

Homeostasis Feedback Systems

For the body to be in health, it must operate within a very narrow range of acceptable values for things such as enzyme function, mucus secretion, blood calcium, somatic reflex arc function, blood glucose, blood oxygen level, heart rate, blood pressure, blood gas levels, gastric secretions, body temperature, water and ion concentrations, and blood pH. The body can regulate its internal environment through a multitude of feedback systems. A feedback system is a cycle of events in which the status of a body condition is continually monitored, evaluated, changed, remonitored, reevaluated, and so on.

Homeostasis is a condition in which the body's internal environment remains within normal physiological ranges. Homeostasis in the human body is continually being disturbed. The disruption may come from the external (outside the body) environment in the form of physical insults such as intense heat or lack of oxygen. Other disruptions originate in the internal (within the body) environment – for example, a blood glucose level that is too low. Homeostasis imbalances may also occur due to psychological stresses in our social environment – the demands of work and school, for example. In most cases the disruption of homeostasis is mild and temporary, and the responses of body cells quickly restore balance in the internal environment. In other cases the disruption of homeostasis may be intense and prolonged, as in poisoning, overexposure to temperature extremes, severe infection, or death of one's spouse. Under these circumstances, regulation of homeostasis may fail.

Fortunately, the body has many regulating systems that bring the internal environment back into balance. Most homeostatic responses of the body are regulated by the nervous and endocrine systems, working separately or together.

The nervous system regulates body activities by sensing deviations from normal physiological limits and then sending messages in the form of nerve impulses, to organs to alter the trend of the deviations.

The endocrine system also monitors body functions and sends messages, in the form of hormones, to regulate homeostasis. Hormonal regulation is achieved more slowly than nervous regulation of homeostasis.

In the body, achieving homeostasis is often the result of a negative feedback loop. The stimulus alters the controlled condition. The receptors monitor changes in the controlled condition resulting from some stimuli. The control center determines the value of the controlled condition. The response of the effectors is to alter body activities to return the controlled condition to within normal limits.

A negative feedback system reverses a change in a controlled condition. Negative feedback systems tend to maintain conditions that require frequent monitoring and adjustment. Examples would include blood pressure, blood glucose levels, and body temperature. A positive feedback system tends to strengthen or reinforce a change in one of the body's controlled conditions. Normal childbirth is a good example of a positive feedback system.

Homeostasis of Enzyme Function

Chemical reactions are fundamental to the existence of living cells. These reactions determine the type and quantity of cellular molecules and are promoted by enzymes, which are typically protein in structure. As such, each cell's appearance and function depends on the activity of its enzymes.

Each cell may have thousands of enzymes that are responsible for normal metabolic processes. Among the more important metabolic processes is the ability to convert different types of fuel molecules, found in food, into energy and thereby fuel cellular processes. The molecule most used by cells for this purpose is glucose, a monosaccharide.

Other types of simple carbohydrates that are present in food and converted to glucose are galactose found in milk, and fructose, found in fruit. Many metabolic processes, including these conversion reactions, occur with normal functioning of liver enzymes. We will use the conversion of galactose to glucose to explain enzyme-mediated metabolic reactions.

If galactose is not converted to glucose, then an adequate amount of glucose may not be available to power cellular functions. Excessive accumulation of galactose in body tissues, a disorder called galactosemia, will cause further health problems.

Galactosemia is an inherited metabolic disorder associated with abnormal liver enzyme function. Enzyme structure is determined by the cell's genetic code. Alterations of the genetic sequence of nucleotides (a gene) may result in inadequate amounts of functioning enzymes.

In the case of galactosemia, the enzyme needed to convert galactose to glucose is defective, resulting in an accumulation of galactose in the body. The homeostatic response that would result from inadequate enzyme function is excessive galactose.

The enzyme reaction that converts galactose to glucose represents only one of nearly 5000 enzyme-mediated metabolic reactions found in liver cells. Understanding the underlying causes of galactosemia emphasizes the fundamental and vital roles that enzymes play in regulating homeostasis.

Homeostasis of Mucus Secretion

Although the stimulus for the production of mucus, a normal secretory cell function, is unknown, mucus plays several positive roles in maintaining good health. In particular, mucus acts as a lubricant for the linings of the digestive and respiratory tracts.

Glandular epithelial cells form the secretory portions of glands. The secretion of mucus by exocrine glands is made possible by transporter proteins, which are coded by sequences of DNA (genes) and assembled by ribosomes and the golgi complex.

By means of transporter proteins, frequently called “transporters,” ions move across the plasma membrane of a cell into the extracellular fluid. Once the ions, and other solutes, are moved into the extracellular fluid, their concentration increases the extracellular fluid’s osmotic pressure. This decreases the relative concentration of extracellular water. Water then follows the solutes out of the cell, via osmosis, to form the watery secretion we call ‘mucus’.

