to the inside, it is said to have a resting potential.

- When a stimulus is applied, a momentary depolarization occurs (Figure 14B) resulting in a sudden reversal of the passage of Na+ through the semi-permeable membrane. Sodium ions now quickly enter the neuron from the extracellular fluid area. (Some potassium ions from the intracellular fluid within the neuron diffuse out at the same time.) Although the sodium and potassium ions are both cations (positively charged) the differing amounts intracellularly and extracellularly create a relative difference in the electrical charges inside and outside the semi-permeable membrane of the neuron.

- The reversal of polarization results in an action potential which allows the stimulus to be carried along the nerve fiber (Figure 14C). Once an action potential is reached within a neuron, impulse conduction will occur along the full length of the fiber (all or none principle).
The action potential continues to travel along the membrane while immediately “behind” it occurs the recovery of the resting potential and a return towards a relative positive charge outside of the semi-permeable membrane (Figure 14D).

Once repolarization is achieved, an impulse may again be initiated and carried.
SYNAPTIC TRANSMISSION OF THE NERVE IMPULSE

A synapse is a very small cleft, located between the end of one neuron (presynaptic neuron) and the dendrite end of the adjacent neuron (postsynaptic neuron) (Figure 15). Tiny sacs or vesicles, each containing a chemical neurotransmitter, are located in the end of the presynaptic neuron.

When the action potential (wave of depolarization) reaches the end of the presynaptic neuron, the vesicles move to the surface of the axon membrane. The vesicles fuse with the terminal surface and release their contents into the synaptic cleft (Figure 16).
The neurotransmitter molecules diffuse across the synapse and contact the membrane of the postsynaptic neuron (Figure 17). Here the neurotransmitter binds to specific protein sites (neurotransmitter receptors). This action facilitates the opening of channels in the membrane which increases the permeability of the membrane to sodium, e.g. creates a site of depolarization. This act initiates the depolarization of adjacent portions along the axon, giving rise to the nerve impulse in the postsynaptic neuron.

**FIGURE 17: NEUROTRANSMITTER**

Knowledge of the neurotransmitter substance is important in understanding the disease processes involving them, as well as the effects of various medications upon the mechanism of impulse transmission at the synapse.

Although there are many different neurotransmitter substances, the most notable are listed below.

**Neurotransmitter**

- **Somatic nervous system** ................................................................. Acetylcholine
- **Parasympathetic – preganglionic fibers** ............................................. Acetylcholine
  - **– postganglionic fibers** ................................................................. Acetylcholine
- **Sympathetic – preganglionic fibers** ............................................... Acetylcholine
  - **– postganglionic fibers** ................................................................. Norepinephrine (Noradrenalin)
*Although within the sympathetic nervous system norepinephrine is generally the neurotransmitter communicating between the postganglionic nerve fiber and the effector organ, there are instances where acetylcholine acts the neurotransmitter communicating between the postganglionic nerve fiber and the effector organ. For instance, sweat glands and skeletal muscle blood vessels both receive postganglionic sympathetic innervation via the neurotransmitter acetylcholine.

**Summary**

The nerve impulse that occurs along a series of neurons is an **electrochemical** process; it is electrical along the axon membrane and chemical at the synapse. The wave of depolarization continues over succeeding neurons, until the message reaches the brain, where interpretation occurs.
ADVANCED LIFE SUPPORT
PRECOUSE
NERVOUS SYSTEM

SELF-ASSESSMENT: TRANSMISSION OF NERVE IMPULSES

Marks

[1] 1. What is the functional unit of nervous tissue?

[2] 2. A synapse is found between the __________________________ and __________________________.

[2] 3. When one touches an ice cube, the impulses are carried to the brain via (a) __________________________ neurons. The message from the brain to the fingers are carried by (b) __________________________ neurons.

[2] 4. For the sympathetic nervous system the chemical released by all preganglionic nerve fibers is (a) ___________. The chemical released by most postganglionic nerve fibers of the sympathetic nervous system is (b) _________________.

[1] 5. The direction of the nerve impulse is from the postsynaptic the the presynaptic neurons. (True or False)

[1] 6. Neurotransmitters are chemicals found at the terminal end of the presynaptic neuron. (True or False)

[3] 7. A change in the internal or external environment of a neuron is known as a (a) _________________. The property of nervous tissue to respond to a change in the environment is known as (b) __________________________. The ability of nervous tissue to transmit nerve impulses is the property of (c) __________________________.
8. The nerve impulse is a wave of ______________________ along the axon membrane.

9. In the following statements indicate which term is most appropriate.

   (polarization, depolarization or repolarization)

   a) A stimulus causes a reversal of the polarity of the axon membrane, creating an electropositive interior and an electronegative exterior. ________________

   b) The restoration of the membrane potential. ____________________________

   c) Due to the fact that in the resting neuron, sodium ions move to the exterior of the cell membrane, the external surface of the axon membrane is electropositive and the interior is electronegative.

10. Describe how the nerve impulse crosses the synapse.

11. Once the threshold of stimulation has been reached, a wave of depolarization (nerve impulse) spreads along the axon. This law is known as the ________________ ________________.

20 TOTAL
SELF-ASSESSMENT ANSWERS: TRANSMISSION OF NERVE IMPULSES

1. Neuron

2. End of one neuron and the dendrite (or cell body) of the next neuron.

3. a) Sensory (afferent)  
   b) Motor (efferent)

4. a) Acetylcholine  
    b) Norepinephrine

5. False

6. True

7. a) Depolarization  
    b) Excitability  
    c) Conductivity

8. Depolarization

9. a) Depolarization  
    b) Repolarization  
    c) Polarization

10. The transmission of the nerve impulse across the synapse is chemically mediated. The chemical, known as, a neurotransmitters is deposited into the synaptic cleft in the face of a wave of receptor sites of the postsynaptic neuron. This is the stimulus for increased membrane permeability to sodium and the initiation of the nerve impulse in the postsynaptic neuron.

11. All-or-nothing law.
CEREBROVASCULAR DISEASES

Cerebrovascular diseases are a prevalent group of disorders related to a lack of sufficient blood flow to brain tissues (cerebral ischemia). Blood flow to cerebral tissues can be impaired in one of three general ways:

- Thrombosis – secondary to artherosclerosis (70 – 80%)
- Embolism (5 – 10%)
  - thromboembolism (travelling blood clot)
  - air embolism (travelling gas bubble), e.g. decompression sickness
  - fat embolism (travelling piece of artherosclerotic plaque)
- Hemorrhage due to
  - hypertension (10 – 20%)
  - ruptured cerebral aneurysm (5 – 10%)
  - intracranial mass (5 – 10%)

Transient Ischemic Attacks (TIA’s) are short-lived, episodic periods of cerebral ischemia that usually last 5 to 30 minutes, and may extend up to 24 hours. These attacks disappear spontaneously with no resultant neurological deficit.

TIA’s may be prodromal to an actual stroke (CVA) and can present with any or all of the following:

- Numbness
- Weakness on one side of the body
- Speech difficulties
- Blurring of vision or blindness in one eye
- Dizziness
- Mental confusion
- Drop attacks, with or without loss of consciousness.

Clinical vignette
These findings are not necessarily unique to TIA’s.
All of the following factors predispose people to atherosclerosis, which can lead to CVA:

- Age
- Hypertension
- Smoking
- Elevated serum lipid levels
- Diabetes mellitus
- Male
- Obesity

Stroke symptoms usually appear suddenly and progress quickly. The signs and symptoms for a CVA depend on the extent of involvement of neural tissue and the side of the cerebrum that is affected. The potential for life-threatening problems may be immediate.

Some patients who have suffered a thrombotic ischemic stroke can be treated with fibrinolytic therapy. This therapy is most efficacious when the patient receives treatment within three hours of symptom onset.

**SEIZURES**

Seizures are periods of abnormal electrical activity in the brain (neurological dysfunction). A seizure is a symptom, not a disease and may present from an idiopathic, a metabolic, or a traumatic origin.

Grand mal seizures present with a sudden loss of consciousness, extensor muscle spasms (tonic stage), apnea and then bilateral clonic movements (generalized shaking). Once the seizure stops, the patient then passes into a postictal stage identified by muscle relaxation, deep breathing and a slowly increasing level of consciousness or sleep.

Focal seizures usually occur unilaterally and are caused by a focal brain lesion. These may present with only one body part involved or may evolve from this to include the whole body, i.e. a grand mal seizure.

These are the most common presentations of seizures. It is important to note that there is no such thing as a benign seizure and that uncontrolled seizures kill brain cells.

The consequences of full body convulsions may include:

- Hypoxia
- Acidosis
- Cardiac arrhythmias
- Circulatory collapse
- Fever
- Increased ICP.
HEAD INJURY

One of the most common traumatic insults is head injury. In the prehospital setting, it must be emphasized that all patients with head injuries from trauma must be considered to have neck or spinal injuries.

In reviewing head injury itself, lacerations, fractures, concussion, cerebral contusion and intracranial, bleeding must be considered.

Scalp lacerations must be cared for as a secondary consideration in relation to other neurological injuries of the skull, brain and spinal cord. However, these wounds should be managed quickly due to the fact that they bleed profusely and may contribute to the development of shock (especially in young children).

SKULL FRACTURES

Skull fractures may be classified as:

- **Simple** – linear crack or cracks (comminuted) in the skull surface with no displacement.

- **Depressed** – part of the skull is indented allowing possible associated complications, e.g. intracranial infection, CSF leakage, profuse bleeding, underlying brain contusion and/or tears in the cerebral tissue.

- **Basal (Basilar)** – involves the base of the skull, e.g. floor, when seen from above. (For the paramedic the classic signs of a basilar skull fracture, e.g. the raccoon eyes, Battle’s sign are usually not apparent for several hours.)

It should be emphasized that a skull fracture does not necessarily indicate a cerebral injury and vice versa, a cerebral injury may occur without a skull fracture.

BRAIN INJURY

**Concussion** is a transient, limited process that usually occurs as a results of a direct blow to the head, or from an acceleration/deceleration injury, in which cerebral tissue impacts with the inside of the skull. It involves a period of unconsciousness. The brief interruption of the RAS which causes temporary unconsciousness may also cause a short period of amnesia. There is no actual damage to brain tissue.

A **contusion**, an actual bruise of the brain surface resulting in a structural alteration of the brain, is caused by the brain raking over the bony irregularities inside the skull. These patients are unconscious, at least initially. With any contusion there is actual damage to the brain of variable degree.

In either uncomplicated concussion or contusion, the patient will remain stable or improve. These injuries, as well as any direct trauma are known as **primary** injuries. **Secondary** injuries resulting from associated hemorrhage and/or edema may be life-threatening since the result is an increase
in intracranial pressure (ICP). The consequences of this condition are important for the prehospital care worker to understand.

**Intracranial hemorrhage** frequently results from blunt head trauma. It usually presents with a loss of consciousness (due to an interruption in the electrical activity in the brain) and with motor and/or sensory hemorrhage most often results from a cerebrovascular accident (CVA), or a ruptured cerebral aneurysm (subarachnoid hemorrhage). The hemorrhage may be **epidural**, **subdural**, **subarachnoid** or **intracerebral**.

1. An epidural (or **extradural**), **hemorrhage** is bleeding between the skull and dura mater. It is usually caused by acute head trauma from a direct blow which results in a skull fracture (50%) and a torn middle meningeal artery (temporal lobe skull fracture). Epidural hemorrhage occurs in 1-2% of patients with severe head injuries.

   This injury should be suspected if there is a momentary loss of consciousness followed by a lucid interval and then a decreasing level of consciousness. In about 5% of patients this is the classic observation; usually there is no lucid interval.

2. A **subdural hemorrhage** is caused by active bleeding (usually venous) between the dura and arachnoid mater. It occurs frequently in patients with blood dyscrasias or those taking anticoagulants. Subdural venous bleeding may also be caused by head trauma, usually an acceleration/deceleration incident.

   Subdural hemorrhage occurs post trauma six times more frequently than does epidural hemorrhage. This type of hematoma may occur acutely, shortly following trauma, or chronically, taking days, weeks or months to manifest symptoms.

   The development of a chronic subdural hematoma following trauma is especially common in the elderly and chronic alcoholics. This is due to brain shrinkage (atrophy) and the resultant stretching of blood vessels associated with these conditions. The hematoma may gradually increase in size, thus gradually creating signs and symptoms of increased ICP.

3. **Subarachnoid hemorrhage** develops from bleeding between the arachnoid and pia mater. It usually results from the rupture of a congenital intracranial aneurysm, hypertension, or head trauma. It is the most common bleed associated with head trauma.

4. **Intracranial hemorrhage** involves bleeding into cerebral tissue. It occurs in 1-2% of patients with head injury, but may also occur spontaneously.

Lesions or bleeding in the head are named according to their location in respect to the three meningeal layers of the brain or the structure involved. We have discussed that the brain is a very delicate collection of tissues cushioned by CSF and protected by the hard, bony skull which encloses the brain within a confined space. The contents of this space, i.e. blood, CSF and brain tissue, cannot increase in size without increasing pressure within this space.
Compensatory mechanisms with increased ICP are limited. They include a shift of CSF and decreased cerebral bloodflow. If ICP reaches a point where brain cells cannot be adequately perfused, the cells become hypoxic and soon permanent neuronal damage or death may result.

Tough dural tissue called tentoria (singular tentorium) support and compartmentalize various parts of the brain. When maximum compensation of CSF and bloodflow is reached, tissue begins to shift. Figure 19 shows three possible areas of shift – lateral, downward through the tentorial notch and downward through the foramen magnum. The resulting signs of brainstem compression are late signs.

**FIGURE 19: SUBDURAL HEMATOMA WITH INCREASED ICP**

- Arachnoid
- Dura
- Hematoma
- Lateral Shift
- Compression of the Occulomotor Nerve (Dilated pupil)
- Herniation of the Temporal Tissue through the Tentorial Notch (Downward shift)
- Tentorium
- Herniation of cerebellar tissue through the Foramen Magnum
- Compression of Brainstem (Brainstem Signs)
- Midline shift
ASSESSMENT OF BRAIN FUNCTION

The **Glasgow Coma Scale (G.C.S.)** is a widely used guide to measure changes in brain function. This scale is designed to allow the person assessing the patient to quantitatively relate the level of consciousness to the various “motor”, “verbal” and “eye opening” response of the patient as determined by a set of standardized stimuli*. The prehospital worker should assess the patient according to each section and tabulate a total score (8 or less indicates coma). The higher the score, the greater the degree of cerebral arousal.

The G.C.S. allows a maximum of 15 points. Testing is divided into three categories:

- **Eye opening**
- **Verbal response**
- **Motor response.**

**Eye Opening**

The examiner determines the **minimum stimulus** that evokes opening of one or both eyes. If the patient cannot realistically open the system because of bandages or lid edema, write “E” after the total test score to indicate omission of this component.

- **4 points**  Eyes open spontaneously.
- **3 points**  Eyes open to speech. Patient opens eyes in response to command or on being called by name.
- **2 points**  Eyes open to noxious stimuli.
- **1 point**  No eye opening in response to noxious stimuli.

**Best Verbal Response**

The examiner determines the **best** response after arousal. Noxious stimuli are employed if necessary. Omit this test if the patient is dysphasic, has oral injuries, or is intubated.

- **5 points**  Oriented patient. Can converse and relate who he is, where he is and the year and month.
- **4 points**  Confused patient. Is not fully oriented or demonstrated confusion.
- **3 points**  Verbalizes. Does not engage in sustained conversation, but uses intelligible words in an exclamation (curse) or in a disorganized manner which is nonsensical.
- **2 points**  Makes moaning or groaning sounds that are not recognizable words.
- **1 point**  No vocalization. Does not make any sound even in response to noxious stimulus.
Best Motor Response

The examiner determines the best response with either arm.

6 points  Obeys simple commands. Raises arm on request or holds up specified number of fingers. Releasing a grip (not grasping, which can be reflexive) is also an appropriate test.

5 points  Localizes noxious stimuli. Fails to obey commands but can move either arm toward a noxious cutaneous stimulus and eventually contacts it with the hand. The stimulus should be maximal and applied in various locations, i.e. sternum pressure or trapezius pinch.

4 points  Flexion withdrawal. Responds to noxious stimulus with arm flexion but does not localize it with the hand.

3 points  Abnormal flexion. Adducts shoulder, flexes and pronates arm, flexes wrist and makes a fist in response to a noxious stimulus (formerly called decorticate rigidity).

2 points  Abnormal extension. Adducts and internally rotates shoulder, extends forearms, flexes wrist and makes a fist in response to a noxious stimulus (formerly called decorticate rigidity).

1 point  No motor response. Exclude reasons for no response, e.g. insufficient stimulus.

REFLEXES

When we speak of subcortical activity associated with motor function, we are referring to primitive responses. Reflex arcs and nerve pathways consisting of two or three neurons, are the structural and functional units of reflex activity.

A reflex is a protective involuntary response to a stimulus. Somatic reflexes are contractions of skeletal muscles; while autonomic (or visceral) reflexes consist either of contractions of smooth or cardiac muscle, or secretion by glands.

One reflex commonly tested by prehospital personnel during insertion of an oropharyngeal airway is the gag. Its presence is protective of oropharynx in that its stimulation results in a forceful outward ejection of the irritant, sometimes accompanied by vomiting.

Another reflex you encounter frequently is pupillary response to a light stimulus. Parasympathetic fibers of the third cranial nerve (oculomotor) pass through a narrow opening in the dura (tentorial notch), which separates the cerebrum from the cerebellum. With increased ICP originating above this area, e.g. from any expanding lesion in the cerebrum or its meninges, third nerve compression occurs at this notch (Figure 19). The reflex response to light is lost on the same side as the compression. Bilateral pressure causes bilaterally fixed dilated pupils.
Both pupillary response and gag reflexes are cranial reflexes. Spinal reflexes tested in the field indicated whether sensory function exists and whether motor function is intact. This is best demonstrated by the application of a painful stimulus for patients not displaying voluntary motor activity.

**FIGURE 20: PATHWAY OF REFLEX THROUGH THE SPINAL CORD**

FACIAL TRAUMA

Facial trauma should be considered separately, when discussing head injury, since it is one of the most commonly seen types of trauma. Facial trauma is mentioned here to point out that, despite the profuse bleeding that may be present, other concurrent injuries of a more serious nature may be developing, e.g. airway problems, shock, spinal cord injuries and intracranial problems.

SPINAL CORD INJURY

Spinal cord injuries are common problems dealt with by the prehospital care worker. The consequences of spinal cord injury depends on the location and extent of the damage sustained and may include spinal shock, sensory losses, or motor losses; all of which may be transitory or permanent. Associated paralysis may be flaccid (accompanied by muscle atrophy) or spastic (accompanied by uncontrolled muscular spasms).
C3, C4, and C5 sections of the spinal cord contain the phrenic nerve which innervates the diaphragm. Injury to this area will result in a loss of control of the main muscle ventilation. Therefore ventilation must be supported if the patient is to survive.