Cells synthesize many chemicals to promote homeostasis. These chemicals are produced or modified by enzymes which, in turn, are created from information in the DNA templates, or genes. Small changes in the genetic code, and therefore enzymes, may cause significant homeostatic imbalances.

Cystic Fibrosis (mucoviscidosis) is a recessively inherited disorder (both parents must contribute its gene to the afflicted offspring) of the exocrine glands, resulting in the excessive buildup of thick mucus. Because it affects exocrine glands, which are widely distributed, cystic fibrosis is a multi-organ disease. It is the most common lethal genetic disease (about 1 in 2000 births), and typically results in death by pulmonary failure.

When the gene that codes for the Cl^- transporters is defective, the resulting defective transporter protein is not inserted into the plasma membrane of the epithelial cell in the exocrine gland.

Cystic fibrosis is only one example of the serious effects of mucus imbalance, but it illustrates the crucial role of proteins in maintaining the balance of fluids and ions transported across the plasma membrane and thus, normal secretory cell function.

Homeostasis of Blood Ca²⁺

The body's regulation of blood Ca²⁺ levels is crucial to maintaining good health. It's very important to many functions of the body including muscle contraction, neurotransmitter release, and blood clotting.

Muscle exertion and pregnancy are two examples of a stimulus that decreases the blood Ca²⁺ levels. (controlled condition)

The receptors in the parathyroid gland cells send message to control center. The PTH (parathyroid hormone) gene is turned on and the output is increased PTH.

The effectors are:

1. Osteoclasts increase bone resorption.
2. Kidney increases Ca²⁺ retention and increased calcitrol production.

The response is increased blood Ca²⁺.

Regulation of blood calcium is very important to many functions of the body, including muscle contraction, neurotransmitter release, and blood clotting.

Abnormal blood calcium levels may result in a homeostatic disorder.

Osteoporosis is a condition of porous bones characterized by decreased bone mass and increased susceptibility to fracture. There are several causes of osteoporosis, but one of the primary causes results from excessive production of PTH (parathyroid hormone), which regulates blood calcium (Ca²⁺) levels.

One risk factor, female menopause, demonstrates a blood calcium imbalance. In females, normal levels of estrogen inhibit PTH production. Lowered estrogen levels, which occur with the onset of menopause, may allow excessive PTH production. Blood calcium imbalances (both high and low) have a wide range of effects. We have examined only two of these to help your understanding of the role of Ca²⁺ levels in essential physiological processes such as bone and teeth formation, blood clotting, neurotransmitter release, maintenance of muscle tone, and excitability of nervous and muscle tissue. Regulating Ca²⁺ levels is a crucial part of maintaining homeostasis.

Energy Sources for Muscle Contraction

1. ATP already in the cell (not much).
2. ATP from cellular respiration – requires oxygen – an aerobic process – too slow for sustained contraction, but continues.
Occurs in the mitochondria.
1 glucose + oxygen yields 36 ATP + Carbon dioxide + water.
3. ATP from creatine phosphate – donates PO_4 to convert ADP to ATP.
Good for only a few seconds. Phosphate donor from skeletal muscles.
4. ATP from Lactic Acid Fermentation
Anaerobic process that kicks in when insufficient oxygen is in the cell to carry out the aerobic process.
Instead of 36-38 ATP's per glucose, yields only 2 ATP.

Breakdown of muscle glycogen into glucose
and the production of 2 peruvic acid from glucose via glycolysis

yields 2 ATP and lactic acid.

But, fibers also contain myoglobin in red muscle fibers to store oxygen and glycogen to store glucose.

Problems with Lactic Acid Fermentation:

- Lowers pH of cell to the point that enzymes are no longer functional and contraction stops – muscle fatigue.
- Oxygen debt. The amount of oxygen required to recycle all lactic acid in the cell – 1/5 changed to carbon dioxide and excreted; 4/5 changed to glycogen and recycled.

Sources of ATP production, in summary:

1. What's already there
2. Creatine phosphate
3. Cellular respiration
4. Lactic Acid Fermentation

The Muscle Contraction Cycle

Sarcomeres shorten through repeated cycles during which the myosin heads (cross-bridges) attach to actin, rotate, and detach.

During the power stroke of contraction, myosin heads rotate and move the thin filaments past the thick filaments in a wratcheting motion toward the center of the sarcomere.

1. Myosin heads hydrolyze ATP and become reoriented and energized.
2. Myosin heads bind to actin, forming cross-bridges.
3. Myosin heads rotate toward center of sarcomere (power stroke)
4. As myosin heads bind ATP, the cross-bridges detach from actin.

Contraction cycle continues if ATP is available and Ca^{2+} level in the sarcoplasm is high.

Four stages of cross-bridge cycle:

1. Cross-bridge binds to actin.
2. Cross-bridge moves
3. ATP binds to myosin causing cross-bridge to detach.
4. Hydrolysis of ATP energizes cross-bridge.

Why rigor mortis when we die?

Ca^{2+} ions leak out of the SR and allow the myosin heads to bind to actin.
But, ATP production has ceased, so the cross-bridges cannot detach from actin.
Therefore, muscles are in a state of rigidity (rigor mortis).

Calcium's Role in Muscle Contraction

Ca²⁺'s role in the regulation of contraction by troponin and tropomyosin.:

- (a) During relaxation, the level of Ca²⁺ in the sarcoplasm is low because calcium ions are pumped into the sarcoplasmic reticulum (SR) by Ca²⁺ active transport pumps.
- (b) A muscle action potential propagating along a transverse tubule opens Ca²⁺ release channels in the SR, calcium ions flood into the cytosol, and contraction begins.

An increase in the Ca²⁺ level in the sarcoplasm starts the sliding of thin filaments; When the level of Ca²⁺ in the sarcoplasm declines, sliding stops.

Therefore, the sarcoplasmic reticulum (SR) releases Ca²⁺ ions to trigger muscle contraction.

Relaxation:

Troponin holds tropomyosin in position to block myosin-binding sites on actin.

Contraction:

Ca²⁺ binds to troponin, which changes the shape of the troponin-tropomyosin complex and uncovers the myosin-binding sites on actin.

Actin binds to active sites when stimulus is switched on.

Physical contact only takes place when sites are uncovered

Sodium / Potassium Pump

Sodium ions are expelled and potassium ions are imported through the sodium pump. (Na^+ / K^+ ATPase).

The pump will not work unless Na^+ and ATP are present in the cytosol and K^+ is present in the extra-cellular fluid.

Na^+ in the cytosol binds to the pump protein.
(Na^+ concentration is high in the ECF and low in the cytosol, whereas K^+ concentration is high in the cytosol and low in the ECF.)

Na^+ binding triggers the breakdown of ATP into ADP and the attachment of a high-energy phosphate group (P) to the pump protein.

This action changes the shape of the pump protein so that the Na^+ is pushed through the membrane and expelled into the ECF.

The new shape of the pump favors binding of K^+ in the ECF.
 K^+ binding triggers release of the phosphate group from the pump protein, which again causes the shape of the pump protein to change.

As the pump protein returns to its original shape, K^+ is pushed through the membrane into the cytosol.

At this point, the pump is ready to bind Na^+ in the cytosol and the cycle repeats again.

How Information is Carried Along a Neuron in the Body

Information transmission along a neuron in the body involves reversal of polarity, shifting of Na^+ and K^+ , action potential, Na^+ permeability and K^+ permeability.

Inter-neurons are triggered by sensory input.

(Motor neurons are not triggered by sensory input; information passes on to interneuron.)

A transmitter substance such as acetylcholine or noradrenalin diffuses to the receptor sites.

Na^+ rushes in to the cell with a change to the permeability of the responding cell.

The neuro-transmitter bonds with the receptor and causes ion channels to open.

Vesicles containing transmitter substance dump the transmitter into the synaptic cleft.

Receptors in the cell membrane of post-synaptic cells receive the transmitter substance.

Na^+ rushes in; K^+ rushes out of the cell.

The electrical gradient is positive on the outside relative to the inside.

The gated channel opens or closes in response to chemical or electrical changes inside or outside the cell. When the gate is open, ions can diffuse through the channel.

Depolarization goes in a wave along the neuron. i.e., the message is carried all the way to the end of the nerve cell. It's all or none.

Active transport pumps Na^+ back out of the cell.

The impulse is the point at which the polarity is reversed.

What the message means is interpreted at the end of the path.

If the neurons are myelinated, it goes faster because the message jumps from Schwann cell to Schwann cell by going from a Node of Ranvier to the next Node of Ranvier.

Therefore, the myelinated sheath allows the message to go faster because it doesn't have to repolarize as often.

Activity at Neuro-Muscular Junction

Acetylcholine released at the neuromuscular junction triggers a muscle Action Potential which leads to muscle contraction.

1. Nerve impulse arrives at axon terminal of motor neuron and triggers release of acetylcholine (ACh).
2. ACh diffuses across synaptic cleft, binds to its receptors in the motor end plate, and triggers a muscle Action Potential (AP).
3. Acetylcholinesterase in synaptic cleft destroys ACh so another muscle Action Potential does not arise unless more ACh is released from motor neuron.

(ACh agonist = nicotine. ACh antagonist = curare.)
4. Muscle AP travelling along T-tubule opens Ca^{2+} release channels in the sarcoplasmic reticulum (SR) membrane, which allows calcium ions to flood into the sarcoplasm.
5. Ca^{2+} binds to troponin on the thin filament, exposing the binding sites for myosin.
If a threshold number of receptor sites are filled, then the muscle responds.
6. Contraction: power strokes use ATP;
Myosin heads bind to actin, swivel, and release;
Thin filaments are pulled toward center of sarcomere in wracheting movement.
7. Ca^{2+} release channels in SR close and Ca^{2+} active transport pumps use ATP to restore low level of Ca ions in sarcoplasm.
8. Troponin-tropomyosin complex slides back into position where it blocks the myosin binding sites on actin.
9. Muscle relaxes.