It should be stressed that vertebral injuries may be present without spinal cord injury. The potential for cord injury is always present and careful handling of these patients is essential.

**FIGURE 21: SPINAL CORD INJURIES**

There are three basic types of spinal column injuries:

- Wedge or compression fracture
- Burst fracture
- Subluxation and/or fracture/subluxation (extremely unstable).
The most common mechanisms of injury for spinal column injuries are:

- Hyperflexion
- Hyperextension
- Axial loading
- Penetrating wounds.

FIGURE 22: MECHANISMS OF SPINAL CORD INJURIES

- Hyperflexion
- Axial Loading
- Hyperextension

- Spinal Cord
- Ligament
- Disc

- Distribution of Disc and Interspinous Ligament
- Burst Vertebral Body with Cord Compression
- Compressed Interspinous Ligament Disruption of Intervertebral Disc
Spinal injuries at the time of occurrence may be complete (with no neurological function) or incomplete (with some neurological function).

Cervical and lumbar spine injuries are the most common type of spinal cord injury. However, another vulnerable area is at the point where the fixed thoracic spine joins the lumbar spine. Trauma to the thoracolumbar junction (T10 to L2), from either a direct blow or from flexion-rotational forces, may produce a very unstable spinal injury. If this type of injury is suspected, the paramedic should be altered to associated intra-abdominal or intrathoracic injuries which may well be life-threatening.

Whiplash injury, most commonly a result of a rear-end automobile accident, may produce damage to muscles, discs, ligaments, and nervous tissue in the region of the cervical spine. It is usually caused by violent hyperextension and flexion of the neck.

It must be emphasized that a cervical spine injury must be assumed in any patient presenting with:

- Injuries above the level of the clavicles
- An altered level of awareness associated with possible trauma
- Multiple severe injuries.

With this in mind, appropriate precautions should be taken when assessing and treating these patients.
SELF-ASSESSMENT: PATHOLOGIES OF THE NERVOUS SYSTEM

Marks

[1] 1. What two kinds of nerve fibers are necessary for a reflex action involving the spinal cord.

__________________________________________________________________________

__________________________________________________________________________

[1] 2. The three general causes of seizures include:

__________________________________________________________________________

__________________________________________________________________________

__________________________________________________________________________

[1] 3. The most rapid increase in ICP will probably be due to what pathological process?

__________________________________________________________________________

__________________________________________________________________________

__________________________________________________________________________

[1] 4. Which two groups of individuals are more likely to develop a chronic subdural hematoma.

__________________________________________________________________________

__________________________________________________________________________

__________________________________________________________________________

[2] 5. An epidural hemorrhage is found a) ________________________, often secondary to b) ________________________.
6. You have applied a painful stimulus to an unresponsive patient. Utilizing the GCS, list in order what specific actions (or observations) you would have performed (or noted) prior to this action.

7. What is the differences between a TIA and a CVA?

8. The two most vulnerable areas of the spinal column in the traumatized patient are:

9. Cranial reflexes tested in the field include:
10. The general causes of cerebral ischemia include:

____________________________________________________

____________________________________________________

____________________________________________________

19 TOTAL
SELF-ASSESSMENT ANSWERS: PATHOLOGIES OF THE NERVOUS SYSTEM

1. Sensory and motor fibers.

2. Metabolic
   Traumatic
   Unknown (idiopathic).

3. Arterial bleeding.

4. The elderly and the alcohol abuser.

5. a) Outside the dura, under the skull
    b) Temporal skull fracture associated with middle meningeal artery tear.

6. 1. Observed for spontaneous eye opening
    2. Observed for eye opening to speech
    3. Observed for any verbal response
    4. Observed for any motor response to verbal command.

7. Although the initial presentation may be similar, a TIA resolves spontaneously, usually within 24 hours or less with no residual neurologic deficit. The ischemia is of a transient rather than a permanent nature.

8. The cervical spine
   The lumbar spine.

9. Pupillary response to light
   Gag.

10. Thrombosis
    Embolism
    Hemorrhage.
ADVANCED LIFE SUPPORT
PRE COURSE
NERVOUS SYSTEM

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.

   Reading  ___________ hours
   Self assessment  ___________ hours
   Total time  ___________ hours

2. Were the objectives of the module clearly stated?

   [ ] yes  [ ] no
   If no, please comment.

3. Did you see any of the resource materials?

   [ ] yes  [ ] no
   If yes, which items
   __________________________________________________________________
   __________________________________________________________________
   __________________________________________________________________
   Were they helpful?
   __________________________________________________________________

4. Were the reference notes adequate?

   [ ] yes  [ ] no
   If no, please comment.

5. Were the reference notes easy to follow?

   [ ] yes  [ ] no
   If no, please comment.
6. Were the examples provided satisfactory?

[ ] yes [ ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[ ] yes [ ] no
If yes, please specify.

8. Was the level of the module satisfactory for your program of study?

[ ] yes [ ] no
If no, please comment.

Base Hospital

9. General comments or suggested improvements.
OJECTIVES: THE ABDOMEN

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. describe the structure of the abdomen, in terms of muscles, organs, peritoneum, attachments, vasculature and bones.

2. describe the location of intraabdominal organs in terms of anatomical landmarks, and/or quadrants and regions.

3. describe the location, within the abdomen, of the abdominal structures in relation to other organs.

4. given an abdominal surgical scar, indicate the probable previous surgical procedure.

5. describe the nature of somatic and visceral pain.

6. explain "referred pain" and give examples.

7. given a patient presenting with pain/trauma/mass in a given location, be able to describe the likely abdominal structures involved.

8. briefly describe the clinical findings associated with the patient described in objective 7.

If you have studied this subject previously, you may test your ability using the self-assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.

Author’s Note:

Appreciating the three-dimensional orientation of abdominal organs is difficult. If at all possible, I would strongly suggest the student try to attend an autopsy dissection.
INTRODUCTION

Patients often seek emergency care as a result of abdominal pain and trauma. Potentially catastrophic causes of abdominal pain include:

- ruptured abdominal aortic aneurysm
- perforated bowel
- ectopic pregnancy
- ruptured spleen

Victims of motor vehicle collisions often present with blunt abdominal trauma, while victims of crime may suffer stab wounds or gunshot wounds. Either blunt or penetrating trauma is capable of causing life-threatening hemorrhage.

To initiate appropriate prehospital care, the paramedic should have a working knowledge of the anatomy of the abdomen.
ANATOMICAL LANDMARKS

The student should take a few moments to become familiar with the following anatomic terms (Figures 1 and 2).

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>COSTAL MARGIN</td>
<td>The lower border of the rib cage.</td>
</tr>
<tr>
<td>XIPHOID PROCESS</td>
<td>The lower end of the sternum.</td>
</tr>
<tr>
<td>PELVIS</td>
<td>A bony framework consisting of the sacrum, ischium, ilium, and pubis. The pelvis protects the urinary bladder and rectum. The internal female genitilia are also within the pelvis (uterus, fallopian tubes and ovaries).</td>
</tr>
<tr>
<td>INGUINAL LIGAMENT</td>
<td>A strong band of connective tissue stretching from the ilium to the pubis.</td>
</tr>
<tr>
<td>DIAPHRAGM</td>
<td>The strong tendinous membrane separating the thoracic cavity from the abdomen.</td>
</tr>
<tr>
<td>RECTUS ABDOMINIS MUSCLE</td>
<td>The strong paired muscles running the length of the abdomen.</td>
</tr>
<tr>
<td>THORACIC VERTEBRAE</td>
<td>Numbered I to XII; abbreviated as T1 to T12</td>
</tr>
<tr>
<td>LUMBAR VERTEBRAE</td>
<td>Components of the lumbar spine; abbreviated as L1 to L5.</td>
</tr>
</tbody>
</table>

The abdomen is bordered by the dome of the diaphragm superiorly and the pelvic organs inferiorly. The abdominal contents consist of the biliary system (liver, gallbladder), pancreas, spleen, the digestive tract (stomach, small bowel, large bowel), the great vessels (aorta, inferior vena cava) and posteriorly the urinary tract (kidneys and ureters).

Paramedics should describe the location of abdominal pain or masses in terms of quadrants or regions (Figure 1). Four quadrants are formed by imaginary lines that cross at the umbilicus; right upper quadrant (RUQ), right lower quadrant (RLQ), left upper quadrant (LUQ) and left lower quadrant (LLQ).

The epigastric region is bordered by the xiphoid process, both costal margins and a horizontal line between L1 and L2.

The right and left flanks are also regions. They are bordered superiorly by the costal margin, inferiorly by the ilium, and extend laterally to approximately the anterior and posterior axillary lines.
FIGURE 1: ANATOMICAL LANDMARKS OF THE ABDOMEN

- Epigastric Region
- Flank
- Inguinal Ligament
- R.U.Q
- L.U.Q
- R.L.Q
- L.L.Q
- Costal margin
- Umbilicus
- Iliac Crest
- Anterior Superior Iliac Spine
Clinical vignette

If a base hospital physician is to be able to clearly understand the location of a patient’s pain, an abdominal mass or a gunshot wound, the paramedic must be able to describe findings in terms common to both.

Valuable information regarding a patient’s past history may result from knowledge of the various types of surgical incisions. A patient with many previous abdominal procedures may develop a bowel obstruction from adhesions. Similarly a patient with a previous abdominal bifemoral bypass graft likely has significant atherosclerosis and may present with symptoms resulting from ischemia to the brain (stroke), the heart (myocardial infarct), the bowel (mesenteric occlusion), or the limbs (arterial embolus).
COMMON INCISIONS SITES, ILLUSTRATED IN FIGURE 3, INCLUDE:

- Midline incision (#1) – often used for exploratory laparotomy, e.g. following trauma or aortic aneurysm.
- Midline plus femoral incision (#1 with #1a) – signifies preceding abdominal bifemoral bypass graft.
- Upper midline incision (#2) – may be used for surgical treatment of ulcer disease.
- Right subcostal (Kocher) incision (#3) – 2 small stab-like incision sub-xiphoid and under right costal margin and over belly button commonly used for cholecystectomy (gallbladder removal).
- McBurney incision (#4) – appendectomy.
- Lower midline incision (#5) – used for Caesarian section, hysterectomy, bladder operations or prostate removal.
- Lower transverse (Pfannenstiel) or “bikini” incision (#6) – also used for Caesarian section.
- Renal surgery scar (#7) – for any surgical procedure involving the kidney.

FIGURE 3: SURGICAL SCARS OF THE ABDOMEN
ABDOMINAL WALL

The anterior abdominal wall is a muscular structure. The muscles originate from the lower rib cage and lumbar spine and attached to the ilium and pubis in the pelvis.

The central muscle of the abdomen is a pair of muscles called the rectus abdominis muscle. Three other muscles are lateral to the rectus and are named the external oblique muscle, the internal oblique muscle and the transversus abdominis muscle. The tendinous portion of the external oblique and internal oblique muscles form the inguinal ligament in the groin. Posteriorly, the quadratus lumborum and a portion of the transversus abdominus make up the abdominal wall.

ABDOMINAL SPACES

Before describing the relationship of the various abdominal organs, it is important to understand the concept of the peritoneum.

The peritoneum is a thin layer of connective tissue that forms the inner lining of the abdominal wall and also covers many abdominal organs. The portion of peritoneum that lines the abdominal wall is referred to as parietal peritoneum. The portion that covers an abdominal organ is referred to as visceral peritoneum. The space between the parietal and visceral peritoneum is known as the peritoneal cavity.

The aorta, kidneys and pancreas are covered only anteriorly by the peritoneal reflection and are referred to as retroperitoneal structures.

Posterior to the peritoneal cavity is the retroperitoneal space. A large amount of blood can accumulate here. This is often an area of hidden blood loss as a result of pelvic fractures or rupture of an abdominal aortic aneurysm.

Clinical vignette

Pain, anxiety, fear and cold hands can cause a patient to involuntarily tense the abdominal muscles and make palpation impossible. Ideally the abdominal exam should take place with the patient lying supine, with arms at his side such that the abdomen is relaxed. Slow gentle palpations, beginning in the area that hurts least, facilitates abdominal palpation.

Defects in the abdominal wall are known as hernias. Hernias most commonly occur in the groin (inguinal hernias) but may also be found at the umbilicus (umbilical hernia) or in a previous abdominal incision (incisional hernia).
ABDOMINAL PAIN

Pain in the abdomen generally results from one of three mechanisms:

1. Distention of a hollow viscus, e.g. bowel obstruction, early appendicitis, or renal colic (ureter).

2. Ischemia, e.g. mesenteric artery occlusion (the main artery to the bowel becomes occluded and bowel becomes gangrenous).

3. Inflammation, e.g. acute cholecystitis, pancreatitis, late appendicitis.

Two types of pain are recognized – visceral and somatic.

Visceral pain results from increased tension in the wall of a hollow viscus, distention of a hollow viscus and ischemia. This discomfort tends to be dull, poorly localized and is often accompanied by sweating, nausea and vomiting. Early in the course of appendicitis, patients complain of dull crampy periumbilical pain which is poorly localized, often accompanied by nausea and vomiting. This is an excellent example of visceral pain, caused by distention of the appendix with secretions.

Somatic pain arises from the abdominal wall (skin and muscle), the parietal peritoneum and the diaphragm. This type of pain is sharper and better localized to the site of stimulation than visceral pain. Late in the course of appendicitis when the parietal peritoneum covering the appendix becomes inflamed, patients complain of sharp, severe, right lower quadrant pain and exhibit muscular rigidity. This well localized discomfort is somatic pain.
Clinical vignette

To review then, during the course of an attack of appendicitis, a patient first notes dull, poorly localized visceral pain resulting from distension of the hollow appendix. Later, the patient feels a well-localized, sharp, severe pain (somatic pain) resulting from inflammation of the parietal peritoneum.

REFERRED PAIN

One must also be cautious in evaluating patients with abdominal discomfort, as pain may be referred to the abdomen from other structures. A classic example of this “referred pain” is the patient with epigastric pain from an inferior myocardial infarction.

The phenomenon of referred pain occurs because the nerves from some abdominal structures feed into the spinal cord at a considerable distance from the diseased organ.

Pain from the abdomen may be referred to other areas, e.g. right scapular pain referred from the gallbladder and shoulder tip pain referred from the diaphragm, or ovaries and fallopian tubes. The diaphragm is innervated form cervical nerves C₃, C₄ and C₅. Hence with diaphragmatic irritation, pain is felt over the shoulder – the area of skin supplied by C₃ and C₄.
FIGURE 5: SURFACE AREAS OF REFERRED PAIN

Heart

Gallbladder

Appendix

Right kidney

Stomach

Ureter
ABDOMINAL ORGANS

The abdomen contains a number of organs. The position of these in relationship to each other is illustrated in Figure 6. The text which follows provides a brief description of each organ.

FIGURE 6: RELATIVE POSITIONS OF ABDOMINAL VISCERA
LIVER

The liver is a large triangular-shaped organ weighing about 2 kilograms. Its function as a gland is both exocrine (production of bile) and endocrine (the production of hundreds of proteins which aid protein and fat metabolism). The liver is also responsibly for the conversion of excess glucose to glycogen and is the main storage organ for glycogen.

Found in the right upper quadrant of the abdomen, the superior aspect of the liver is smooth and convex, resting under the diaphragm. Most of the gland lies behind the rib cage with only about 1 cm projecting below the costal margin in the mid-clavicular line. Hepatic enlargement (hepatomegaly) is commonly associated with many conditions. In these cases the liver may project significantly below the costal margin.

Clinical vignette

Administration of Glucagon to a hypoglycemic diabetic will breakdown glycogen to free glucose. Unfortunately, if a single dose of Glucagon does not result in an improvement of signs and symptoms, glycogen stores in the liver may be depleted and subsequent doses may not be effective.

The gallbladder lies closely adherent to the inferior aspect of the liver (located at about the 9th costal cartilage in the midclavicular line). The gallbladder stores bile, which is secreted from the liver. Bile flows from the liver through the bile ducts into the intestine (duodenum).

Clinical vignette

Blunt trauma to the right upper quadrant may produce liver lacerations – a cause of intra-abdominal bleeding.

Penetrating trauma in the right upper quadrant may cause damage to the liver, lung, diaphragm lining or colon.

Patients complaining of prolonged right upper quadrant abdominal pain who have tenderness to palpation may have distension (biliary colic) or inflammation of the gallbladder (acute cholecystitis).

Spleen

The function of the spleen, a soft-fleshy organ, is to help prevent systemic infection by activation of the immune system. It lies in the left upper quadrant protected by the 9th, 10th, and 11th ribs, running parallel to the 9th rib. The spleen is covered anteriorly by the stomach.

Much smaller than the liver, a normal spleen measures only 13-15 cm in length and 5-8 cm in width (about the size of a clenched fist). Unless the spleen is quite enlarged (at least twice its normal size) it is not palpable below the costal margin.
Clinical vignette
Like hepatic injuries, the spleen is often injured as a result of blunt or penetrating trauma to the left upper quadrant. In particular, because of the spleen's proximity to the ribs, patients with low left-sided rib fractures are at risk of splenic rupture. Interestingly, patients with infectious mononucleosis often develop splenomegaly and are at risk of splenic rupture with very minimal injury.

Pediatric patients are also at higher risk of splenic injury as the growth of the abdominal organs occurs sooner then the growth of the rib cage and therefore organs such as the liver and spleen are more exposed.

PANCREAS

The pancreas lies behind the peritoneum and is therefore a retroperitoneal structure. This gland has both exocrine function (production of digestive enzymes) as well as endocrine (production of insulin and glycogen). Anatomically, the gland lies at the level of L2 anterior to the great vessels and kidneys and behind the stomach. The head of the pancreas lies within the “C” shaped curve of the duodenum while the tail just barely touches the spleen (Figure 6).

DIGESTIVE TRACT

The alimentary canal consists of esophagus, stomach, small bowel (duodeum, jejunum, ileum) and large bowel (ascending colon, transverse colon, descending colon). The esophagus lies within the thoracic cavity and will not be discussed here.

STOMACH

The stomach is the first portion of the gastro-intestinal tract below the diaphragm. It is a large “reverse J-shaped” organ. The function of the stomach is to begin the digestion of foods and to propel food into the duodenum.

Within the abdominal cavity, the stomach is only partially covered by the left lobe of the liver. It lies anterior to the pancreas and just under the anterior abdominal wall.
SMALL BOWEL

The duodenum is firmly anchored in the abdominal cavity and like the pancreas is a retroperitoneal structure. The rest of the small bowel, however, is relatively mobile. The blood supply courses through a pedicle or stalk which is referred to as the mesentery. Foodstuff is propelled through the small bowel by peristalsis. The terminal ileum empties into the proximal portion of the large bowel at the cecum.

LARGE BOWEL

The large bowel begins at the cecum – the proximal portion of the ascending colon.

Stool is delivered from the small bowel in a liquid state. During propulsion through the colon absorption of water occurs such that solid feces is delivered into the descending (sigmoid) colon.

The vermiform appendix is a portion of large bowel which is a remnant, in that it has no true function.