Somatic Reflex Arc Function

The nervous system pathway that rapidly and predictably responds to changes in the internal and external environment is known as a reflex arc. Autonomic reflex arcs that control smooth and cardiac muscles as well as endocrine and exocrine glands are very important to normal body functions.

You are probably most aware of the somatic reflex arcs that control contractions of skeletal muscles. Somatic reflex arcs provide very rapid adjustments to body movements.

In most cases, these reflexes also act to prevent injury.

This type of neural pathway, called reciprocal innervation, that contracts one muscle and relaxes the opposing muscle, allows the reflex to respond very rapidly to abrupt changes in muscle tension.

Several somatic reflexes can be used to assess neurological damage. Physicians often use the patellar reflex or knee jerk as one method of evaluating a patient's neurological condition.

In this examination, the physician lightly strikes the patellar ligament with a specialized rubber-tipped instrument shaped somewhat like a hammer. This action of instrument against patellar ligament temporarily overstretches the quadriceps muscle group. Increased motor impulses cause the quadriceps muscles to contract and extend the leg. The same applied stimulus (hammer strike) causes the hamstring muscles to relax and allow rapid extension of the leg in a normal knee jerk response.

The patellar reflex is affected when damage occurs to (1) the motor or sensory neurons that supply the quadriceps, or (2) the lumbar region of the spinal cord. Damage would cause an absence of control center output to the prime mover muscle.

Unfortunately, if the damage that affects the neurons of the patellar reflex, or any other nervous pathway, is permanent then effective treatment may not exist. Studies aimed at regrowing both nerves and the spinal cord are underway and provide the most hope for victims of spinal cord injury.

Homeostasis of Blood Glucose

Cells use glucose (sugar) molecules as their primary energy source. Maintaining normal blood glucose levels is one of the most important regulatory processes in the body. Many mechanisms, in both the endocrine and nervous systems, can alter blood glucose levels to bring the body back to homeostasis.

A critical mechanism for regulating blood sugar involves the secretions of the pancreatic hormones, insulin and glucagon. These hormones operate in an antagonistic manner: to reach a normal blood sugar level, one hormone acts to lower the blood sugar level while the other hormone acts to raise it.

Exercising is an example of a stimulus that would result in a decreased blood glucose level (controlled condition). Receptors in the pancreatic cells send a message to the control center (glucagon gene in alpha cells). The output is increased glucagon production which affects the liver. The increased glucagon acts on liver cells to convert glycogen to glucose, thereby increasing blood glucose levels.

This new stimulus affects the controlled condition (increased blood glucose) which is detected by the receptors in pancreatic cells. They send a message to the control center insulin gene in beta cells to cause an increased output of insulin.

The effectors are:

Liver

Increased conversion of blood glucose to glycogen.

Body Cells

Accelerated uptake of glucose and amino acids into cell and increased protein synthesis.

Fat Cells

Increased conversion of blood glucose to fat.

The response is decreased blood glucose. The levels of insulin and glucagon change frequently to provide nearly constant regulation of blood glucose levels.

Both low levels of blood glucose (hypoglycemia) and elevated levels of blood glucose (hyperglycemia) can have adverse effects on many structures of the body.

Diabetes Mellitus

Diabetes mellitus comprises a group of disorders caused by the inability of cells to transport glucose into their interior. As a result of this inability, the unused glucose accumulates in the blood resulting in high blood sugar (hyperglycemia). Without glucose, the body turns to other fuel sources, such as proteins (amino acids) and lipids (fatty acids) for cellular metabolism.

These disturbances in carbohydrate, protein, and fat metabolism are characteristic of the two major forms of diabetes mellitus, type I and type II. Medical researchers believe that both types of diabetes are caused by interactions between genetic and environmental factors.

Type I diabetes is caused by a specific homeostatic imbalance. There is a deficient insulin output which affects the body cells. The response is Type I diabetes mellitus. Type I diabetes mellitus is also called insulin-dependent diabetes mellitus and is caused by a deficiency of insulin. Medical researchers believe that it is an autoimmune disease that attacks the beta cells of genetically predisposed individuals.

Type II diabetes, also called non-insulin-dependent diabetes mellitus, is the most common form of diabetes. Obesity is the major environmental risk factor that increases an individual's chance of contracting type II diabetes mellitus.

The metabolism of an individual with type I diabetes is similar to that of a starving person. The primary clinical manifestations of type I diabetes are:

- Excessive thirst (polydipsia),
- Excessive urination (polyuria), and
- Excessive hunger (polyphagia).

Elevated blood glucose causes water to be osmotically transported out of all body cells including hypothalamic cells. Stimulation of the hypothalamus triggers increased thirst. This situation causes water to be osmotically transported into the kidney filtrate resulting in high urine output. Excessive levels of glucose overwhelm the ability of the kidney to retain glucose in blood thereby causing high levels of glucose in the urine. Cellular starvation triggers an increase in hunger – inadequate carbohydrate, fat and protein energy sources.

The most common treatment for insulin-dependent diabetes mellitus (Type I) is the administration of insulin. The injection of insulin brings the insulin levels in the blood back to normal thereby promoting a return to homeostasis of blood glucose and, hopefully, leading to cessation of clinical symptoms.

Homeostasis of Blood Oxygen Levels

The major function of red blood cells (erythrocytes or RBCs) is to transport oxygen throughout the body. Oxygen chemically binds to hemoglobin molecules and is transported by the red blood cells. Both the number of red blood cells and the concentration of hemoglobin within them determine the blood oxygen level.

Cells use oxygen during the process of harvesting energy from fuel molecules, such as glucose. Not having enough oxygen adversely affects cell metabolism and can result in cell damage or death.

When the oxygen carrying capacity of the blood drops because red blood cell production (erythropoiesis) is not adequate, low blood oxygen triggers a negative feedback mechanism. This mechanism, regulated by the hormone erythropoietin, increases red blood cell production to bring the body back into homeostasis.

Low blood oxygen is an example of a controlled condition that would promote production of erythropoietin by kidney cells. The control center red bone marrow causes an output of increased immature RBCs. The effector is red blood cells – increased RBCs in circulation. The response is increased blood oxygen. Increased numbers of RBCs will elevate blood oxygen levels.

If the homeostasis of blood oxygen is disrupted and blood levels are reduced, a condition called hypoxia occurs.

Anemia is a condition in which the oxygen-carrying capacity of blood is reduced. Several kinds of anemia exist, all characterized by reduced RBC count, reduced hemoglobin, or abnormal hemoglobin.

Hemorrhagic, pernicious, and aplastic anemia result from inadequate red blood cell count.

Hemorrhagic anemia is caused by abnormally low RBC count. The response is decreased blood oxygen. Hemorrhagic anemia is caused by blood loss due to wounds, ulcers, or heavy menstrual bleeding.

Pernicious anemia is caused by low hemopoiesis, which is caused by the lack of intrinsic factor, a molecule needed for the absorption of vitamin B₁₂. Adequate vitamin B₁₂ levels are needed to make red blood cells.

Aplastic anemia can occur following the destruction of red bone marrow from a tumor, toxins in the body, radiation, and some medicines.

Iron deficiency, thalassemia, and sickle-cell anemia result from inadequate or improper hemoglobin production.

The effector is the red blood cell with abnormally low hemoglobin in RBCs. The response is decreased blood oxygen.

Iron deficiency anemia is caused by inadequate absorption or loss of iron. This is probably the most common type of anemia, particularly in children.

Thalassemia anemia is a hereditarily determined inability to produce hemoglobin. There are several forms of thalassemia that differ, depending on the number of defective genes and form of inheritance. Symptoms of thalassemia can vary from mild to severe. Severe forms can cause significant cardiovascular burden resulting in congestive heart failure. The effector is the red blood cell. Abnormally formed hemoglobin in RBCs. The response is decreased blood oxygen.

Sickle-cell anemia is a hereditarily determined inability to produce normal hemoglobin molecules. The shape of the hemoglobin molecule distorts the erythrocyte into the sickle-shaped cell that gives the disorder its name. These sickled cells rupture easily thereby reducing blood carrying capacity. Furthermore, sickled cells tend to get stuck in small blood vessels and further reduce blood supply.

Regardless of the cause and type of anemia, the condition usually results in the symptoms of fatigue and intolerance to cold. Both of these symptoms are the result of decreased cellular metabolism due to low oxygen levels in the body. This decreased cellular metabolism provides less energy for muscle movement and heat generation.

Homeostasis of Heart Rate

The heart provides pressure to circulate blood so that all of the cells in the body receive adequate oxygen and nutrients. Cardiac output is the amount of blood that is ejected from the left ventricle of the heart each minute. Factors that determine cardiac output include the heart rate (the number of beats per minute) and stroke volume (the volume of blood ejected per beat).

These primary factors regulate stroke volume:

1. the degree of stretch of the heart in response to venous return prior to contraction,
2. the forcefulness of the contraction due to sympathetic stimulation; and
3. the pressure of the blood resisting ventricular ejection.

Heart rate is regulated primarily by sympathetic stimulation and adrenal medulla hormones such as epinephrine and norepinephrine. The following explores the role of the interplay between the sympathetic and parasympathetic outputs in regulating cardiac output:

Under certain physiological conditions, cardiac output is inadequate to supply the oxygen needed by the cells. Increased physical activity is an example for which elevated cardiac output would be necessary.