The ascending colon passes up from the cecum along the right side of the abdominal cavity.

Just below the liver in the right upper quadrant, the bowel turns (at the hepatic flexure) becoming the transverse colon. This portion of the bowel passes anterior to the small bowel to again turn in the left upper quadrant (the splenic flexure).

The descending colon passes down along the left abdominal wall and in the left iliac fossa the sigmoid colon is formed. The large bowel ends at the rectum – which is really a pelvic organ.
Clinical vignette

Obstruction of the large or small bowel either from within (by hard stool, tumors) or without (adhesions or scarring, tumor, twisting upon itself) is a serious clinical problem. Patients usually present with accompanying abdominal distention, failure to pass gas or stool per rectum, and vomiting.

Another emergent abdominal concern is that of mesenteric vascular occlusion (ischemic bowel). Elderly patients with atrial fibrillation may embolize to the superior or inferior mesenteric artery causing abdominal pain and, if not diagnosed and managed urgently, can go on to have gangrene of a portion of the bowel with the risk of death.
THE GREAT VESSELS

AORTA

The abdominal aorta enters the abdomen at the level of T12 by passing through the diaphragm (Figure 8). Oxygenated blood from the left ventricle is carried to the renal arteries, the mesenteric arteries and the iliac arteries, thus supplying blood to the kidneys, bowel and lower extremities (as well as everything in between!).

Remember the aorta is a retroperitoneal structure running just anterior to the vertebral bodies behind the pancreas and stomach.

At the level of L4 (at about the umbilicus), the aorta divides into two common iliac arteries. In the pelvis, these iliac arteries are further subdivided into the internal and external iliac vessels.

INFERIOR VENA CAVA

The inferior vena cava (IVC) runs along side the aorta in the abdomen receiving blood from the iliac veins. The IVC is formed at about the level of L5 ascending to the right of the aorta anterior to the vertebral bodies. After passing behind the liver, the inferior vena cava empties blood into the right atrium (at the junction of the superior vena cava).
Clinical vignette
Rupture of an abdominal aortic aneurysm is life threatening. Most abdominal aneurysms (98%) occur below the renal arteries (Figure 8) and may be palpable as a pulsatile mass above the umbilicus or in the epigastrum. Patients with dissecting (blood between the tunica intima and media that separates the layers and the aneurysm expands) or expanding aneurysm present with severe abdominal pain and/or back pain.
URINARY TRACT

The kidneys lie upon the posterior abdominal wall protected by the ribs and are retroperitoneal. Each kidney is bean-shaped and is about 13 cm long by 6 cm wide. The upper pole of each kidney is at about the T11 or T12 level; the right kidney usually being slightly lower than the left.

From the hilum (point of entry/exit of vessels and ducts) of each kidney, a ureter is formed allowing urine to flow down into the urinary bladder. The ureters run retroperitoneally and empty into the bladder in the pelvis.

Clinical vignette

Trauma to the flank or abdomen can cause bruising (contusion) or laceration of the kidney. This is usually manifested by hematuria (i.e. gross or microscopic).

Patients with kidney stones and renal colic complain of severe waves of flank or abdominal pain as a result of visceral pain (obstruction of a hollow viscus).

Recall this pain is vaguely localized and usually associated with nausea and vomiting.
Marks

[2] 1. a) On the diagram below place the lines dividing the abdomen into quadrants.

[2]  b) Label the quadrants, using the common abbreviations for each.

[3] 2. a) On the diagrams below draw and label the epigastric region, flanks, and costovertebral angle.

[3]  b) Identify the probable surgical procedures performed with the scars labelled 1, 2, and 3.
3. Name the four muscles which comprise the anterior abdominal wall.

4. Differentiate between visceral and parietal peritoneum.

5. Name the organs which come into contact with the spleen.

6. The pancreas is situated anterior to _____________ vertebra.

7. The posterior landmark for bifurcation of the aorta is ___________________ vertebra.

8. Which of the following structures are retroperitoneal?

   IVC  o  liver  o  spleen  o  colon  o
   Kidneys  o  pancreas  o  stomach  o
   Ureters  o  duodenum  o  aorta  o
9. Where would you palpate expecting to find the pulsatile mass of an abdominal aneurysm?

10. Why does the pain of cholecystitis often increase on inspiration?

11. Fractures of the left-side 9th, 10th and 11th rib are often associated with what underlying injury?

12. Name the large pair of muscles which tend to hold in the abdominal contents anteriorly.

13. a) Which area of the abdominal contents is the most prone to shearing injury and why?

b) Assuming the above was an isolated injury, the most likely early clinical finding would be _________________ due to ________________.

34 TOTAL
1. 1 mark for each line.
½ mark for each label.

Quadrants:

2. a)

Costovertebral angle

b) 1. Cholecystectomy
2. Aorto-bifemoral bypass
3. Renal surgery
3. Rectus abdominis  
   Internal oblique  
   External oblique  
   Transversis abdominis

4. Parietal peritoneum lines the interior of the abdominal wall. Visceral peritoneum is continuous with the parietal peritoneum, but covers the surface of the organs.

5. Pancreas  
   Left kidney  
   Colon  
   Stomach

6. L2

7. L4 about the umbilical level.

8. Pancreas, duodenum, both kidneys, ureters, aorta, IVC

9. Superior to the umbilicus, in about the midline, or in the epigastric region

10. The gallbladder contacts the liver inferiorly. The liver contacts the diaphragm superiorly. During inspiration the diaphragm descends, increasing downward pressure, and often pain.

11. Rupture of the spleen.

12. Rectus abdominis.

13. a) The junction of the jejunum and the duodenum, due to the fixed position of the duodenum vs. the mobility of the jejunum.

   b) RUQ pain due to local peritoneal irritation from the spilled contents of the digestive system.
ADVANCED LIFE SUPPORT
PRE COURSE
THE ABDOMEN

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.

   Reading _________ hours
   Self assessment _________ hours
   Total time _________ hours

2. Were the objectives of the module clearly stated?

   [   ] yes     [   ] no
   If no, please comment.

3. Did you see any of the resource materials?

   [   ] yes     [   ] no
   If yes, which items
   ____________________________________________________________
   ____________________________________________________________
   ____________________________________________________________
   Were they helpful?
   ____________________________________________________________

4. Were the reference notes adequate?

   [   ] yes     [   ] no
   If no, please comment.

5. Were the reference notes easy to follow?
[ ] yes [ ] no
If no, please comment.

6. Were the examples provided satisfactory?

[ ] yes [ ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[ ] yes [ ] no
If yes, please specify.

1. Was the level of the module satisfactory for your program of study?

[ ] yes [ ] no
If no, please comment.

Base Hospital

9. General comments or suggested improvements.
OBJECTIVES: ENDOCRINE SYSTEM

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. define and give an example of:
   a) Endocrine gland
   b) Exocrine gland
   c) Mixed gland
   d) Double gland.

2. name and locate the principal endocrine glands.

3. list the major functions and secretions of each principal gland.

4. describe the action of each secretion listed in 3.

5. describe the mechanism for maintenance of normal blood glucose levels.

6. define gluconeogenesis and glycogenolysis.

7. describe the pathophysiology of diabetes, differentiating between:
   - Type I and Type II diabetes
   - Hypoglycemia and hyperglycemia
   - DKA and HNK.

8. differentiate clinically between hypoglycemic and hyperglycemic emergencies.

If you have studied this subject previously, you may test your ability using the self assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.
MAJOR ENDOCRINE GLANDS

The endocrine system, which exerts control over development, growth and metabolic activities, is comprised of a number of glands distributed throughout the body (Figure 1). The eight major glands of the system are:

1. Pituitary
2. Thyroid
3. Parathyroid
4. Thymus
5. Islets of Langerhans in the pancreas
6. Adrenal (suprarenal)
7. Testis (in the male)
8. Ovary (in the female).

FIGURE 1: MAJOR ENDOCRINE GLANDS
This module confines itself primarily to a discussion of the anatomy and physiology of the major endocrine glands. There are, however, other hormonal organs, tissue or cells in the body which also have a secretory function. These include the kidney, pineal gland, placenta and cells in the gastrointestinal tract.

Although dysfunction may occur in any gland, the pancreas is the one most commonly involved in pathologies seen by the Paramedic. Therefore, a more detailed discussion of the pancreas is presented in the second section of this module.

**TYPES OF GLANDS**

**ENDOCRINE GLAND**

Endocrine glands are ductless glands, although not all of the ductless glands in the body are endocrine glands, e.g. spleen. Their secretions are always hormones which pass directly into the bloodstream. Endocrine glands are made up of clusters of glandular epithelium.

Hormones are the secretions produced by the endocrine glands, and may be a form of protein, amine or steroid (lipid or fat). The function of the hormones is to maintain homeostasis (a constant internal body environment) by changing the physiological activities of cells. A hormone may stimulate changes in the cells of an organ or groups of organs, called target organs; a hormone may also directly affect the activity of all the cells in the body.

**MIXED GLAND**

A mixed gland is a gland which has both endocrine and exocrine functions. The exocrine component has ducts, through which its secretions pass. The pancreas is a gland with both endocrine (insulin production) and exocrine (production of digestive enzymes) functions.

**DOUBLE GLAND**

A double gland consists of two main parts in which the two components differ in their embryological derivation, nature of hormones released and the mechanism of neural control. The pituitary and adrenal glands are examples of double glands.
PITUITARY GLAND (HYPOPHYSIS CEREBRI)

The pituitary was formerly referred to as the master endocrine gland, because of its controlling influence on many of the other endocrine glands. This reddish-brown gland, the size of a garden pea, lies in the sella turcica (pituitary fossa, hypophyseal fossa), a fossa on the superior aspect of the body of the sphenoid bone in the base of the skull.

The pituitary is attached to the hypothalamus at the base of the brain by a stalk called the infundibulum.

The anterior lobe (adenohypophysis) of this double gland is responsible for the secretion of:

1. Growth hormone - somatotropic (STH)
2. Thyroid stimulating hormone - thyrotropic (TSH)
3. Adrenal cortex stimulating hormone - adrenocorticotropic (ACTH)
4. Sex gland stimulating hormones - gonadotropic (LH and FSE)
The posterior lobe secretes:

1. Oxytocin
2. Vasopressin (antidiuretic hormone - ADH).

Both of these cause the contraction of the smooth muscle in various organs.

**FIGURE 3: HORMONES RELEASED BY THE PITUITARY**

**HYPOTHALAMUS**

Most of the hormones secreted by the anterior pituitary stimulate the production of hormones by other endocrine glands (target glands).

Control of the rate of secretions in these target glands is accomplished by the hypothalamus through cells sensitive to changes in hormone levels in the blood. Since the hypothalamus
responds to an absence of pituitary hormones by producing substances (releasing factors) which enhance hormone secretion, this is referred to as a negative feedback mechanism.

**THYROID GLAND**

This gland is situated below the larynx at the upper part of the trachea. It consists of two lateral pear-shaped lobes united at the lower part by the isthmus, which lies in front of the second, third, and fourth tracheal rings.

**FIGURE 4: THYROID AND PARATHYROID GLAND**

![Diagram of Thyroid and Parathyroid Gland]

- Thyroid cartilage of the Larynx
- Parathyroid Glands (Behind the Thyroid)
- Trachea
Inadvertent penetration of this very vascular gland during needle or surgical cricothyroidotomy may result in severe hemorrhage.

The function of the thyroid gland is to:

1. Take up the inorganic iodine (derived from food) present in the blood.
2. Produce the hormone thyroxine.
3. Store thyroxine as thyroglobin.
4. Release thyroxine when stimulated to do so by the thyroid stimulating hormone (TSH) secreted by the adenohypophysis (pituitary gland).

**THYROID Glands**

- Thyroxine controls the metabolic rate of the body (the rate at which the body uses oxygen and metabolizes food products).
- Thyroxine is necessary for normal physical and mental development to occur.

**PARATHYROID Glands**

These, the smallest of the endocrine glands, may be variable in number and position. Normally there are four - two situated on the posterior surface of each of the lateral lobes of the thyroid gland. They lie beneath the capsule of the thyroid and may in fact be embedded in its tissue.

The parathyroid glands secrete a hormone, parathyroid hormone (parathormone) (PTH), which is concerned with calcium metabolism. The secretion of this hormone is regulated by blood calcium levels, and is not under the control of the adenohypophysis.

**THYMUS**

The thymus lies in the anterior and superior parts of the thorax. It is a relatively large structure at birth and continues to grow until puberty. Although the thymus decreases in size during adult life its physiological importance does not. It provides the mold, or model, for other organs, such as the liver and spleen, to produce T-lymphocytes (white blood cells concerned with the defense against infection and disease).

**ADRENAL (SUPRARENAL) Glands**

These two asymmetrical glands are found in close proximity to the upper poles of the kidneys. The right one is pyramidal and embraces the upper pole of the right kidney; while the left is crescentic and is located on the supramedial aspect of the left kidney above the hilum. Each measures about 5 cm long and 3 cm thick. Each gland is supplied by three relatively large arterial branches, indicating its relative importance to the body.
The adrenal glands are comprised of two functionally separate parts, the cortex and medulla.

The adrenal cortex secretes three main groups of hormones:

1. Gluocorticoids which are concerned with helping the body to cope with stressful situations.
2. Mineralcorticoids which have a similar function to that of the glucocorticoids, as well as affecting sodium and water balance in the body.
3. Androgens, the male sex hormones, which are only produced in small amounts.
The action of the hormones secreted by the adrenal medulla is to prepare the body for sudden action at a time of pain or fear (fight or flight situations). These hormones are:

- Adrenalin (epinephrine)
- Noradrenalin (norepinephrine).

**Clinical vignette**

Pheochromocytoma is a rare tumor of the adrenal gland that causes excess secretion of adrenalin. This may lead to hypoglycemia in non-diabetics. Administration of Glucagon may affect the adrenal gland/tumor resulting in even greater release of adrenalin and this may, paradoxically, worsen hypoglycemia.

**GONADS**

At the time of puberty, the adenohypophysis starts to secrete progressively more gonadotropic hormones. There are two in particular which affect both the ovaries and the testes. These are:

- Follicle stimulating hormone (FSH)
- Luteinizing hormone (LH) or interstitial cell stimulating hormone (ICSH) as it is called in the male.

**OVARIES**

The FSH and LH secreted by the adenohypophysis stimulate the development of all the female reproductive organs and initiate the menstrual cycle. The menstrual cycle is a series of rhythmic changes which occur throughout the childbearing years.

Each cycle lasts about 28 days and during this time three major hormone related events occur.

1. The FSH stimulates the maturation of the follicle containing an ovum which in turn produces the hormone estrogen.

2. The FSH and LH together bring about ovulation (the release of the ovum from the follicle and ovary).

3. The LH causes the lining of the uterus, the endometrium, to thicken in preparation for the implantation of a fertilized ovum. LH also causes the follicular cells left behind in the ovary to change their characteristics and colour to become the corpus luteum. The corpus luteum in turn produces large quantities of the hormone progesterone and small quantities of estrogen.

If fertilization and implantation do not take place, the corpus luteum will degenerate, the production of progesterone and estrogen will decrease and menstruation (bleeding) will occur.
Clinical vignette
The ovaries and corpus luteum are sites where the formation of cysts is relatively common. These cysts can cause torsion of the ovary resulting in pain, or they can bleed or rupture.

TESTES
The seminiferous tubules of the testes are stimulated by the secretion of FSH to produce spermatozoa and bring them to maturation.

The secretion of the ICSH stimulates the interstitial cells, the cells of Leydig, to produce the male hormone testosterone. The action of testosterone is to stimulate development and activity of male sex glands, as well as bringing about the changes that produce the secondary sexual characteristics (growth of body hair, and voice change). Testosterone levels are controlled by a negative feedback loop.

“FRINGES” OF THE ENDOCRINE SYSTEM
There are body structures whose tissue have a glandular appearance and have been suspected, despite the lack of convincing evidence, of having endocrine properties.

PINEAL GLAND
Located in the forebrain, the pineal gland was the subject of speculation for years. In fact, as recently as 1985, when this pre-course manual was first written, understanding of the pineal gland’s function was very poorly understood. Today, a little bit more is understood about the Pineal gland, however, Physiologists and Endocrinologists still have a long way to go.

The pineal gland produces Melatonin which plays a role in the body’s circadian rhythm or “biological time clock”. In the dark, the pineal gland is stimulated to produce and release melatonin and in the daylight, melatonin production is reduced. It’s recently been suggested that treatment with melatonin may prevent and/or reduce jetlag, reduce the risk of developing certain tumors and may slow the aging process. The Pineal Gland also regulates our patterns of eating (hunger), sleeping, reproduction (female reproductive cycle), and behaviour.

Seasonal affective disorder (SAD) has been linked to the Pineal Gland, as there is a reduction in Melatonin during the winter months when daylight is shortened. In some occult religions, it is sometimes referred to as the "third eye" and is believed to be a dormant organ that can be awakened to give mystical powers.
Clinical vignette
The pineal gland has a midline position within the brain and in some adults it is calcified, making it a visible landmark on skull x-rays. If it has shifted out of the midline, it may indicate the presence of a pathological process such as an expanding lesion.

INTESTINAL TRACT GLANDS

Intestinal tract glands that secrete gastrin, secretin and cholecystokinin are endocrine glands because their secretions pass directly into the circulatory system. The significance of these hormones are not discussed in this precourse material.

THE KIDNEY

The peptide angiotensin, found in the bloodstream, is effective in causing high blood pressure and its presence is primarily the result of kidney activity. This "hormone" is not produced in the kidney, but an enzyme from this organ, renin, is responsible for initiating chemical changes in a protein in the blood stream which cause eventual production of the "hormone". Also, evidence indicates that red blood cell formation is influenced by a hormonal agent, erythropoietin and again the kidney is involved.

In this section, we have primarily examined the anatomy of the organs other than the pancreas that have been conveniently classified as endocrine glands. However, we should remain aware there are certain organs, tissues or cells, whose primary role is not endocrine, but may emit secretions to the circulation and assume an endocrine function.
MARKS

[2] 1. Give one example of a:
   
a) mixed gland

[2] 2. What is a mixed gland?

[2] 3. What is a double gland?

[2] 4. The (a) _______ gland is attached to the base of the brain and its body rests in the b) ______ of the sphenoid bone.
5. The secretions of endocrine glands pass directly into the bloodstream. True or False.

6. Name two different endocrine glands that are located in the abdomen.

7. What is the anatomical location of the thyroid gland?

8. Where are the parathyroid glands found?

9. What chemical substances are released by the adrenal medulla?

10. Which of the following hormones are produced by the adenohypophysis?
   a) Oxytocin
   b) Estrogen
   c) Growth hormone
   d) Thyroxine
   e) Adrenalin
11. Complete the following chart.