Normal heart rate and stroke volume is a controlled condition with receptors in the baroreceptor, Proprioceptor. The control center in the medulla oblongata increases impulses and the output is increased sympathetic impulses. The effectors in the heart are increased heart rate and increased contraction force. The response is increased cardiac output, and increased contraction force results in greater stroke volume.

As long as the cells, especially skeletal muscle cells, require an increased cardiac output to provide them with adequate oxygen, the heart rate and stroke volume will remain elevated. However, once the muscles no longer need the greater level of oxygen, their lower level of activity promotes greater parasympathetic stimulation. These increased parasympathetic impulses inhibit heart rate and thereby reduce cardiac output.

Coronary heart disease (CAD) is a condition in which the coronary arteries are narrowed. As a result, the heart muscle does not receive adequate blood or oxygen, and become damaged. Throughout the industrialized world, heart disease is the number one cause of premature death.

Two common forms of heart disease, myocardial infarction (heart attack) and congestive heart failure (CHF) occur as a result of inadequate blood supply to the myocardium. In a heart attack, heart muscle cells actually die and are replaced by scar tissue. CHF occurs when the weakened heart muscle can no longer adequately pump blood.

Risk factors for heart disease are high cholesterol level, high blood pressure, smoking, and obesity. These factors affect the controlled condition of inadequate cardiac output.

Receptors are baroreceptor, Proprioceptor. The control center is the medulla oblongata. The effector is decreased contraction force.

Congestive heart failure is an example of a positive feedback loop. As a result of the damaged heart muscle, blood tends to pool in the atria which increases stretching of the myocardium. At first, increased stretching elevates stroke volume, but eventually the heart muscle becomes overstretched and contracts less forcefully. Less forceful contractions result in less blood flow through the coronary vessels which further weakens the myocardium.

This cycle of the heart becoming an increasingly ineffective pump continues until the heart can no longer move blood. At that point, blood pools in the lungs causing pulmonary edema or in the extremities causing peripheral edema.

Homeostasis of Blood Pressure

Providing adequate circulation of blood and the substances suspended and dissolved in it is the major function of the cardiovascular system. Blood flows down a blood pressure gradient produced during cardiac muscle contractions.

Normal blood pressure is a primary factor in moving blood. It is crucial to the normal functioning of every organ in the body. Both the endocrine and the nervous systems provide mechanisms of achieving blood pressure homeostasis. Hormonally controlled mechanisms function more slowly than rapid nervous system mechanisms.

Blood pressure and flow are regulated by an autonomic reflex arc, one of the nervous system mechanisms.

Blood loss is an example of a stimulus that would temporarily cause a drop in blood pressure. Another example is beginning to exercise:

The controlled condition is decreased blood pressure. Receptors are baroreceptors in aorta and carotid artery. The control center is the medulla oblongata whose output is increased sympathetic nerve impulses, increased secretion of epinephrine and norepinephrine. The effectors are:

Heart
Increased heart rate.
Increased heart stroke volume.

Vessels
Increased vasoconstriction.

Response
Increased cardiac output will
Elevate blood pressure.

Response
Increased vascular resistance will
Elevate blood pressure.

The proper regulation of blood pressure guarantees the delivery of adequate oxygen and nutrients to meet the metabolic needs of all of the body's cells. If blood pressure becomes abnormally low (i.e. in the case of shock), then cellular metabolism becomes dysfunctional and, if this condition persists, may result in death.

In industrialized nations, hypertension, or persistent elevated blood pressure, is one of the most common disorders affecting the heart and blood vessels.

The cause of most cases of hypertension cannot be directly related to any one identifiable factor. However, several risk factors including diet, stress, and an individual's genetic makeup or heredity, predispose a person to hypertension.

Examples of stimuli that may result in hypertension are stress, genetic disposition, high sodium diet, and obesity.

The controlled condition is excessive blood pressure. Receptors are baroreceptors in the aorta and carotid artery. The control center is the medulla oblongata when output is increased sympathetic nerve impulses, increased secretion of epinephrine and norepinephrine.

The heart is the organ most commonly affected by hypertension. In an individual with hypertension, the heart must work harder than normal to move the blood against the elevated blood pressure to supply all of the organs with adequate levels of oxygen. This overwork may result in inadequate perfusion of oxygen in the heart, potentially resulting in cardiac ischemia (angina pectoris) or even cardiac infarction (heart attack).

In the case of prolonged hypertension, brain arteries may burst causing a stroke and eventual brain damage. In the kidneys, persistent high blood pressure thickens the arterioles thereby reducing blood flow to the kidney cells and resulting in kidney damage.

The effectors are:

Heart

Vessels

The response is:

Increased cardiac output

Increased vasoconstriction

Hypertension is sometimes known as the “silent killer” because it adversely affects the heart, kidney and brain before the person becomes aware of the damage.

Homeostasis of Blood Gas Levels

Maintaining proper levels of oxygen and carbon dioxide in the blood is crucial to establishing each cell's normal metabolic processes. An important process occurs at the alveoli of the lungs: oxygen is introduced into the blood and carbon dioxide is removed from the blood.

The cells use oxygen to harvest chemical energy from fuel molecules. Carbon dioxide is a byproduct of this process. Increasing carbon dioxide levels promotes the binding of carbon dioxide with water to form carbonic acid which, in turn, then gives off a hydrogen ion. Therefore, increased carbon dioxide levels lead to increased acidity (a lower pH value) in the blood.

Blood levels of oxygen and carbon dioxide gases are regulated through an autonomic reflex arc that alters respiration rate. An increase in respiration rate will increase oxygen levels while decreasing carbon dioxide levels in the blood.

The regulation of blood gases is a vital process for maintaining normal health. Inadequate oxygen (hypoxia) in the brain may result in fainting and the depression of the control center in the medulla oblongata. If this process is not interrupted, it may trigger a positive feedback loop causing even greater brain damage. Excessive changes in carbon dioxide will alter blood pH and can potentially cause malfunction in many organs.

Respiratory failure is caused by the inability of the cardiovascular system to provide adequate exchange of oxygen and carbon dioxide in the lungs. This condition occurs when there is too little arterial oxygen or too much arterial carbon dioxide.

Chronic respiratory failure is characterized by respiratory acidosis, a condition in which an individual's blood levels become acidic. Abnormally high CO₂ would result in respiratory acidosis.

Treatment for respiratory failure includes correcting initial cause (i.e. removing pulmonary obstructions) and administering oxygen. Treatment of respiratory acidosis may include intravenous administration of sodium bicarbonate to buffer and raise pH to normal levels, bringing the body back to homeostasis.

Homeostasis of Gastric Secretions

The digestion and absorption of food plays an important role in maintaining normal health. The stomach and intestines use the movements of smooth muscle as well as the secretion of digestive juices, including acid, to break down food into smaller molecules that can be absorbed into the blood. The absorbed food molecules will be used as energy sources and to build other molecules needed for various cellular functions.

Smooth muscle movements are regulated by nervous system stretch reflexes. These reflexes measure the increased amount of stretch in the stomach walls, resulting from introduced food, and respond by stimulating greater stomach motility which aids digestion.

The secretion of gastric juices, particularly hydrochloric acid, is regulated by a negative feedback loop that monitors the level of acid secreted by the parietal cells of the stomach. Ingested proteins act as buffers and tend to reduce the level of acidity in the stomach.

Ingested proteins and higher pH would result in increased nerve impulses from the gastric chemoreceptors to the control center. This causes the parietal cells in the stomach to increase HCl (hydrochloric acid) production. The response is increased gastric acidity. This response increases gastric acid to promote digestion and returns pH to the normal levels that were present before eating.

Excessive production of gastric acid (HCl), bacterial infection, and anti-inflammatory drugs (NSAIDs) such as aspirin are the three main causes of Peptic Ulcer Disease (PUD).

Peptic Ulcer Disease (PUD), a common digestive ailment, occurs in 5% - 10% of adults. Peptic ulcers, crater-like lesions in the digestive mucosal membrane, occur where the digestive tract is exposed to gastric acid.

When an individual has PUD, the protective functions of the digestive mucosa (bicarbonate buffers from the pancreas, gastric mucus production, and epithelial tissue regeneration) are overwhelmed and cannot function normally. As a result, the ulcerous condition persists and cannot be repaired.

Stress, caffeine, alcohol, smoking, and NSAIDs are all stimuli that may result in excessive production of gastric acid.

PUD is medically treated through the prescription of certain drugs, such as Pepcid™, Zantac™, and Tagamet™, that slow the production of gastric acid by inhibiting hydrogen ion pumps.

Homeostasis of Body Temperature

Maintaining a normal body temperature is a vital homeostatic process. The body produces heat by expending chemical energy during normal cellular metabolism.

When the balance between the physiological factors that increase heat production and those that contribute to heat loss is altered, changes in body temperature follow. Thermoreceptors in the hypothalamus and skin monitor body temperature.

Impulses from the hypothalamus control outer trigger autonomic reflexes that act to alter body temperature when it goes out of the normal range. Under abnormal body temperatures, the hypothalamus also secretes thyroid-releasing hormone (TRH) which promotes thyroid gland secretion.

Decreased temperature is a stimulus that will increase the frequency of nervous impulses from the thermoreceptors to the control center.

Extremes in both high and low temperatures can be fatal. Body temperatures that are too low can affect heart activity and cause rhythm aberrations. Next, we will explore the homeostatic imbalances that occur when the body temperature rises above normal values:

When the body is unable to cool itself adequately, heat cramps, heat exhaustion, and heat stroke can ensue.