<table>
<thead>
<tr>
<th>GLAND</th>
<th>HORMONE (S) PRODUCED</th>
<th>FUNCTION (S) OF HORMONE (S) PRODUCED</th>
</tr>
</thead>
<tbody>
<tr>
<td>adrenal cortex</td>
<td>thryoxine</td>
<td></td>
</tr>
<tr>
<td></td>
<td>parathromone</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ADH</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1. glucocorticoids</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2. mineralocorticoids</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3.</td>
<td>similar to glucocorticoids</td>
</tr>
<tr>
<td></td>
<td>sex hormones</td>
<td></td>
</tr>
<tr>
<td>adrenal medulla</td>
<td>1. epinephrine</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2. norepinephrine</td>
<td></td>
</tr>
</tbody>
</table>

12. The kidneys secrete hormones that control (a) _________________ and regulate (b) _________________ production.

28 TOTAL
SELF-ASSESSMENT ANSWERS: MAJOR ENDOCRINE GLANDS

1. a) Pancreas  
b) Pituitary gland (hypophysis)

2. A gland with both endocrine and exocrine function.

3. A gland consisting of at least two major components which differ in their hormone production.

4. (a) Pituitary, (b) Sella Turcica

5. True.

6. Adrenals (suprarenals) and Pancreas

7. Below the larynx, anterior to the upper end of the trachea.

8. One pair on the posterior aspect of each of the lobes of the thyroid gland.

9. Adrenalin (epinephrine), and Noradrenalin (norepinephrine)

10. c) Growth Hormone
11. | GLAND                  | HORMONE(S) PRODUCED | FUNCTION OF HORMONE(S) |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>thyroid</td>
<td>thyroxine</td>
<td>control of metabolic rate of tissues</td>
</tr>
<tr>
<td>parathyroids</td>
<td>parathormone</td>
<td>calcium metabolism</td>
</tr>
<tr>
<td>pituitary-posterior lobe</td>
<td>ADH</td>
<td>conserves water</td>
</tr>
<tr>
<td>adrenal cortex</td>
<td>1. glucocorticoids</td>
<td>stress response</td>
</tr>
<tr>
<td></td>
<td>2. mineralocorticoids</td>
<td>similar to glucocorticoids</td>
</tr>
<tr>
<td></td>
<td>3. androgens</td>
<td>sex hormones</td>
</tr>
<tr>
<td>adrenal medulla</td>
<td>1. epinephrine</td>
<td>“fight or flight response”</td>
</tr>
<tr>
<td></td>
<td>2. norepinephrine</td>
<td></td>
</tr>
</tbody>
</table>

12. (a) Blood pressure; (b) Red blood cell
THE PANCREAS

The pancreas consists of a head, body and tail. It lies behind the stomach, lying transversely on the posterior abdominal wall. Its head lies in the loop of the duodenum and its tail reaches the hilum (point of entry or exit of ducts, nerves, vessels) of the spleen.

PANCREATIC ISLETS

Although the greater part of the glandular tissue of the pancreas is devoted to the production of digestive enzymes which pass through ducts to the duodenum (exocrine function), cells of another type of tissue can be seen distributed through the gland as isolated islands. These irregular clumps of cells, called the Islets of Langerhans comprise approximately 2% of the pancreas and constitute the endocrine secretory units of the pancreas. An extensive capillary network is associated with each islet to transport its secretions from the pancreas.

Since the pancreas has both endocrine and exocrine functions, it is therefore a mixed gland. Its endocrine secretions are the hormones insulin and glucagon.
The islet cells may be classified as alpha or beta; the beta cells being most abundant. Insulin is produced by the beta cells in the Islets of Langerhans to lower blood sugar levels. The release of insulin by the pancreas occurs when blood sugar levels are high.

Glucagon, produced by the alpha cells in the Islets of Langerhans, has the opposite effect to insulin. It stimulates the conversion of glycogen (the storage form of glucose) to glucose which is released into the blood, thus raising the level of blood sugar.

There is little nervous influence on insulin hormonal secretion, which appears to be mainly controlled by the level of glucose passing through the islets.

The action of insulin is to facilitate the transport of glucose across the cell membrane, making it available to the cell for oxidation in the Kreb's Cycle. Insulin also facilitates storage of glucose as glycogen in depots primarily in the liver, muscle and kidney.

Glucose is the primary cell fuel. Certain cells can only use glucose. The most important of these are the cells of the CNS. Ninety percent of brain cell metabolism utilizes glucose. In fact, glucose is so important to normal brain cell function that it does not require insulin to facilitate its passage across the cell membrane. To some degree this compensates for lack of a backup stockpile of stored glucose (glycogen) here. The brain is therefore dependent on a constant supply of glucose to maintain its cellular activities. Let us examine how this is achieved.

REGULATION OF BLOOD GLUCOSE LEVELS

About 50% of western world diets consist of carbohydrates which are readily absorbed from the gut after breakdown by digestive enzymes. The bulk of digested carbohydrate is in the form of various sugars, the most plentiful being glucose which is ready for transport by the bloodstream and immediate use by the cell.

Since insulin is the key for the entry of glucose into many cells (brain and liver excluded) it must be produced and transported to the cells, along with the glucose.

Circulating levels of glucose are the trigger for the beta cells in the Islets of Langerhans to release insulin. Promoting the storage of glucose as glycogen (primarily in the liver) is another important role of insulin. In doing so, it inhibits the conversion of glycogen to glucose at this time. However, most of us have long periods, i.e. overnight, where we do not have a food intake.

As glucose from a meal is either used up or stored, insulin levels fall. The hormone glucagon (from the alpha cells of the islets of Langerhans) is then released along with epinephrine from the adrenal medulla. This causes mobilization of glycogen stores, again primarily from the liver. Conversion of glycogen to glucose (glycogenolysis) raises blood sugar levels, ensuring a constant supply to the cells.

However, these stores are limited. In fact, the liver barely stores enough glycogen to meet the metabolic needs of brain tissue for a 24 hour period. As glycogen stores are consumed proteins and fats from other sources (mostly muscle tissue and adipose) are freed for conversion to
glucose by the liver (gluconeogenesis). This process is mediated by glucocorticoids from the adrenal cortex. In times of prolonged starvation the kidneys become involved in gluconeogenesis from fats. This process is facilitated by insulin and enhanced by epinephrine and growth hormone (from the pituitary).

**Clinical vignette**

Administration of Glucagon will raise the blood glucose level in a patient who has sufficient glycogen stores. A second dose of Glucagon may be indicated for patients whose blood glucose level remains ≤ 4 mmol/L after the first dose, however, if the glycogen stores are inadequate, there will be no effect on BGL.

It should now be apparent that our homeostatic systems are geared to provision of a constant supply of glucose to the CNS, sparing protein and fat for other duties. As with any homeostatic regulatory mechanism, there exists a potential for problems at any one of the stages.

By far the most common endocrine emergency encountered by prehospital personnel will involve the diabetic patient. What follows is a discussion of the more important features of diabetes mellitus, and the implications for you as a care provider. Before proceeding, let's clarify one point ... diabetes insipidus is a different pathology involving primarily the pituitary gland. It will not be discussed in this unit.

**DIABETES MELLITUS (DM)**

Diabetes is a chronic disorder affecting the way the body uses and stores the foods eaten and is caused by a deficiency or the ineffectiveness of insulin secreted by the pancreas. Over 2 million Canadians live from diabetes (http://www.diabetes.ca/Section_about/index.asp).

Insulin can be unavailable for many reasons:

1. Destruction of the beta cells in the Islets of Langerhans which is believed to involve a genetically transmitted autoimmune problem.

   A viral infection either directly destroys beta cells or the resulting autoimmune reaction is responsible.

   In either case - no beta cells, means no insulin.

   These patients are dependent upon insulin for life, and without it will develop ketosis or ketoacidosis and die. More about this later.

   Since most, but not all, of these patients are young at onset, this type of diabetes was formerly referred to as "juvenile diabetes". More appropriately, it is now called insulin dependent diabetes mellitus (IDDM).
2. Resistance to normal or elevated serum insulin levels. Predisposition to this process is also believed to be genetically transmitted. The defect is believed to be related to a decrease in the number of insulin receptors on the cell membrane, which reduces the ability of insulin to act at this level.

There is a correlation between overeating and the onset of this problem. These patients tend to be over 40 and overweight at onset. This accounts for the former term of "age onset diabetes". Currently, it is referred to as non-insulin dependent diabetes (NIDDM) or Type II.

Investigators believe that between 35 and 40 etiologies of this type of disease may be possible.

Management of NIDDM may consist of weight reduction, with or without the use of oral hypoglycemic agents. Insulin may be added temporarily to prevent hyperglycemia, but without it, these individuals can still survive.

3. Other conditions such as surgical removal of the pancreas, pancreatitis, pancreatic injuries and rare endocrine disorders may lead to diabetes.

The goals of management of the diabetic patient are to maintain as nearly normal levels of blood glucose as possible using current therapies. In the non-diabetic person this is achieved by homeostatic feedback mechanisms which constantly monitor and adjust blood glucose levels. Current therapy involving extensive patient education in the regulation of diet and exercise, the monitoring of blood glucose levels and administration of medication sometimes by continuous infusion (insulin pump) is still very inexact, compared with natural mechanisms.

Unfortunately, there is a relatively narrow range of "normal" blood glucose values tolerated by human tissues. Just as glucose deficits within the cell are problematic, glucose excesses are, over time, toxic.

Diabetics may develop:

- Blindness
- Kidney failure
- Heart disease and stroke: diabetes is the underlying cause of 50% of all heart attacks and strokes
- Peripheral neuropathies
- Peripheral vascular disease.
HYPOGLYCEMIC EMERGENCIES

Decreased levels of blood glucose cause the patient to be symptomatic by two mechanisms:

1. SNS stimulation (beta adrenergic) which results in anxiety, trembling, tachycardia, palpitations, diaphoresis, faintness, dilated pupils, pallor.

2. CNS depression (primarily at the cortical level) resulting in weakness, headache, blurred vision, impaired judgement, confusion, lack of co-ordination, seizures and coma.

The respiratory pattern is usually normal in these patients.

Most often hypoglycemia is seen in the IDDMs who has missed a meal, over-exercised, altered insulin regime, or has an infection. Other causes can include oral hypoglycemic agents, and ethanol abuse over time (primarily due to inadequate glycogen stores and defective gluconeogenesis).

Prolonged or recurrent hypoglycemia can result in brain damage (often in the areas affecting personality) and/or death. The damage is irreversible.

The goals of therapy are directed at rapidly restoring blood glucose levels to the normal range and replacing depleted glycogen stores.

HYPERGLYCEMIC EMERGENCIES

Hyperglycemic states involve increased levels of blood glucose which may be present in some diabetics. However, we will focus on those patients in whom hyperglycemia presents as a life-threatening emergency. These conditions are:

- diabetic ketoacidosis (DKA)
- hyperglycemic, hyperosmolar, nonketotic coma (HHNK).

DIABETIC KETOACIDOSIS (DKA)

Increased levels of blood glucose and ketones due to a relative lack of insulin are usually, but not always, seen in IDDM. Symptoms can be attributed to an excess of glucagon, glucocorticoids, growth hormone and catecholamines usually due to stressful events, such as illness or emotional stress.

These hormones, you'll recall, are the ones associated with the body's homeostatic mechanisms in response to decreased blood glucose at the cellular level. They are the hormones of glycogenolysis, and gluconeogenesis in response to cellular starvation.

Schematically, the sequence of events is represented in Figure 7.
It is apparent that a number of vicious cycles operate here:

1. In the absence of insulin, and the cell's inability to utilize glucose, the hyperglycemia will tend to get worse due to the glucose production from protein and fat breakdown.

2. As the serum glucose becomes very high, the kidney is unable to reabsorb the high concentration of glucose in the renal tubules. The glucose trapped in the renal tubules (in urine) results in a large volume of water being extracted (polyuria or osmotic diuresis). This osmotic diuresis also removes electrolytes such as Na\(^+\) and K\(^+\).

3. As fats are mobilized for glucose production, the glycerol portion is metabolized to glucose (causing ↑ hyperglycemia). The breakdown of the free fatty acid portion results in ketonuria, again contributing to the diuresis and electrolyte depletion.
Ketones in blood are acidic. Compensatory buffering systems (see acid/base unit) become activated and depleted. The respiratory system attempts to compensate by blowing off ketone byproducts (acetone breath) and CO₂ resulting in the hyperventilation (Kussmaul respirations) characteristic of the condition. This may be the presentation of an undiagnosed Type I diabetic.

HYPERGLYCEMIC, HYPEROSMOLAR, NONKETOTIC COMA (HHNK)

Isolated hyperglycemia without ketoacidosis is usually seen in NIDDM. NIDDMs usually have enough endogenous insulin that fats are not mobilized. They do however mobilize proteins in response to precipitating events with the resulting hyperglycemia, polyuria, electrolyte depletion and dehydration without the acidosis component. Therefore a similar sequence of events occurs as illustrated in Figure 7, but without the breakdown of fats.

The history and findings which differentiate diabetic ketoacidosis (DKA) from hyperglycemic, hyperosmolar, nonketotic coma (HHNK) are summarized in Table I.

<table>
<thead>
<tr>
<th></th>
<th>DKA</th>
<th>HHNK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetic History</td>
<td>IDDM (TYPE I)</td>
<td>NIDDM (TYPE II)</td>
</tr>
<tr>
<td>May be undiagnosed diabetic</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Respiration</td>
<td>Kussmaul (acetone odour)</td>
<td>Normal</td>
</tr>
<tr>
<td>Heart Rate (HR)</td>
<td>Elevated</td>
<td>Elevated</td>
</tr>
<tr>
<td>Blood Pressure (BP)</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Level of Consciousness (LOC)</td>
<td>Depressed</td>
<td>Depressed</td>
</tr>
<tr>
<td>Abdominal Pain</td>
<td>Common</td>
<td>No</td>
</tr>
</tbody>
</table>

Symptoms of hyperglycemia have a slower and less dramatic onset usually over several days, than do those of hypoglycemia. They may in fact go unrecognized by the patient and relatives until they present a life-threatening emergency.

The mortality rate from hyperglycemic emergencies remains significant at 5-10%. Death can result from organ failure subsequent to hypovolemic shock, arrhythmias subsequent to cellular starvation and electrolyte depletion, or embolic events associated with underlying disease and/or increased viscosity (thickness) of plasma.

Therapy is directed at rehydration, restoration of electrolytes and pH levels, reduction of blood glucose levels to within normal limits and identification of underlying precipitating event.
MARKS


__________________________________________________________________________
__________________________________________________________________________
__________________________________________________________________________

[2] 2. Define:

Gluconeogenesis

__________________________________________________________________________
__________________________________________________________________________

Glycogenolysis

__________________________________________________________________________
__________________________________________________________________________

[4] 3. The pancreas produces two hormones; these are:
   (a) ________ which (raises/lowers) ________ blood glucose levels and (b) which (raises/lowers) ________ blood glucose levels.

__________________________________________________________________________
__________________________________________________________________________

[1] 4. Other factors which lower blood glucose are ________ and ________.

__________________________________________________________________________
__________________________________________________________________________
5. Secretion of insulin is mainly controlled by _______.

6. Compare Type I and Type II diabetes by completing this chart.

<table>
<thead>
<tr>
<th></th>
<th>TYPE I</th>
<th>TYPE II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common abbreviation</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>Usual age of onset</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>Controlled by</td>
<td>1. _______</td>
<td>1. _______</td>
</tr>
<tr>
<td></td>
<td>2. _______</td>
<td>2. _______</td>
</tr>
<tr>
<td></td>
<td>3. _______</td>
<td>3. _______</td>
</tr>
<tr>
<td></td>
<td>4. _______</td>
<td></td>
</tr>
<tr>
<td>Ketoacidosis prone?</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>Non-Ketotic Hyperosmolar coma prone?</td>
<td>_______</td>
<td>_______</td>
</tr>
</tbody>
</table>

7. The mechanism of action of insulin is:

8. In the absence of insulin brain cells (are/are not) _______ able to utilize glucose.
9. Diabetes is a result of _______.

10. Many diabetic patients have major illnesses secondary to diabetes. Name two of these illnesses that you are likely to encounter in the field.

11. In response to cellular starvation the body breaks down ____________, ____________, and ____________.

12. The cause of ketoacidosis is related to the breakdown of ____________.

13. The conditions resulting from ketoacidosis are primarily (a) ____________ acidosis, ____________ and (c) ____________ depletion. The (d) ____________ pain may serve to mislead the examiner.

14. Differentiate between the important features of hypoglycemia and pathologic hyperglycemia below.

<table>
<thead>
<tr>
<th>HYPOGLYCEMIA</th>
<th>HYPERGLYCEMIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of symptoms</td>
<td>____________</td>
</tr>
<tr>
<td>Skin condition</td>
<td>____________</td>
</tr>
<tr>
<td>Respirations</td>
<td>____________</td>
</tr>
<tr>
<td>Onset of coma</td>
<td>____________</td>
</tr>
</tbody>
</table>

30 TOTAL
SELF-ASSESSMENT ANSWERS: THE PANCREAS

1. Behind the stomach, resting on a loop of duodenum on the right, touching the hilum of the spleen on the left.

2. Gluconeogenesis - production of glucose from the breakdown of proteins and fats in the liver.
   Glycogenolysis - production of glucose from the breakdown of glycogen in the liver.

3. a) Insulin, lowers
   b) Glucagon, raises

4. Exercise, starvation

5. Blood glucose levels (passing through the Islets of Langerhans).

6. | TYPE I          | TYPE II         |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Common abbreviation</td>
<td>IDDM</td>
</tr>
<tr>
<td>Usual age of onset</td>
<td>younger</td>
</tr>
<tr>
<td>Controlled by</td>
<td>1. insulin</td>
</tr>
<tr>
<td></td>
<td>2. diet</td>
</tr>
<tr>
<td></td>
<td>3. exercise</td>
</tr>
<tr>
<td></td>
<td>4. +/- insulin</td>
</tr>
<tr>
<td>Ketoacidosis prone?</td>
<td>Yes</td>
</tr>
<tr>
<td>Non-Ketotic Hyperosmolar coma prone?</td>
<td>Not usually</td>
</tr>
</tbody>
</table>

7. Facilitating glucose transport across the cell membrane.

8. Are
9. Lack of insulin (either relative or absolute)

10. (Arteriosclerotic) heart disease and stroke Renal disease

11. Glycogen, protein, fats

12. Fat

13. a) Metabolic
    b) Dehydration (osmotic diuresis)
    c) Electrolyte
    d) Abdominal

14. HYPOGLYCEMIA HYPERGLYCEMIA

   Onset of symptoms Rapid over Gradual over
   minutes to hours hours to days
   Skin condition Pale, wet Hot, dry
   Respirations Normal Rapid, deep
   (Kussmaul) in DKA Normal in HHNK
   Onset of coma Rapid Much longer,
ADVANCED LIFE SUPPORT
PRE COURSE
THE NERVOUS SYSTEM

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.

   Reading   ________ hours
   Self assessment   ________ hours
   Total time   ________ hours

2. Were the objectives of the module clearly stated?

   [ ] yes   [ ] no
   If no, please comment.

3. Did you see any of the resource materials?

   [ ] yes   [ ] no
   If yes, which items
   _______________________________________________________________
   _______________________________________________________________
   _______________________________________________________________
   Were they helpful?
   _______________________________________________________________

4. Were the reference notes adequate?

   [ ] yes   [ ] no
   If no, please comment.
5. Were the reference notes easy to follow?

[ ] yes [ ] no
If no, please comment.

6. Were the examples provided satisfactory?

[ ] yes [ ] no
If no, please comment.

7. Were any of the self-assessment questions poorly worded?

[ ] yes [ ] no
If yes, please specify.

1. Was the level of the module satisfactory for your program of study?

[ ] yes [ ] no
If no, please comment.

Base Hospital

9. General comments or suggested improvements.
OBJECTIVES: MEDICAL MATH

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self-assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. state the six base units of the SI system and name the physical quantity that they measure.

2. differentiate between a base unit and a derived unit of the SI system.

3. with specific reference to the SI unit of volume, state the “special” derived unit that is recommended for use in the health technologies.

4. with specific reference to the SI unit for temperature:
   a) name the base unit
   b) state the derived unit of measurement recommended for use in the health technologies
   * c) given a temperature in one of these units convert to the other unit.

5. name and define the prefixes used in the SI System to denote multiples and submultiples of any unit.

*6. convert SI units of different magnitudes.

7. write all symbols and numbers used in the SI system according to the basic rules for writing symbols and numbers.

*8. interconvert between SI, metric and imperial systems of measurement for the following physical quantities:
   a) temperature
   b) weight
   * c) length

9. given a series of lines, estimate the length within one centimetre without aid of a measure or calculator.

* Calculations for these must be done without the aid of a calculator.
10. Given mathematical problems similar to those encountered in A.L.S. field situations, correctly perform the appropriate calculations without aid of a calculator.

If you have studied this subject previously, you may test your ability using the self-assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.
INTRODUCTION

Unit of measurement have evolved from many sources. Many of the systems found their origins in local customs and usage. Most lacked a rational structure and had poorly correlated units. Interconversion between the systems was difficult if not impossible.

Of all the systems that have evolved, two (imperial system and metric system) have had common usage in Canada.

THE IMPERIAL SYSTEM

This system, used for everyday measurements, uses units such as the yard, pound and quart. Not only is there poor correlation between the units but also two different sets of unit have evolved. The first being developed by countries of British origin and the second by the United States.

THE METRIC SYSTEM

This system, developed in France over two hundred years ago, uses such units as metre, litre and gram. Although widely accepted by most European countries and by the scientific community, it too was often confusing to work with. Several versions of the metric system have evolved and many of the measurements made with this system incorporated both imperial and metric units.

In 1960, the international system of Units was established. Basically it is a modernized metric system designed to replace all former systems of measurement, including former versions of the metric system. It is commonly referred to as the SI system of measurement, i.e. Le Systeme International.

Canada officially adopted the SI system of measurement in 1970.

On April 1, 1983, all members of the Hospital Council of Metropolitan Toronto converted to the SI system. Since this changeover has taken place, and since many people still use the imperial system and/or the metric system for everyday measurements, this module will deal with the SI system for everyday measurements, This module will deal with the SI system, as related to the health sciences, and where applicable, the conversion of the SI system to imperial and/or metric units of measurement.
METRIC REVIEW

It is important that the student have a good command of the metric system in order to fully understand pharmacology, fluid administration, and body weight.

The metric system, which employs the decimal scale, is composed of units measuring length, volume and weight.

Because the metrics system employs the decimal scale, its numerical scale is based on 10. The prefixes used in the metric system indicate which unit of 10 applies to the measure in use. Three of the prefixes are used to indicated multiples of 10

- Deca refers to units of 10
- Hecto refers to units of 100
- Kilo refers to units of 1000

Of these, the one most often used is Kilo.

Three of the prefixes are used to indicate fractional units.

A litre is a liquid measure and a centimetre is a linear measure. However, in the metric system, the two are directly related, since 1 litre occupies the space of 1000 cubic centimetres.
EQUIVALENTS

Linear Measure

1 Metre (m) = 10 Decimetres (dm)
1 Metre = 100 Centimetres (cm)
1 Metre = 1000 Millimetres (mm)
1 Metre = 1 000 000 Micrometres (µm)
1 Metre = 1 000 000 000 Nanometres (nm)
10 Metre = 1 Decametre (dam)
100 Metre = 1 Hectometre (hm)
1000 Metre = 1 Kilometre (km) (5/8 Mile)

Volume

1 Litre (L) = 10 Decilitres (dL)
1 Litre = 100 Centilitres (cL)
1 Litre = 1000 Millilitres (mL)
10 Litre = 1 Decalitre (daL)
100 Litre = 1 Hectolitre (hL)
1000 Litre = 1 Kilolitre (kL)

Weight

1 Gram (G) = 10 Decigrams (dg)
1 Gram = 100 Centigrams (cg)
1 Gram = 1000 Milligrams (mg)
1 Gram = 1 000 000 Micrograms (µg)
10 Gram = 1 Decagram (dag)
100 Gram = 1 Hectogram (hg)
1000 Gram = 1 Kilogram (kg) (2.2 lb)
.000001 Gram or .001 Milligram = 1 Microgram (µg)

The most useful of these measures in prehospital care are highlighted in this module.
TYPES OF UNITS

There are three types of units in the SI system:

1. Base units
2. Derived units
3. Supplementary units.

BASE UNITS

The six base units in the SI system most relevant to prehospital care serve as the reference units for the derived units of measurement. By using appropriate combinations of the base units, any physical quantity can be expressed.

The base units (Table 1) can be independently define or calculate.

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>BASE UNITS</th>
</tr>
</thead>
<tbody>
<tr>
<td>PHYSICAL QUANTITY</td>
<td>BASE UNIT</td>
</tr>
<tr>
<td>length</td>
<td>metre</td>
</tr>
<tr>
<td>mass</td>
<td>kilogram</td>
</tr>
<tr>
<td>time</td>
<td>second</td>
</tr>
<tr>
<td>amount of substance</td>
<td>mole</td>
</tr>
<tr>
<td>thermodynamic temperature</td>
<td>Kelvin</td>
</tr>
<tr>
<td>electric current</td>
<td>ampere</td>
</tr>
</tbody>
</table>

DERIVED UNITS

Derived units cannot be independently defined or calculated without reference to base units, supplementary units and/or other derived units.

EXAMPLE 1:

A newton is the derived unit for the physical quantity force.

A newton (N) is defined as the force which, when applied to a mass of one kilogram (kg) gives it an acceleration of one metre (m) per square second (s²).

\[1 \text{ N} = 1 \text{ kg.m/s}^2\]

In this example, the derived unit newton (N) is defined in terms of the base units kilogram, metre and second.
A list of derived units can be found in Appendix A. It is not intended that you memorize these units at this time, but rather, that this list serve as a reference source for future studies.

There are two derived units of measurement, volume and temperature, which are routinely used, or referred to, by all health technologies. They will be discussed here in detail because each is "special" in the SI system.

**Volume**

The SI derived unit for volume is the cubic metre (m³). However, the litre (L) is also accepted as a "special" name for a derived unit of volume. In fact, because of its convenience, the litre (L) is recommended as the unit for volume of fluid. Concentrations of all fluids are expressed per litre not per cubic metre.

The abbreviation cc (cubic centimetre) formerly used in medicine to express fluid volume is now replaced by the millilitre (mL), e.g. if we have a 10 cubic centimetre syringe, we can fit exactly 10 millilitres of fluid in it.

**TEMPERATURE**

Reference to the base units of the SI system will show that the unit for measuring temperature is the Kelvin (K). However, since the Kelvin temperature scale has limited application in medicine, a derived unit for measurement with wider applications, was approved. This accepted derived unit is the degree Celsius (°C). However, some household thermometers still record in Fahrenheit degrees.

The Celsius and Fahrenheit temperature scales are related as shown in Table 2.
### TABLE 2
CELSIUS/FARENHEIT TEMPERATURE SCALES

<table>
<thead>
<tr>
<th>CELSIUS °C</th>
<th>FAHRENHEIT °F</th>
</tr>
</thead>
<tbody>
<tr>
<td>100°</td>
<td>212</td>
</tr>
<tr>
<td>41</td>
<td>105.8</td>
</tr>
<tr>
<td>40</td>
<td>104</td>
</tr>
<tr>
<td>39</td>
<td>102.2</td>
</tr>
<tr>
<td>38</td>
<td>100.4</td>
</tr>
<tr>
<td>37</td>
<td>98.6</td>
</tr>
<tr>
<td>0</td>
<td>32</td>
</tr>
</tbody>
</table>

### SUPPLEMENTARY UNITS

Certain units of the SI have not yet been classified as either base units or derived units. They are referred to as supplementary units and may be regarded as either base or derived. Since there are no supplementary units used in general medical practice, they will not be discussed in this module.

### SI UNITS USED IN HEALTH CARE

Appendix B has a list of more common SI units applicable to the health technologies. It is meant as a reference source for future studies.
PREFIXES OF THE SI SYSTEM

Multiples and submultiples of SI units are denoted using a system of prefixes (Table 3).

<table>
<thead>
<tr>
<th>PREFIX</th>
<th>SYMBOL</th>
<th>FACTOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mega</td>
<td>M</td>
<td>1,000,000</td>
</tr>
<tr>
<td>*kilo</td>
<td>k</td>
<td>1,000</td>
</tr>
<tr>
<td>Hecto</td>
<td>h</td>
<td>100</td>
</tr>
<tr>
<td>Deca</td>
<td>da</td>
<td>10</td>
</tr>
<tr>
<td>*deci</td>
<td>d</td>
<td>0.1</td>
</tr>
<tr>
<td>*centi</td>
<td>c</td>
<td>0.01</td>
</tr>
<tr>
<td>*milli</td>
<td>m</td>
<td>0.001</td>
</tr>
<tr>
<td>*micro</td>
<td>u</td>
<td>0.000001</td>
</tr>
<tr>
<td>*nano</td>
<td>n</td>
<td>0.000000001</td>
</tr>
</tbody>
</table>

*Prefixes commonly used in the health technologies.

Examples of Using Prefixes

EXAMPLE 5:

Kilo means $1 \times 10^3$

Therefore,  
1 kilogram (kg) = $1 \times 10^3$ g = 1000 g
1 kilometer (km) = $1 \times 10^3$ m = 1000 m

EXAMPLE 6:

Milli means $1 \times 10^{-3}$

Therefore,  
1 millilitre (mL) = $1 \times 10^{-3}$ L = 0.001 L
1 milligram (mg) = $1 \times 10^{-3}$ g = 0.001 g

EXAMPLE 7:

Nano means $1 \times 10^{-9}$

Therefore,  
1 nanometre (nm) = $1 \times 10^{-9}$ m = 0.000000001 L
RULES FOR WRITING SYMBOLS AND NUMBERS IN SI

One of the main advantages of SI is that there is a unique symbol for each unit. Because these are international symbols and not abbreviations, they do not change for different languages. It is easier and in most cases faster to use the SI symbol than writing the name of the unit in full.

Examples of Symbols

\[
\begin{align*}
m & = \text{metre} \\
g & = \text{gram} \\
\mu g & = \text{microgram} \\
^\circ \text{C} & = \text{degree Celsius} \\
s & = \text{second}
\end{align*}
\]

RULES FOR WRITING NUMBERS

1. Use decimals, not fractions: 0.25 g (not ¼ g).

2. Use a zero before the decimal marker if the numerical values are less than one: 0.45 g (not .45 g). \textbf{This is particularly important when documenting decimals associated with drug usage.}

3. As a decimal marker, both the point and the comma are widely used in the world today. \textit{*Metric Commission Canada’s policy is to use the comma in French-language documents and the point in English-language. This practice is being followed by the Department of Justice in printing legislation.}

RULES FOR WRITING SYMBOLS

1. Symbols are written in lower case, except when the unit name is derived from a proper name: \( m \) for metre; \( s \) for second; but \( N \) for newton.

Exception: \( \text{The symbol for litre (L) is always written as an upper case letter.} \)

2. When the names of the units are written out in full, lower case letters are always used even if the unit is derived from a proper name: newton not Newton.

Exception: \( \text{Celsius is the only unit that when written in full, is capitalized.} \)

3. Prefix symbols are printed in upright type without spacing between the prefix symbol and the unit symbol: \( \text{kg for kilogram; km for kilometre.} \)

4. Symbols are never pluralized: 1 g, 45 g (not 45 gs).

\textit{* Source: How to Write SI (fifth edition) Metric Commission Canada}
5. Names and symbols should not be mixed. N\(^{\circ}\)m or newton metre, but not N metre or newton m.

6. Never use a period after a symbol, except when the symbol occurs at the end of a sentence.

7. Symbols should be used in conjunction with numerals instead of writing out the unit names; when no numerals are involved, unit names should be written out:

   Weigh out 16 g of chemical (not 16 grams). Calculate the weight of chemical in grams (not g).

8. The product of two or more units in symbolic form is indicated by a dot. The dot must be positioned above the line to distinguish it from a decimal marker dot on the line: kg\(^{\circ}\)m\(^{\circ}\)s\(^{-2}\).

COMMON CONVERSION FACTORS

Although SI has been adopted by Canadian hospitals, there is still a need to know some units in the metric and imperial systems and be able to convert these units to SI.

Patients, unfamiliar with SI may give pertinent information in the imperial system.

Textbooks and periodicals used as reference sources may have been written either before the adoption of SI or by individuals in other countries where the conversion to SI is not yet completed.

TEMPERATURE CONVERSIONS

Formulas:

\[ \text{\(^{\circ}\)C} = \frac{5}{9} (\text{\(^{\circ}\)F} - 32) \]

\[ \text{\(^{\circ}\)F} = \frac{9}{5} (\text{\(^{\circ}\)C} + 32) \]

WEIGHT CONVERSIONS

2.2 pounds (lb) = 1 kg.

LENGTH CONVERSIONS

1 inch = 2.54 cm

1 mile = 1.6 km

1 mile per hour = 1.6 km per hour
ADVANCED LIFE SUPPORT
PRE COURSE
UNITS OF MEASUREMENT AND MEDICAL MATH
SELF-ASSESSMENT

Marks

[3] 1. Name six base units of the SI systems and state the physical quantity that they measure.

[2] 2. Differentiate between a base unit and a derived unit in the SI system.

[1] 3. Name the special derived unit of volume used by the health technologies.

[1] 4. a) State the derived unit for temperature used by the health technologies. State the scale used commonly in the home.

b) State the relationship between these two units.
5. Convert the following:

a) 500 mL = ________________ L

b) 500 mL = ________________ µL

c) 0.5 cc = ________________ mL

d) 4.55 kg = ________________ g

e) 495.8 mg = ________________ g

f) 294 µg = ________________ mg

g) 56.5 cm = ________________ m

6. Convert the following:

a) 175 lb = ________________ kg

b) 43 inches = ________________ cm

c) 27°F = ________________ °C

d) 48°C = ________________ °F

e) 56.7 kg = ________________ lb

7. You have an I.V. bag containing 250 mL of fluid. Your administration set delivers 60 drops per minute. Sixty drops comprise 1 mL of fluid. The physician orders an administration rate of 75 cc/hr.

a) How many drops per minute will you give?

b) How long will it take to empty the bag?

c) 250 mL = ________________ L
8. One gram of a medication is supplied pre-mixed in 250 mL of fluid. Express the concentration of the drug in mg/mL.

9. A medication is supplied in a 200 mg pre-loaded syringe. You add this to a 250 mL bag of IV fluid. Express the concentration of the medication in mg/mL.

27 TOTAL
ADVANCED LIFE SUPPORT
PRE COURSE
UNITS OF MEASUREMENT AND MEDICAL MATH

SELF-ASSESSMENT ANSWERS

1. For each incorrect answer deduct ¼ of a mark.

### BASE UNIT | PHYSICAL QUANTITY
---|---
metre (m) | length
kilogram (kg) | mass
second (s) | time
mole (mol) | amount of substance
kelvin (K) | temperature
ampere (A) | electrical current

2. ○ A base unit can be independently defined or calculated.
○ A derived unit is defined in terms or base units or other derived units.

3. The special derived unit of volume is the litre (L).

4. a) The derived unit is the degree Celsius (°C). The common unit is the degree Fahrenheit (°F).

b) These units are related as follows:

\[
°\text{C} = \frac{5}{9} (°\text{F} - 32) \quad \text{[or]} \quad °\text{F} = \frac{9}{5} °\text{C} + 32
\]

5. a) \(500 \text{ mL} = 500 \times 10^{-3} = 0.500 \text{ L}\)
b) \(500 \text{ mL} = 500 \times 10^{-3} = 5 \times 10^5 \mu\text{L} \text{ or } 500,000 \mu\text{L}\)
c) \(0.5 \text{ cc} = 0.5 \text{ mL}\)
d) \(4.55 \text{ kg} = 4.55 \times 10^3 = 4550 \text{ g}\)
e) \(495.8 \text{ mg} = 495.8 \times 10^{-3} = 0.4958 \text{ g}\)
f) \(294 \mu\text{g} = 294 \times 10^{-3} = 0.294 \text{ mg}\)
g) \(56.5 \text{ cm} = 56.5 \times 10^{-2} = 0.565 \text{ m}\)

6. Convert the following:

a) \(175 \text{ lb} = 175 \div 2.2 = 79.5 \text{ kg}\)
b) \(43 \text{ inches} = 43 \times 2.54 = 109 \text{ cm} = 1.09 \text{ m}\)
c) \(27°C = -2.8°C\)
d) \(48°C = 118.4°F\)
e) \(56.7 \text{ kg} = 56.7 \times 2.2 = 124.7 \text{ lb}\)
7. a) 75 cc/hr = 75 mL/60 min
   \[\frac{75}{60} = 1.25 \text{ mL/min} = 1.25 \times 60 = 75 \text{ drops/min}\]

b) 75 mL/hr; 250 mL will take \(\frac{250}{75}\) = 3.33 hours

c) \(\frac{0.25 \text{ L}}{75}\)

8. 1 g = 1000 mg
   \[\frac{1000 \text{ mg}}{250 \text{ mL}} = 4 \text{ mg/mL}\]

9. \(200 \text{ mg}\)
   \[\frac{250 \text{ mL}}{4 \text{ mg/mL}} = 0.8 \text{ mg/mL}\]

If you have experienced difficulties in the self-assessment, proceed to the mathematics review exercises to assist you in developing accuracy in handling mathematical calculations and conversions.
## APPENDIX A

### COMMON DERIVED UNITS OF THE SI SYSTEM

#### SI DERIVED UNITS WITH SPECIAL NAMES*

<table>
<thead>
<tr>
<th>QUANTITY</th>
<th>NAME OF UNIT</th>
<th>SYMBOL FOR UNIT</th>
<th>DERIVATION OF UNIT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Celsius temperature</td>
<td>degree Celsius</td>
<td>°C</td>
<td>K</td>
</tr>
<tr>
<td>force</td>
<td>newton</td>
<td>N</td>
<td>Kg m/s²</td>
</tr>
<tr>
<td>pressure</td>
<td>pascal</td>
<td>Pa</td>
<td>N/m²</td>
</tr>
<tr>
<td>work; energy; quantity of heat</td>
<td>joule</td>
<td>J</td>
<td>N°m</td>
</tr>
<tr>
<td>power</td>
<td>watt</td>
<td>W</td>
<td>J/s</td>
</tr>
<tr>
<td>electric charge; quantity of electricity</td>
<td>coulomb</td>
<td>c</td>
<td>A°s</td>
</tr>
<tr>
<td>electrical potential; potential difference</td>
<td>volt</td>
<td>v</td>
<td>W/A</td>
</tr>
<tr>
<td>electric resistance</td>
<td>ohm</td>
<td>O</td>
<td>V/A</td>
</tr>
</tbody>
</table>

*These will be useful to the students who will be studying defibrillation in subsequent modules.
<table>
<thead>
<tr>
<th>QUANTITY</th>
<th>SI UNIT</th>
<th>SYMBOL</th>
<th>CUSTOMARY UNIT</th>
<th>TYPICAL APPLICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>8. energy</td>
<td>joule</td>
<td>J</td>
<td>Calorie kilocalorie, calorie</td>
<td>food energy, metabolic energy, kinetic energy</td>
</tr>
<tr>
<td></td>
<td>kilojoule</td>
<td>kJ</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. pressure</td>
<td>pascal</td>
<td>Pa</td>
<td>millimetre of mercury, inches of water</td>
<td>ocular, cerebrospinal, and blood pressure</td>
</tr>
<tr>
<td></td>
<td>kilopascal</td>
<td>kPa</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. frequency</td>
<td>hertz</td>
<td>Hz</td>
<td>cycle per second</td>
<td>audio, radio and x-ray frequencies</td>
</tr>
<tr>
<td>11. substance</td>
<td>mole/litre</td>
<td>mol/L</td>
<td>milligram per cent milliequivalent per litre</td>
<td>composition of body fluid.</td>
</tr>
<tr>
<td>concentration</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
MATHEMATICAL REVIEW EXERCISES

STANDARD OR SCIENTIFIC NOTATION

Frequently it is necessary to represent very large or very small quantities numerically.