Extreme increases in body temperature, like extreme changes in pH, can alter protein structures. Changes in protein structures, especially enzymes, render them nonfunctional and threatens the body's homeostasis.

The body's response to increasing body temperature is the opposite of its response to decreasing body temperature. Decreased nerve impulses and less TRH is output from the hypothalamus.

In addition to these heat-reducing mechanisms, cutaneous sweat glands are activated to promote perspiration, an evaporative cooling mechanism.

These are two conditions that would result in a temperature imbalance.

Temperature imbalances occur following the loss of fluids and electrolytes causing multi-organ disturbances to ensue. However, treatment for heat exhaustion and cramps is simple. Replacing water and electrolytes and rest allows the patient to regain normal homeostatic cooling ability.

Because normal cooling mechanisms do not work for victims of heat stroke, the patient must be externally cooled by immersion in cool water to return the body to normal body temperature values.

Homeostasis of Water and Ion Concentrations

Maintaining the body's water volume is crucial to normal cell and organ functions. Inadequate blood volume, associated with decreased water volume, results in diminished blood pressure. Low blood pressure reduces the flow of blood and therefore impacts the distribution of blood components including cells, nutrients, and gases.

The amount of water in blood is affected by its osmotic pressure. Blood osmotic pressure increases when greater concentrations of dissolved substances, particularly electrolytes, and/or lesser amounts of water are found in the blood.

Under higher blood osmotic pressure levels, antidiuretic hormone (ADH) is released from the posterior pituitary gland. The increased concentrations of ADH enhances water reabsorption (retention) by the principal cells of the nephron of the kidney.

Maintaining the proper levels of ADH is instrumental in directly regulating blood osmotic pressure. Furthermore, blood osmotic pressure indirectly affects blood volume, blood pressure, and blood flow. As a result, blood osmotic pressure imbalances can have adverse effects on multiple organs.

Diabetes insipidus (DI) is characterized by excessive dilute urine production. There are two main forms of DI. Central DI is caused by inadequate production of ADH. Nephrogenic DI, often the result of kidney damage or certain drugs, is caused by an insensitivity of the principal cells in the nephron to ADH.

Medical treatment of diabetes insipidus involves taking a modified form of ADH which returns the blood osmotic pressure to normal homeostatic values. Nephrogenic DI is usually the result of damage to the kidney cell. When kidney cells are damaged, they do not respond to ADH replacement therapy. In those cases, dialysis or kidney transplant may be the only forms of treatment.

Homeostasis of Blood pH

Careful homeostatic regulation of the level of hydrogen ions (pH) in the blood is a critical factor in the acid-base balance of body fluids. Some hydrogen ions enter the body as good, but most hydrogen ions are produced as a result of cellular metabolism.

The body has three major ways to maintain pH: chemical buffers, urinary system mechanisms and respiratory mechanisms. Chemical buffers, such as bicarbonate ions, act quickly to prevent significant changes in hydrogen ion levels by binding with excess hydrogen ions to remove them from solution. The urinary system acts slowly to remove hydrogen ions by secreting the ions into urine which then leaves the body.

Increasing amounts of carbon dioxide cause the formation of more carbonic acid which, in turn, will release H^+ ions and thereby acidify the blood. Respiration rate is increased, removing excess carbon dioxide.

Increased exercise is an example of a stimulus that would promote an increase in nerve impulses from the chemoreceptors monitoring blood pH levels.

Maintaining the correct pH is critical because even small changes in hydrogen ion concentrations may change the three-dimensional shape of body proteins. Any changes in a protein's structure will adversely affect that protein's function. This is particularly important to many enzymes that control cellular reactions in the body. Without mechanisms to rid the body of excess hydrogen ions, death would soon result.

Normal blood pH values range from 7.35 to 7.45. When the blood pH is lower than 7.35, a condition called acidosis occurs. When the blood pH is higher than 7.45, a condition called alkalosis occurs. The body must compensate for these changes in blood pH in order to maintain normal health.

If a pH imbalance caused by compromised respiratory function (respiratory acidosis or alkalosis) occurs, then the urinary system must compensate for that imbalance. If the pH is altered as a result of a metabolic problem (metabolic acidosis or alkalosis), then the respiratory system must compensate to maintain normal pH values.

Damage to diaphragm or airways, lung disorder (i.e. emphysema) or depression of respiratory center may result in respiratory acidosis.

Diarrhea (loss of bicarbonate ions), renal dysfunction or ketoacidosis (diabetes mellitus) may result in metabolic alkalosis.

Treatments for acidosis, acidic pH imbalances, have to be rapid because very low pH levels (below 7.0) can cause severe depression of the central nervous system which may result in coma or death. Severe alkalosis, alkaline pH imbalances, tends to cause overexcitability of the central nervous system which may result in muscle spasms, convulsions and death.