The mass of an electron is:
0.000 000 000 000 000 000 000 000 911g

The number of molecules in a mole, known as Avogadro’s number is:
620 000 000 000 000 000 000 000.

Writing and working with numbers in this form is difficult and may lead to mathematical errors. To avoid this, such numbers are expressed in a standard form using powers of 10.

\[10^4\text{ is a power of 10}\]

[10 is the base] \quad [4 \text{ is the exponent}]

The standard or scientific form of a number consists of:

A value between 1 and 10 \( \times \) a power of 10 to locate the decimal point

Example:

\[2 \times 10^4\]
\[2 \times 10^{-3}\]

For numbers greater than 1, the exponent is positive and indicates how many places the decimal point is located to the right.

Example:

\[2 \times 10^4 = 2 \times 10 \times 10 \times 10 \times 10 = 20,000\]

For numbers less than 1, the exponent is negative and indicates how many places the decimal point is located to the left.

Example:

\[2 \times 10^{-3} = \frac{2}{10 \times 10 \times 10} = 0.002\]

The standard or scientific form of the mass of an electron is written: \(9.11 \times 10^{-23}\) g
The standard or scientific form of the mass of Avogadro’s is written: \(6.02 \times 10^{23}\) g
Exercise 1.1

Study the following tables and complete the missing information.

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>40</td>
<td>4 x 10</td>
<td>4 x 10¹</td>
</tr>
<tr>
<td>400</td>
<td>4 x 100</td>
<td>4 x 10²</td>
</tr>
<tr>
<td>4000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7680</td>
<td>7.68 x</td>
<td></td>
</tr>
<tr>
<td>76,800</td>
<td></td>
<td>7.68 x</td>
</tr>
<tr>
<td>370</td>
<td></td>
<td>3.7</td>
</tr>
<tr>
<td>6,200,000</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Standard</th>
<th>Exponent Number</th>
<th>Form of 10</th>
</tr>
</thead>
<tbody>
<tr>
<td>52,600</td>
<td>5.26 x 10,000</td>
<td>5.26 x 10⁴</td>
</tr>
<tr>
<td>5,260</td>
<td>5.26 x 1,000</td>
<td>5.26 x 10³</td>
</tr>
<tr>
<td>526</td>
<td>5.26 x 100</td>
<td>5.26 x 10²</td>
</tr>
<tr>
<td>52.6</td>
<td>5.26 x 1</td>
<td>5.26 x 10⁰</td>
</tr>
<tr>
<td>0.526</td>
<td>5.26 x 10⁻¹</td>
<td>5.26 x 10⁻¹</td>
</tr>
<tr>
<td>0.0526</td>
<td>5.26 x 10⁻²</td>
<td>5.26 x 10⁻²</td>
</tr>
<tr>
<td>0.00526</td>
<td>5.26 x 10⁻³</td>
<td>5.26 x 10⁻³</td>
</tr>
</tbody>
</table>

Exercise 1.2

Rewrite the following in standard form:

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a) 579</td>
<td>b) 1300</td>
<td>c) 47.6</td>
<td>d) 724.8</td>
</tr>
<tr>
<td>e) 12,000,000</td>
<td>f) 38,200.</td>
<td>g) 6</td>
<td>h) 980,000.</td>
</tr>
<tr>
<td>i) 0.0523</td>
<td>j) 0.246</td>
<td>k) 0.00761</td>
<td>l) 0.00864</td>
</tr>
<tr>
<td>m) 0.000002</td>
<td>n) 0.179</td>
<td>o) 0.000751</td>
<td>p) 0.0297</td>
</tr>
</tbody>
</table>
Exercise 1.3

Expand each of the following:

a) $4 \times 10^2$  
b) $5.72 \times 10^5$  
c) $6.3 \times 10^1$

d) $1.89 \times 10^{-2}$  
e) $2.34 \times 10^{-1}$  
f) $9.09 \times 10^4$

g) $5.55 \times 10^7$  
h) $4.12 \times 10^{-6}$  
i) $8.76 \times 10^3$

j) $7.34 \times 10^{-4}$  
k) $6.19 \times 10^{-3}$  
l) $5.82 \times 10^3$

m) $4.79 \times 10^{-3}$  
n) $5.26 \times 10^{-1}$  
o) $4.38 \times 10^6$

p) $3.24 \times 10^{-2}$

UNITS OF MEASURE

In dealing with units of measurement, e.g. length, mass and volume, it is necessary to convert units from one form into another quickly and accurately. This is particularly important when dealing with drugs, their concentration (mass per unit volume) and their dosages (concentration per unit body mass). The following examples and exercises are designed to give the student practice with the units of measurement singly and then in combination.

MASS

The units of mass are expressed in terms of the gram (g).

1 kilogram (kg) = $1000 \, g \, (10^3 \, g)$
1 milligram (mg) = $1/1000 \, g \, (10^{-3} \, g)$
1 microgram ($\mu g$) = $1/1000000 \, g \, (10^{-6} \, g)$

Interconversion Units of Mass

<table>
<thead>
<tr>
<th>kg</th>
<th>g</th>
<th>mg</th>
<th>$\mu g$</th>
</tr>
</thead>
</table>
| kilogram | kg x $10^3$ = g | kg x $10^6$ = mg | kg x $10^9$ = $\mu g$
| g x $10^{-3}$ = kg | gram | g x $10^{-3}$ = mg | g x $10^{-6}$ = $\mu g$
| mg x $10^{-6}$ = kg | mg x $10^{-3}$ = g | milligram | mg x $10^{-3}$ = $\mu g$
| $\mu g$ x $10^{-9}$ = kg | $\mu g$ x $10^{-6}$ = g | $\mu g$ x $10^{-3}$ = mg | microgram |
Examples

Convert 0.05 g to mg

\[ g \times 10^3 = mg \]
\[ 0.05 \, g \times 10^3 = 50 \, mg \]

Convert 0.05 g to \( \mu g \)

\[ g \times 10^6 = \mu g \]
\[ 0.05 \, g \times 10^6 = 50000 \, \mu g \]

Convert 7.2 mg to g

\[ mg \times 10^{-3} = g \]
\[ 7.2 \, mg \times 10^{-3} = 0.0072 \, g \]

Convert 7.2 mg to \( \mu g \)

\[ mg \times 10^3 = \mu g \]
\[ 7.2 \, mg \times 10^3 = 7200 \, \mu g \]

Exercise 2.1

Express each of the following in grams.

a) 52 mg 
   b) 645 \( \mu g \) 
   c) 2.7 kg 
   d) .032 mg 
   e) 9 \( \mu g \) 
   f) 8763 mg 
   g) 71860 \( \mu g \) 
   h) 0.45 kg

Exercise 2.2

Express each of the following in milligrams.

a) 0.021 g 
   b) 62.3 \( \mu g \) 
   c) 0.00731 kg 
   d) 175 g 
   e) 89 kg 
   f) 0.49 \( \mu g \) 
   g) 0.000056 g 
   h) 12347 \( \mu g \)
WEIGHT CONVERSIONS

Patients, unfamiliar with SI will give their weight in terms of pounds.

1 kg = 2.2 pounds (lb)

\[ \text{kg} = \frac{\text{weight in pounds}}{2.2} \]

Exercise 2.3

Express each of the following in kilograms. Do all calculations mentally. Express your answer to the nearest whole kg.

a) 45 lbs  b) 130 lbs  c) 105 lbs  d) 175 lbs
e) 114 lbs  f) 150 lbs  g) 200 lbs  h) 80 lbs
i) 210 lbs  j) 27 lbs  k) 183 lbs  l) 142 lbs

VOLUME

The units of measurement for volume are expressed in terms of the litre (L).

1 litre (L) = 1000 millitres (mL)

The abbreviation cc (cubic centimetres) is now replaced by the millilitre (mL).

Another measure of volume applies specifically to Intravenous (IV) drips where the drip rate (number and size of drops) determines the volume of fluid delivered to the patient.

Exercise 3.1

Express the following in millilitres (mL).

a) 0.250 L  b) 1.86 L  c) 0.00013 L

\[ \underline{\text{d) 0.0782 L}} \quad \underline{\text{e) 59.2 L}} \quad \underline{\text{f) 0.00037 L}} \]

\[ \underline{\text{g) 0.563 L}} \quad \underline{\text{h) 0.00945 L}} \]
Exercise 3.2

For an IV drip, regular tubing, 10 drops (10 gtt) equals 1 mL. How many drops are required to deliver the following volumes?

a) 5.0 mL b) 250 mL c) 0.003 L

d) 75 mL e) 0.5 L f) 0.10 mL

g) 12 mL h) 1 L

Exercise 3.3

For an IV minidrip, 60 drops (60 gtt) equals 1 mL. How many drops are required to deliver the following volumes?

a) 0.125 mL b) 186 mL c) 7.8 mL

d) 13 L e) 563 mL f) 0.0009 L

g) 0.75 mL h) 0.08 L

CONCENTRATION

Concentration is the quantity of a substance, e.g. a drug, dissolved in a given volume of solution. There are many ways of expressing concentration:

- mol/L moles per litre (SI unit)
- g/L grams per litre
- g/dL grams per decilitre = grams per 100 mL
- g% or % grams per 100 mL
- mg% milligrams per 100 mL
- Eq/L equivalents per litre
- mEq/L milliequivalents per litre

Though the SI unit is mol/L, all other units are commonly used particularly in expressing the concentration of drugs. Given the concentration of a drug, the student must be able to determine quickly and accurately the volume required to obtain a given concentration or the quantity of drug in a given volume.
Exercise 4.1

Calculate the concentration in mg/mL for each of the following solutions.

a) 1 g/L  ___________  b) 0.6 g%  ___________  c) 200 mg%  ___________

d) 0.5 g/dL  ___________  e) 0.2 g/L  ___________  f) 10 g/dL  ___________

g) 20 g/L  ___________  h) 4000 mg%  ___________  i) 0.1 g/L  ___________

Exercise 4.2

For each of the solutions in Exercise 4.1, calculate the number of mg contained in the volumes shown.

a) 0.5 cc of (a)  ___________  b) 3.5 cc of (b)  ___________  c) 0.2 cc of (c)  ___________

d) 2 mL of (d)  ___________  e) 5.0 mL of (e)  ___________  f) 10.00 mL of (f)  ___________

g) 2.5 mL of (g)  ___________  h) 4.0 mL of (h)  ___________  i) 9 mL of (i)  ___________
Exercise 4.3

For each of the solutions in Exercise 4.1, calculate the number of mL required to obtain the following quantity.

a) 7.3 mg of (a) _________  
b) 12 mg of (b) _________  
c) 1 mg of (c) _________

d) 7.5 mg of (d) _________  
e) 0.8 mg of (e) _________  
f) 650 mg of (f) _________

g) 85 mg of (g) _________  
h) 30 mg of (h) _________  
i) 0.4 mg of (i) _________

PROBLEMS

The following problems require the student to integrate calculations involving mass, volume and concentration. They are representative of the type of problems that can be encountered in the prehospital setting.

5.1 An IV, regular tubing (10 gtt/mL) is to be adjusted to deliver 250 cc per hour. How many drops per minute are required?

5.2 Nitroglycerine tablets contain 0.3 mg per tablets. The physician orders 0.6 mg. How many tablets are required?
5.3 Your drug kit contains a 10 cc pre-loaded syringe containing epinephrine in a concentration of 1:10000 (that is 1 g in 10 000 mL). How many mL must be used to provide a dose of 0.5 mg?

5.4 An IV bag contains 250 mL 5% D/W mixed with 5 mL Dopamine, concentration 40 mg/mL. What is the resulting concentration of Dopamine in mg/mL and in g/L? (For the total volume in IV bag, ignore the 5 mL volume of drug in your calculations.)

5.5 Lidocaine is provided in an IV solution containing 1 g/250 mL and administered as a minidrip (60 gtt/mL). How many drops per minute are required to achieve a rate of administration of 2 mg/min?

5.6 Your drug kit contains a 5 cc pre-loaded syringe containing lidocaine in a concentration or 20 mg/mL. The patient weights 70 kg. You are ordered to prepare 3 doses of 1 mg/kg. How many mL will you require?
MATHEMATICAL REVIEW EXERCISES

The answers are given in the complete form including units, decimal points, and a zero before the decimal as required. An answer is correct only if complete in all detail.

Exercise 1.1

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Exercise 1.2

Rewrite the following in standard form:

a) $5.79 \times 10^2$

b) $1.3 \times 10^3$

c) $4.76 \times 10^1$

d) $7.248 \times 10^2$

e) $1.2 \times 10^7$

f) $3.82 \times 10^4$

g) $6 \times 10^0$

h) $9.8 \times 10^5$

i) $5.23 \times 10^{-2}$

j) $2.46 \times 10^{-1}$

k) $7.61 \times 10^{-3}$

l) $8.64 \times 10^{-3}$

m) $2 \times 10^{-6}$

n) $1.79 \times 10^{-1}$

o) $7.51 \times 10^{-4}$

p) $2.97 \times 10^{-2}$

Exercise 1.3

a) 400

b) 572 000

c) 63

d) 0.0189

e) 0.234

f) 90 900

g) 55 500 000

h) 0.00000412

i) 8760

j) 0.000734

k) 0.00619

l) 5820

m) 0.00479

n) 0.526

o) 4 380 000

p) 0.0324
Exercise 2.1

a) 0.052 g  
b) 0.000645 g

c) 2700 g  
d) 0.00032 g

e) 0.000009 g  
f) 8.763 g

g) 0.07186 g  
h) 450 g

Exercise 2.2

a) 21 mg  
b) 0.0623 mg

c) 7310 mg  
d) 175 000 mg

e) 89 000 000 mg  
f) 0.00049 mg

g) 0.056 mg  
h) 12.347 mg

Exercise 2.3 (Answer correct if within +1 kg)

a) 20 kg  
b) 59 kg

c) 48 kg  
d) 80 kg

e) 52 kg  
f) 68 kg

g) 91 kg  
h) 36 kg

i) 95 kg  
j) 12 kg

k) 83 kg  
l) 64 kg
Exercise 3.1

a) 250 mL  
b) 1860 mL  
c) 0.013 mL  
d) 78.2 mL  
e) 59 200 mL  
f) 0.37 mL  
g) 563 mL  
h) 9.45 mL  

Exercise 3.2

a) 50  
b) 2500  
c) 30  
d) 750  
e) 5000  
f) 1  
g) 120  
h) 10 000  

Exercise 3.3

a) 7500  
b) 11 160  
c) 468  
d) 780 000  
e) 33 780  
f) 54  
g) 45  
h) 4800
Exercise 4.1

a) 1 mg/mL  
b) 6 mg/mL  
c) 2 mg/mL  
d) 5 mg/mL  
e) 0.2 mg/mL  
f) 100 mg/mL  
g) 20 mg/mL  
h) 40 mg/mL  
i) 0.1 mg/mL

Exercise 4.2

a) 0.5 mg  
b) 21 mg  
c) 0.4 mg  
d) 10.0 mg  
e) 1.0 mg  
f) 1000 mg  
g) 50 mg  
h) 160mg  
i) 0.9 mg

Exercise 4.3

a) 7.3 mL  
b) 2.0 mL  
c) 0.5 mL  
d) 1.5 mL  
e) 4.0 mL  
f) 6.5 mL  
g) 4.25 mL  
h) 0.75 mL  
i) 4.0 mL
5.1  42 drops
5.2  2 tablets
5.3  5 mL
5.4  0.8 mg/mL; 0.8 g/L
5.5  30 drops per min
5.6  3.5 mL
ADVANCED LIFE SUPPORT
PRE COURSE

MEDICAL MATH

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.

   Reading  __________ hours
   Self assessment  __________ hours
   Total time  __________ hours

2. Were the objectives of the module clearly stated?

   [ ] yes  [ ] no
   If no, please comment.

3. Did you use any of the resource materials?

   [ ] yes  [ ] no
   If yes, which items
   ..................................................................................
   ..................................................................................
   ..................................................................................
   Were they helpful?  ..................................................................................

4. Were the reference notes adequate?

   [ ] yes  [ ] no
   If no, please comment.
5. Were the reference notes easy to follow?

[ ] yes [ ] no
If no, please comment.

6. Were the examples provided satisfactory?

[ ] yes [ ] no
If no, please comment.

7. Were the diagrams helpful?

[ ] yes [ ] no
If yes, please specify.

8. Was the level of the module satisfactory for your program of study?

[ ] yes [ ] no
If no, please comment.

Base Hospital ____________________________________________

9. General comments or suggested improvements.
INTRAVENOUS PRECOURSE PACKAGE

2007 Ontario Base Hospital Group
PCP
INTRAVENOUS
PRECOURSE PACKAGE

Authors

Mike Muir AEMCA, ACP
Kevin McNab AEMCA, ACP
**Purpose**

This module will instruct Primary Care Paramedics, through a combination of take home packages, in class practical, and in hospital practical training, how to establish intravenous access. The paramedic taking the IV/IO access module will learn standing orders, indications, complications, anatomy, physiology, and become practically proficient in IV cannulation.

**Components**

1. **Theoretical:** This component will consist of a take home package, which the paramedic will have to be intimately familiar with on the first day of class. It will also contain a take home theoretical test, to self-test his or her knowledge prior to attending class.

2. **Theoretical testing:** On the training day, the paramedic will be tested on his or her knowledge of the take home package. The Passing mark is 80%. Those not achieving 80% must return to a future class and will not be able to continue that day.

3. **In Class Practical:** This consists of theory review and practical instruction using IV practice limbs.

4. **In Hospital Practical:** This is the practical component where paramedics use their knowledge to initiate IV cannulation in Day Surgery, Emergency Departments and other clinical settings to gain practical knowledge and experience.
Certification

As with other skills, yearly certification of IV cannulation will be required by the Medical Director. This may be achieved through a skills inventory (database review) and/or include a writing a pre-test, and starting an IV with BHP assessment.

OBJECTIVES

*In completing this module, the PCP will:*

**Effective**

Maintain a patient’s dignity at all times

Use appropriate language

Maintain patient confidentiality

Demonstrate ethical behaviour

Function as a patient advocate

Function within the scope of practice defined by provincial regulating agencies and local medical control

Explain to the patient, when asked, “patient rights”, and be mindful of those rights on the role of provider

Work collaboratively with other members of the healthcare team

Accept and deliver constructive feedback

Demonstrate reasonable and prudent judgement

Practice effective problem solving skills
Cognitive

Be familiar with the anatomy and physiology of both upper and lower extremities, as related to IV cannulation.

Identify pathophysiology of the immune and cardiovascular system, hypovolemia, hypoperfusion and shock.

Be able to relate factors that effect vasodilation.

Relate indications and contraindications for IV cannulation.

Describe the properties of Normal Saline, its uses and overdose symptomology.

Describe circumstances where a “bolus” of NS may be required.

Have knowledge of Base hospital policies and provincial ALS standards with regard to IV cannulation.

PSYCHOMOTOR

SIMULATED

Demonstrate the selection of appropriate equipment for given situations.

Demonstrate proper technique in “flushing” an IV line with natural saline.

Identify criteria for vein selection.

Demonstrate aseptic techniques required for IV and intraosseous infusions.

Identify steps required to secure IV cannula and IV tubing.
“Troubleshooting” difficult IVs including removing air bubbles, and checking for IV patency.
Demonstrate competence in giving infusions under pressure
Calculate IV “drip rates”
Identify “interstitial” IVs
Demonstrate the skill of infusing non-Colloids and Volume Expanders

CLINICAL
Demonstrate proper technique in intravenous access

Rights of the Patient
Any patient may refuse any treatment for any reason at any time.
Some patients may refuse simply because they are afraid of needles or for complex reasons such as religious beliefs. The paramedic must balance the need for an IV with the person’s wishes. If the patient refuses because of a fear of needles, and the IV/IO is a necessity, it is best to talk the patient into the treatment with reasoning such as it “only last a moment” or the “needle part” does not stay in. The cannula that stays in is soft and flexible.
People with religious objections may require further assurances. Religious orders such as Jehovah’s Witnesses object to the introduction of whole blood, packed red cells, white blood cells, plasma or platelets. They will accept non-blood replacement fluids like hetastarch and crystalloids. If questioned, the fluid being instilled is Normal Saline, a non-blood product (see composition of N/S in equipment section of this package).
Implied consent with the unconscious Jehovah’s Witness patient has been ruled on by the Ontario Supreme Court. Jehovah’s Witnesses carrying a dated, witnessed card stating their intentions, are to be honoured even after unconsciousness.

Anatomy Relevant to Intravenous Starts

Definitions
An intravenous refers to the cannulation of a vein in order to introduce fluid, blood or medication in to the circulatory system

VEINS, by definition deliver un-oxygenated blood (at relatively low pressures) back to the heart and lungs for re-oxygenation and
distribution. Veins are superficial in nature, located just below the subcutaneous tissue, making them ideal for cannulation. On successful cannulation of a vein, blood should look dark and flow slowly to IV cannula “window”.

**ARTERIES**, by definition, deliver oxygenated blood (at a higher pressure than veins) to the body. Arteries are thick walled, deeply inset vessels that should not be cannulated by prehospital care providers. If cannulated, the blood will appear bright red and fill the IV cannula window rapidly. If inadvertently accessed, remove the cannula, apply a sterile dressing with firm pressure over the site for five minutes.
VALVES: medium to large veins use a structure called a valve to help move blood against gravity, toward the heart. These valves consist of folds in the tunica intima, which act in the same manner as the semi lunar valves in the heart. The valves overlap, and when blood attempts to flow backward at diastole, the valves occlude the vein. There are many valves in a section of vein, and the number of valves generally increases with need (The number of valves in the lower extremities are greater than the number in the upper extremities).

With age, these valves can become “incompetent”, resulting in backflow and damage to the distal structures. This is called a varicose vein. The area is generally reddened and edematous. With sufficient damage, the vein can develop stagnant areas, and blood clots may develop. Therefore, cannulating a varicose vein is contraindicated.

Valves can also become “stenotic”. They do not open or close fully. It allows blood to flow backward and impedes distal blood flow. The paramedic cannulating a vein distal to a stenotic valve will find it difficult to advance the cannula past it, and if the cannula stops short of the valve, the flow of IV fluid will be diminished or blocked. A stenotic valve can generally be identified as a small (1-2 mm) raised and enlarged section in the vein. When palpated, it is harder than the rest of the vein.
Veins of the Upper Extremity

_Proximal Cephalic, Basilic and Median Cubital Veins_

- Most commonly referred to as “antecubital veins”
- Are large and usually very prominent, even without a tourniquet
- Normally reserved for venipuncture and emergency IV infusions (e.g. necessity to give large quantities of fluid or blood products due to burns, hypovolemia, and shock states)
- Reserve for short term/emergency use, due to it being at a moveable joint
- Readily accepts large # 14, 16, 18 IV catheters
- Care should be taken not to cannulate the brachial artery or any of the numerous nerve endings in the area
- Site most successfully cannulated during transport
**Distal Basilic Vein**

- Runs along the ulnar aspect of the arm
- Large and prominent in males
- Often overlooked due to its location on the underside of the arm
- This vein is prone to “rolling”, making cannulation for difficult
- Best cannulated with the arm in an “arm wrestling stance”
- Best cannulated with # 18, 20 catheters
**Distal Cephalic Vein**

- Runs along the radial aspect (thumb side) of the arm
- Bones of the forearm provide natural splint
- Is a medium sized vein; ideal, for example, for long term infusion of antibiotics, non emergency cardiac medication
- Sometimes difficult to cannulate due to it’s tendency to roll, and the need to cannulate around the thumb and thanar prominence
- Best accessed with a #18, 20 catheter

**Medial Ante Brachial Vein**

- Runs down the center of the anterior aspect of the forearm
- Medium sized vein
- Easily visualized, difficult to palpate
- Does not tend to roll
- **DO NOT** cannulate the extreme distal end of this vein due to the numerous nerve endings, moveable joint (impossible to secure) and extreme pain caused
- Appropriately cannulated with a #18, 20 catheter

**Metacarpal Veins**

- Located in the back of the hand
- Small to medium sized veins
- Ideal for long term /TKVO therapy
- Positional IV if catheter is located too far back on the hand/wrist
- Best cannulated with a #18, 20 or 22 catheter
Digital Veins

- Small veins, located in the fingers (not generally used in the prehospital setting)
- Area of last resort as it is positional, painful and difficult to secure
- Cannulated only with # 22 catheter
Veins of the Lower Extremities

Great Saphenous Vein

- Mid calf to the internal malleolus
- Large vein
- Cannulation limits patient’s mobility
- Moderate risk of keep vein thrombosis
- Risk of impaired circulation in the lower leg and foot
**Dorsal Venous Network**

- Located on the dorsal surface (top) of the foot
- Medium sized to small veins
- Painful access
- Increased risk of DVT

**Factors Influencing Vasodilation/Vasoconstriction**

There are many factors, which influence vasoconstriction and vasodilation, all of which affect the pressures inside. Manipulating the pressure upward is what makes IV cannulation easier. Natural influences can conversely make IV starts more difficult through vasoconstriction or collapse.

**Vasodilation**

- **Tourniquet** - application of a tourniquet constricts the flow of blood at the afferent end of the vein, mechanically enlarging and expanding the vessel. A tourniquet may consist of a blood pressure cuff or a soft rubber band.

- **Gravity** – lowering the limb below the level of the heart dilates veins as the force of gravity pulls blood into dependant areas. Use this in conjunction with a tourniquet.

- **Mechanical Stimulation** – “flicking” fingers or gentle “slapping” with 2 fingers over the venipuncture site produces a short lived venous dilation.

- **Muscular Activity** – opening and closing the fist is the most popular method.

- **Application of Heat** – applying heat for 10 minutes will increase blood flow to the area, causing vasodilation of arteries and veins in the area.
Volume Loading – patients in CHF or women during pregnancy have increased intravascular blood volume. Patients with these conditions generally do not require further mechanical methods.

Vasoconstriction

Gravity – raising the limb above the level of the heart reduces the blood flow and induces vasoconstriction. This factor is already employed by the paramedic as a means to control bleeding.

Cold – Application of cold packs reduces flow to the affected region, and induces vasoconstriction. This is useful to the paramedic wanting to reduce the swelling of missed IV attempts, and the prevention of haematomas. Hypothermic patients should be warmed prior to attempting IVs due to the difficulty in cannulating a shivering, vasoconstricted patient.

Hypovolemia & Shock – the body’s natural mechanism in dealing with a decreased circulating blood volume is to shunt blood from the periphery to the core, making cannulation of a peripheral vein difficult. The paramedic should recognize the need to increase circulating volume rapidly and attempt to cannulate a large, antecubital vein.

Vasovagal Response – fear or anxiety may trigger a vasovagal response, resulting in an undesirable vasodilation, drop in BP and syncopal symptoms. Pain and anxiety from further IV sticks may increase these symptoms. Patients may identify this at first contact, saying “People have a hard time getting blood from me”. This may be reversed with decreasing their anxiety, confidence shown by the paramedic, and a calm demeanour.
Physiology Relevant to IV Starts

There are some basic principles, which affect the flow of blood through the circulatory system. It is the interrelationships between these factors, which regulate blood pressure, blood flow, and play a vital role in the function of the circulatory system.

- **Viscosity** – viscosity is the measure of the resistance of a liquid to flow. In other words, as the viscosity of liquid increases, so does the pressure required to force it to flow. The viscosity of blood is largely influenced by the amount of hematocrit (blood cells e.g. RBCs, WBCs).

- **Laminar and Turbulent Flow in Vessels** – laminar flow describes the flow of blood, or a fluid, through a smooth walled vessel. It flows slowest where the blood makes contact with a vessel wall, and fastest toward the center (where there is little resistance). Laminar flow is interrupted, and becomes turbulent, when it comes into contact with a constriction, sharp bend in a vessel or a rough surface. Turbulent blood flow is what makes it possible to auscultate a blood pressure (the paramedic hears the turbulent blood flow at the antecubita when the vessels are compressed with a BP cuff).

- **Blood Pressure** – BP is a measure of the force blood exerts against the vessel walls. It can be measured by auscultating and occluding a blood vessel, or by inserting a cannula into an artery and connecting an electronic pressure transducer to it. This commonly referred to as an ART (or arterial) line.

**Rate of Blood Flow** – The rate of blood flow is measured by the amount of blood that passes through a specific amount of blood vessel (or organ), and is usually measured in Litres per Minute.
Poiseuille’s Law

According to Poiseuille’s law, the flow of blood is dramatically increased when the radius of the blood vessel is increased. Conversely, a small decrease in the size of a blood vessel, results in a dramatic decrease in blood flow. Additionally, an increase in viscosity, or an increase in vessel length decreases blood flow.

Law of LaPlace

The Law of LaPlace helps to explain the phenomenon known as Critical Closing Pressure. The law states that the force that stretches the vascular wall is proportional to the diameter of the vessel, times the blood pressure. As the pressure in the vessel decreases, the vessel wall size also decreases. Some minimum pressure is required to keep the blood vessel open; if the pressure falls so that the force is below the minimum required, the vessel will collapse. Conditions causing collapse may include shock states.

Conversely, the development of aneurisms may be explained. As the diameter increases, the force applied to the vessel wall also increases, even with a constant pressure. If an artery has a weakened wall, and it develops a “bulge”, the force applied to that area is greater than any other section of the artery, because the diameter is larger. A negative feedback loop follows (i.e. the larger the weakened area becomes, the more force applied to it, resulting in a larger bulge, resulting in more force being applied…)

Vascular Compliance

Vascular compliance is the tendency of a blood vessel volume to increase as the pressure increases. Venous compliance is greater than arterial compliance by a large margin. This high compliance results in the body using the venous system as a storage area.

Systemic Circulation

System circulation will, depending on vessel type, location, and size, have different pressure in them. Also, because the heart is pulsatile, pressure will vary during systole and diastole.
The aorta, which has a large diameter and is close to the heart, loses very little pressure. Its systolic pressure is 120mmHg and diastolic is 80mmHg. As blood flows through the arteries, capillaries and veins, the pressure progresses toward 0mmHg, or even lower by the time it returns to the right atrium.

Blood pressure is centrally regulated by the nervous system; namely the vasomotor center of the Medulla Oblongata. Baroreceptors, Pressoreceptors and Chemoreceptors in major vessels in both the arterial and venous circulatory systems are sensitive to stretch, shrinkage or abnormal blood gases. On reception of abnormal readings, messages are transmitted to the cardio-regulatory and vasomotor centres.

In the case of low BP, sympathetic stimulation increases heart rate, vasoconstricts peripheral vessels.

Increased BP results in, again, cardio regulatory centres in the medulla, being notified. Vasodilation and increased parasympathetic vagal innervation of the heart results. The heart rate falls and blood pressure returns to normal.
Chemoreceptors react to low oxygen levels, high levels of carbon dioxide or abnormal blood pH. These act under “emergency” conditions, most notably, if BP falls below 80-mmHg or oxygen levels in the blood falls markedly.

In addition to nervous regulation of arterial pressure, 4 hormonal mechanisms also have influence on blood pressure.

1. **Adrenal Medullary Mechanisms** – Sympathetic nervous stimulation also acts on the adrenal medulla. Epinephrine and Norepinephrine are secreted, affecting the cardiovascular system by increasing heart rate and causing vasoconstriction.

2. **Renin/Angiotensin/Aldosterone Mechanism** – On sensing low blood pressure, the kidney secretes an enzyme called “Renin” from juxtaglomerular apparatuses. Renin acts on a plasma protein called *Angiotensinogen*, splitting it up.

One of the resultant fragments is called “*Angiotensin I*”. Other enzymes in the lung further act to change Angiotensin I into a chain of amino acids called *Angiotensin II*. Angiotensin II causes vasoconstriction of arteries and, to some degree, in veins. Blood pressure rises slightly.
Angiotensin II also stimulates Aldosterone release from the adrenal medulla. Aldosterone decreases the production of urine, again raising blood pressure by retaining volume in the vascular system.

Angiotensin II also stimulates the sensation of thirst, increased salt appetite, and anti diuretic hormone secretion.
3. **Vasopressin Mechanism** – with low BP or an increase in plasma concentration the neurohypophysis increases the amount of anti diuretic hormone, or vasopressin secreted. The result is vasoconstriction and a decrease in the rate of urine production.

4. **Atrial Naturetic Mechanism** – Elevated atrial BP results in a release of Atrial Naturetic factor from the atrium of the heart. The substance increases the rate of urine production, decreasing circulatory blood volume, resulting in a lower blood pressure.

<table>
<thead>
<tr>
<th>Fluid Components</th>
<th>Intravascular</th>
<th>Intracellular</th>
<th>Interstitial</th>
</tr>
</thead>
</table>

**Circulation**
- Arteries → arterioles
- Vein → Venules

**Capillaries** – are the place where waste products and nutrients are exchanged in the process called diffusion. Nutrients are pulled from the capillaries and diffuse into the interstitial spaces while waste products are drawn into the capillaries for eventual removal. There are several forces that assist this to happen. Since blood borne proteins tend to be large, they are kept in capillaries, drawing fluid back into the arterial side of the capillaries. This is called Colloid Osmotic Pressure. Pushing fluid into the interstitial (or 3rd space) is hydrostatic pressure (or the force – Blood pressure – exerting pressure from inside the blood vessel itself). The third force exerting influence on fluid balance in capillaries is a slightly negative (in the normal/healthy person) interstitial
pressure. The lymphatic circulation creates this.

Edema is the result of decreased plasma protein concentration (resulting in a lack of “pull” of fluid back into the capillaries). It can also result in an increased capillary permeability, resulting in plasma proteins being lost into the interstitial spaces. This pulls more fluid from the capillaries, increasing edema. Edema can also be a result of an increased BP in the capillaries; Hydrostatic Pressure. This last cause may for example result from Cor Pulmonale or a rapid transfusion of fluid.

*Fluids & Electrolytes*

Body fluids contain dissolved chemicals, divided into 2 separate categories – Electrolytes and Non Electrolytes. Non-Electrolytes are mostly organic compounds like glucose, urea and creatinine. Electrolytes are inorganic compounds (e.g. acids, bases & salts), which have the ability to band together. They are either positively or negatively charged.

Electrolytes have 3 general functions. Many are essential minerals. Secondly, they control movement of water (osmosis) between components (osmosis pulls water from areas of low concentration to areas of high concentration). Finally, they help maintain acid base balance.

The major electrolytes are Sodium (Na⁺), Chloride (Cl⁻), Potassium (K⁺), Calcium (Ca²⁺), Phosphate (H₃PO₄⁻) and Magnesium (Mg²⁺).

Sodium is the most abundant positively charged extra cellular electrolyte (about 90%). Normal levels of sodium in the blood are 136 – 142 mEq/L. It is necessary for the transmission of impulses through nervous and muscle tissue. It also plays a significant role (depending on its concentration) in fluid & electrolyte balance by contributing most of the osmotic pressure. The kidneys regulate the
amount of sodium in the blood stream – excreting excess sodium and conserving it during periods of low concentrations.

High levels of sodium (possibly from large amounts of “Normal Saline” IV fluids, or dehydration) lead to cellular dehydration. Signs and symptoms include thirst, fatigue restlessness agitation and eventually coma states.

Chloride is the most abundant negatively charged extra cellular electrolyte. Normal blood concentration is 95 – 103 mEq/L. It moves easily between extra cellular and intracellular compartments, making it an important component in regulating osmotic pressure between components. It also combines with hydrogen to form hydrochloric acid in the gastric mucosal glands.

The kidneys indirectly regulate chloride, as it tends to follow sodium through a natural bonding process.

Potassium is the most abundant positively charged intra cellular electrolyte. Normal blood concentration of potassium is very narrow, between 3.8 – 5.8 mEq/L. Potassium is the key element in the functioning of nervous and muscle tissue. Abnormal serum levels affect neuromuscular and cardiac function. Intracellularly, it helps maintain fluid volume in cells. The movement of potassium out of cells is replaced by sodium and hydrogen. This shift of hydrogen ions helps to regulate pH.

The regulation of potassium in the kidney is exactly the opposite of sodium. The hormone that regulates both electrolytes is Aldosterone. When Aldosterone is secreted from the adrenal cortex, sodium is retained and potassium is excreted.

Hypokalemia (or low serum levels of potassium) may result from vomiting, diarrhea, high sodium intake, kidney disease or some types of diuretic therapy. Symptoms include cramps, fatigue, flaccid paralysis, nausea, vomiting, mental confusion, increased urine output, shallow respirations and changes in the ECG (lengthening of the QT interval and flattening of the T-wave).
Calcium

98% of calcium is found in the skeleton and teeth, combined with phosphate. The serum calcium is found both extracellularly and intracellularly (in skeletal muscle). It functions in coagulation, neurotransmitter release, neuromuscular conduction, maintenance of muscle tone and excitability of nervous and muscle tissue.

Low levels of Calcium (hypocalcemia) may be due to calcium loss, reduced calcium intake, elevated phosphate levels (as phosphate levels elevate, calcium levels decrease). Symptoms include numbness or tingling of the fingers, hyperactive reflexes, muscle cramps, tetany and convulsions. It may cause laryngeal muscle spasms that can cause death by asphyxiation.

Phosphate

Primarily found combined with calcium in bones. The remaining amount combines with other substances and structures for many purposes. It is a necessary component in forming nucleic acids, and high-energy compounds, as well as substances that serve as buffers (“The phosphate buffer system”).

Magnesium

Magnesium activates enzymes involved in the metabolism of carbohydrates and proteins and triggers a mechanism called the “sodium/potassium pump”. It is also important in neuromuscular activity, neural transmission and myocardial functioning.

Movement of Fluid

Fluid movement between the vascular compartment and interstitial space occurs across capillary membranes. Basically, fluid movement depends of 4 different pressures.

1. Blood hydrostatic pressure
2. Interstitial fluid hydrostatic pressure
3. Blood osmotic pressure
4. Interstitial fluid osmotic pressure

**Blood Hydrostatic Pressure** – is the “blood” pressure within the walls of the capillaries, exerted outward; forcing fluid, plasma and electrolytes into the interstitial spaces.

**Interstitial Fluid Hydrostatic Pressure** – is the pressure exerted outward by the accumulation of fluid in the interstitial spaces and into capillaries and cells.

**Blood Osmotic Pressure** – is the “pulling” pressure the plasma proteins exert on fluid in the interstitial spaces. It pulls fluid back into capillaries.

**Interstitial fluid Osmotic Pressure** – is a “pulling” pressure that electrolytes and other substances exert to pull water into the interstitial spaces.

**Fluid movement between the Interstitial and Intracellular Compartments**

- Under normal circumstances intracellular and interstitial osmotic pressures are the same.
- Is normally controlled with the movement of Na\(^+\) and k\(^+\) into and out of the cell.
- Concentration changes of Na\(^+\) and k\(^+\) can result in fluid imbalance.
- Over hydration of cells is disruptive to nerve cell function.
- Severe over hydration or water intoxication, produces neurological symptoms ranging from disorientation to death.

**Acid Base Balance**

- Acid/Base balance is accomplished by controlling the hydrogen concentration of body fluids.
- Normal extra cellular pH is 7.35 – 7.45.
- Keeping this narrow range possible is essential to survival.

The responsibility for maintaining a normal pH depends on 3 major mechanisms.

1. The Buffer system
2. Respirations
3. Kidney Excretion

**Buffer systems**

- Act as subtle, buffering agents to balance pH using electrolytes, proteins and organic materials to manipulate hydrogen ions.
- Examples are the Carbonic Acid/Bicarbonate Buffer System, the Phosphate Buffer System, the Hemoglobin/Oxyhemoglobin Buffer System, and the Protein Buffer System.

**Respirations**

- An increase in an individual’s respiratory rate, decreases the amount of carbon dioxide concentration in body fluid, and also raises the blood pH (makes it more basic).
- A decreased respiratory rate increases serum CO₂ concentration, decreasing pH (making it more acidic).
- This is regulated by the respiratory center in the medulla, sensing either an increase or decrease in hydrogen ions (accumulating CO₂ leads to a hydrogen ion increase / low CO₂ levels lead to a low hydrogen level).

**Kidney Excretion**

- The kidney can directly excrete hydrogen ions in the urine (distal portion of the nephron).
- Through dissociation, bicarbonate ions and sodium ions can be reabsorbed and transported back into the extracellular fluid; raising body pH.
- If the pH of the body increases, the rate of hydrogen secretion into the nephron decreases. Excess bicarbonate ions are excreted, as a consequence the pH decreases.

**Normal Saline** – an Isotonic Crystalloid Solution

**Actions**
- Increases circulating volume
- Source of Na⁺, Cl⁻ & H₂O

**Indications**
- Head injury
- Dilutes/warms blood
- Main line for blood transfusion
- To restore intravascular volume
- Initial fluid electrolyte replacement in hypovolemia, dehydration (Na⁺ & Cl⁻ depletion) & burns
- Irrigation, cooling burns (external use)
- Dilutent & vehicle for reconstitution, injection or infusion of most drugs
- Ketoacidosis, diabetes, septic shock, fresh water drowning, crush injuries

Adverse Affects
- Large amounts causes volume overload e.g. pulmonary edema or exacerbation of CHF
- Electrolyte dilution and acid/base imbalance following large infusions

Precautions
- Renal impairment
- CHF
- Pulmonary edema
- Room temperature fluid may induce hypothermia with multiple infusions
- Use Buretrol IV tubing with all patients less than 5 years of age

Contains
- 900 mg NaCl/100ml
- pH 5.0
- Na⁺ 154 mmol/L
- Cl⁻ 154 mmol/L

Supplied
- 250 cc bags
- 1000 cc bags
Priming IV Tubing

1. Select the appropriate IV tubing. Inspect the tubing, the roller clamp, the injection ports & the luer lock connector for cracks, use and sterility.

2. Select the appropriate sized IV bag. Inspect for cloudiness, precipitate and cracks/leakage. Check the expiry date, and ensure the fluid is the proper type (Normal Saline or 0.9% saline).

3. Close the roller clamp.

4. Remove the cap on the IV tubing closest to the drip chamber. Ensure sterility! Remove the plastic tab on the IV bag. Connect the two.

5. Squeeze the drip chamber. Fill it ½ full.

6. Open the roller clamp slightly to advance the fluid down the tubing.

7. When the fluid approaches an injection point, invert the port and strike the port, forcing air out and fluid in. Repeat as many times as needed.

8. Close the roller clamp when all the air is expelled from the tubing.

IV Equipment

- Gloves
- Tourniquet
- Alcohol swabs
- IV cannula
- IV tubing
  - Micro drip
  - Macro drip
  - Blood tubing
• Buretrol
  □ Saline lock
  □ Occlusive dressing
  □ Tape
  □ 2 x 2 gauze
  □ Sharps container

*Gloves are an essential part of infection control*

**IV Catheter**

Most needles are made of stainless steel, while the outer cannula is made of Teflon coated plastic to inhibit clot formation. The “sharp”, or inner stainless steel trochar is used for ease of insertion. After signs of the needle being in a vein, the trochar is withdrawn. The plastic cannula is radio opaque, meaning the cannula shows up as a ghost image on x-ray, in case of loss of part of the cannula in the patient’s circulation. Sizes range from 16, 18, 20, 22 and 24 gauge (smaller the number – the larger the diameter).

**IV Tubing**

The infusion tubing drip chamber achieves the maximum flow rate when suspended approximately 3 feet above the IV site. This is due to the force of gravity

- **Micro drip** – a micro drip is an IV set which is designed to precisely deliver small volumes of solution over a long time. E.g. CHF patient with microdrip, (60 drops = 1mL).

- **Macro drip** – is an IV set used to deliver moderate volumes of solution over long periods of time. Because of a larger drop size, the time between drops is longer (than a micro drip), the risk of clot formation of the tip of the IV cannula is greater at TKVO rates. The number of drops, depending on the manufacturer, may be 10, 15 or 20 drop = 1 mL.
- **Blood Tubing** – is an IV set with 2 IV ports, one for a crystalloid to prime and maintain flow after blood administration; the other is a port for the blood product. It also contains a large flexible drip chamber with a screen to filter out clots formed in the blood product.

- **Buretrol** – A Buretrol is a large chamber with a measurement scale printed on the side. IV tubing with a buretrol has a roller clasp between the IV fluid and the Buretrol to deliver a set amount of fluid into the chamber and no more. The buretrol chamber is filled hourly to prevent fluid overload in small children.

- **Saline Lock/Heparin Lock** – These locks are used when a continuous infusion is not required, but occasional infusion of IV medications is desired. This lock consists of a short plastic tube (filled with saline or diluted heparin to prevent clotting) and a multi access later injection point.

- **Occlusive dressing** – an occlusive dressing is applied over the site of the IV insertion to provide a barrier to infection. It may consist of a 2 x 2 gauge, a band-aid or a clear adhesive film.

- **Tape** – the cannula, once in place, requires securing. Hypoallergenic tape (e.g. silk or plastic) should be used to secure the cannula and IV tubing to the patient’s arm/hand/foot.

- **Alcohol Swabs** – should be used to disinfect the potential IV site. Allow a few seconds for the alcohol to dry prior to venipuncture, as isopropyl alcohol is for external use only.

- **2 x 2 gauze** – depending on the site, some “propping” may be required at the cannula site (usually digital or metacarpal IVs). They are also useful for cleaning the site of any blood that may prove disconcerting to the patient.

- **Tourniquet** – The tourniquet is latex band applied around the arm or leg. It takes advantage of venous circulation to mechanically enlarge the vein making cannulation easier.
Remove the tourniquet prior to removal of the stainless steel IV trochar.

- **Sharps Container** – Immediate disposal of the trochar is essential to reduce the risk of accidental needle stick injury to paramedics. Needle stick injuries need to be reported immediately to:

  a) Paramedic Supervisors

  b) The same ER department that the patient has been admitted to

  Treatment and/or testing should occur as soon after the exposure as possible.

**Guidelines for Catheter Size**

# 16 - Adolescents & Adults Only
- Critical Trauma/Burns
- Pt’s requiring large amount of fluids

# 18 - Adolescents & Adults Only
- Fluid resuscitation
- Colloid Infusion

# 20 - Children, Adults & Adolescents
- Most infusions requiring medication, TKVO lines
- Smallest size for colloid infusion

# 22 - Infants, toddlers, children, Adolescents & Adults (especially elderly patients
- TKVO infusions
- Minor medication needs
**IV Stats**

1) Start trying IVs (depending on the severity of the pt’s problem) distally and work your way proximally.

2) Critical patients, requiring rapid infusions need IVs in large veins.

3) For TKVO infusions, be picky about sites. Palpate, inspect for “valves”, crooked veins, or otherwise poor quality veins.

4) Avoid veins around joints for TKVO and potential long-term infusions.

5) Avoid veins in injured arms. Do not start IVs in the same side as radical mastectomies or dialysis fistulas.

6) Elderly patient’s hands can have thin, easy tearing skin. They also can have sclerosed, crooked veins. If so, try IVs above the wrist.

**IV Starts**

1) *Prepare the patient* – explain the need for the IV to the patient, and the brief discomfort that will be encountered. A patient may refuse treatment at any time.

2) *Select the equipment* – Prior to the call starting, prime an IV line with a 250 cc bag of saline to save time. Select the proper sized cannula.

3) *Select a site* – Use proper vasodilation techniques, ensure plenty of light. As best a possible, ensure patient comfort. Choose an appropriate site.

4) *Apply gloves*
5) **Prepare the site** – cleanse a site at least the size of a $2 coin with an alcohol prep. If necessary, shave the arm. Apply a tourniquet.

6) **Inspect the cannula** – If there are any imperfections, discard the needle. Separate the cannula and trochar slightly to ensure smooth movement.

7) **Insert the cannula** – at a 30 - 45° angle and slightly to one side of the vein, insert the needle (bevel up). When the end of the trochar enters the vein, a “pop” should be felt, followed by dark coloured blood filling the cannula “window”

8) **Advancing the cannula** – Decrease the angle of the needle until parallel with the skin. Holding the needle hub securely, advance the plastic cannula over the needle. Alternative, advance the catheter ¼”, then advance the cannula and trochar until the hub meets the insertion site. *If you feel resistance, do NOT force the cannula*

9) **Release the tourniquet**

10) **Apply an occlusive dressing to secure the cannula**

11) **Withdraw the needle** – with firm pressure approximately the length of the catheter proximal to the site (and with the IV tubing close), withdraw the needle. Dispose of the trochar immediately in a sharps container.

12) **Connect the IV tubing to the hub of the catheter** – Open the roller clamp and assess fluid flow in the drip chamber. Check the connections for fluid leakage. Set the flow rate.

13) **Secure the IV tubing (with tape)**
Complications and Troubleshooting IVs

Complications at the IV site

*Extravasation/Interstitial IV* – is an IV whose cannula has come out of the vein. The fluid leaks out of the IV site and into the interstitial spaces. It is identified by a slow IV drip rate, a puffy appearance surrounding the IV site, (due to the accumulation of fluid), pain at the site (as reported by the patient), fluid leaking from the IV site (not the connection of IV tubing & IV cannula).

IVs can also be checked for patency by momentarily lowering the IV container below the level of the IV insertion site. Blood appearing in the IV tubing indicates a patent IV site/cannula. Interstitial IVs need to be removed and re-sited.

*Phlebitis* is an irritation and inflammation of the vein & surrounding structures. They may indicate the presence of an infection. IVs with redness & swelling at site should be re-sited. These symptoms are normally seen in long term IV therapy (> 72 hours).

Systemic Complications

*Septicaemia, Bacteraemia and Septic shock* may occur if signs of local phlebitis are ignored. Watch for large areas of redness involving arms, fever, nausea, vomiting, headache and shock-like states.

*Pulmonary Embolism* – may occur if a blood clot slips from the IV site and follows the circulatory system to the pulmonary artery. Signs of pulmonary embolism include chest pain, blood tinged sputum, shock.

*Air Embolism* – occurs when approx. 10 ml of air inadvertently enters the vascular system, reaches the heart and produces cardiac arrest. Smaller amounts may have serious affects. Paramedics should prevent air from entering the vascular system. Most patients may tolerate small bubbles. To ensure air embolus do not occur, always ensure fluid is in the IV bag, that the drip chamber is ½ full and that all connections are tight.
Catheter Embolus – occurs when a portion of the plastic cannula breaks off and flows into the vascular system. Paramedics should check the removed catheter to ensure the bevelled end remains when discontinuing IVs. Treatment includes immediate identification of this condition and application of a tourniquet proximal to the IV site to trap the broken piece.

Troubleshooting

Dislodged IV/Interstitial IV
- Shut off flow
- Remove/discontinue IV
- Apply pressure to site with a gauze pad
- Document appearance of site, amount of fluid left in bag
- Re-site IV proximal to the affected site

Loose IV tubing connection
- ↓ IV flow rate
- Loosen securing tape
- If completely detached, clean both ends with alcohol wipe and reattach
- Clean site/replace tape/occlusive dressing
- Regulate IV rate

Flow Rate Problem
- Check height of tubing drip chamber
- Check level of fluid in bag. Replace the bag at 150 cc remaining
- Check for signs of infiltration
- Ensure the roller clamp is open
- Check the tube for kinking
- Is the cannula near a joint? Does straightening the joint help?
- Adjust tape, apply on arm board
- Is the catheter too small?
- Venous spasm – is the flow rate problem intermittent? (Cold IV fluids?).
Calculating IV rates

A paramedic receives a Code 4 call for “collapse”. The patient is conscious, alert at the time of your arrival. His chief complaint is syncope. His blood pressure is 80/40. His chest is clear. He weighs 85 kg.

Provincial standing orders state, “In cases of adults with symptomatic hypotension (BP<90mmHg) not due to suspected acute hypovolemia and without signs of pulmonary edema, the paramedic may administer an IV fluid bolus of 250 mL”

How fast should the IV run?

\[
\frac{mL/\text{hr}}{\text{total mL to be given}} = \frac{250\text{ml}}{1\text{ hr (all boluses to be given over 1 hr)}} = 250\text{ml/ hr}
\]

To more precisely regulate the amount of solution, it is advised that the paramedic calculate the number of drops/min.

\[
\frac{\text{Drops/ min}}{\text{drops/mL (see the infusion set packaging) } \times \text{amount of fluid to be infused}} = \frac{15 \text{ drops/mL} \times 250\text{mL}}{60\text{ min}} = 63 \text{ drops/ min}
\]
That number may be further divided into 20 or 15 seconds to save time

\[
\frac{60 \text{ sec}}{15 \text{ sec}} = \frac{63 \text{ drops/min}}{x}
\]

\[60 \times x = 15 \times 63\]

\[x \geq 15 \text{ drops in 15 seconds}\]

**Documentation**

In the procedures section of an ACR, the paramedic should document

- Unsuccessful IV attempts, times and where they were attempted
- Time, location, size of catheter
- Fluid (N/S) and rate
- Amount instilled, amount “to be absorbed” and any urine output enroute
- Any adverse reactions/complications

<table>
<thead>
<tr>
<th>Time</th>
<th>Procedure</th>
<th>Code</th>
<th>Remarks</th>
<th>Initial</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:10</td>
<td>18g Rt antecubital IV 250 mL n/s bolus</td>
<td>50cc N/S TBA</td>
<td>JG</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>
Pre-Test

1. Indicate a catheter for the following scenarios
   - Unconscious diabetic with a blood sugar of 2.6 mmol/L
   - Stable chest pain
   - Unstable patient involved in an MVC (requiring fluid resuscitation)
   - Fractured tibia/fibula (stable v/s ) age 7

2. List 3 reasons for starting an IV

3. Describe the structure of a vein.

4. Describe characteristics of an interstitial IV.

5. An Iv is running at 125 cc/hr. In a macrodrip (15 drops/mL), how many drops/min should you observe?

6. A patient, unstable due to abdominal trauma, requires fluid resuscitation. He weighs 85 kg. His blood pressure is 80/40. His chest is clear.
a) Describe the relevant standing order

b) Assuming a full fluid bolus is required, how much fluid will this patient initially receive?

c) Knowing the amount of fluid to be given, what rate should this bolus be given at?
Glossary

Afferent
Anion
Buretol
Cation
Crystalloid
Colloid
Diastole
DVT
Efferent
Extracellular
Hypertonic
Hypotonic
Intracellular
Ion
Ischeal Tuberosity
Isotonic
Positional (IV)
Thanar Prominance
Vasovagal response
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