

unit guide

HUMAN PHYSIOLOGY (Systems)

SHB – 2 – 532

Faculty of Engineering, Science and
the Built Environment

2008 / 2009

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1.0 UNIT DETAILS

Unit Title:	HUMAN PHYSIOLOGY (Systems)
Unit Level:	2
Unit Reference Number:	SHB-2-532
Credit Value:	1
Student Study Hours:	150
Contact Hours:	42
Private Study Hours:	108
Pre-requisite Learning (If applicable):	
Co-requisite Units (If applicable):	Principles of Physiology
Course(s):	BSc Sport & Exercise Science, BSc Bioscience and Nutrition schemes
Year and Semester	Year 2 Semester 1
Unit Coordinator:	Mike Hibbs
UC Contact Details (Tel, Email, Room)	hibbsm@lsbu.ac.uk , J214, ext. 7962
Teaching Team & Contact Details (If applicable):	
Subject Area:	Human & Exercise Science
Summary of Assessment Method:	examination (60% of marks) and coursework (40% of marks)

2.0 SHORT DESCRIPTION

There are numerous physiological systems which act to maintain normal body function. Based on your knowledge of the principles of human function, you will study three of these systems in more detail and investigate how they interact with each other.

3.0 AIMS OF THE UNIT

The unit aims to:

- Provide an understanding of human systemic physiology within the framework of homeostasis.
- Characterise normal and some abnormal physiological processes.

4.0 LEARNING OUTCOMES

4.1 Knowledge and Understanding

1. Describe physiological function of the circulatory, respiratory and renal systems in the context of homeostasis.
2. Give examples of integration between systems.
3. Contrast normal and abnormal function.

4. Describe the mode of action of some commonly prescribed drugs.
- 4.2 Intellectual Skills
Appreciate from practical experience, the value and limitations of the quantitative study of human function.
- 4.3 Practical Skills
Some specialised skills in the measurement of physiological function will be developed during the practical sessions and there will be an opportunity to communicate in discussion groups and analyse information.
- 4.4 Transferable Skills
Written communication skills, the use of IT and numeracy skills are required in the fulfilment of coursework assignments.

5.0 ASSESSMENT OF THE UNIT

The unit will be assessed by an examination (60% of marks) and coursework (40% of marks).

The examination assesses Learning Outcomes 1 – 4. The coursework assignment assesses Learning Outcomes 1, 2 and 5.

The pass mark for the unit is 40% and a minimum mark of 30% must be achieved in both the examination element and the coursework element.

Satisfactory attendance at laboratory sessions is required in order to pass the unit.

The coursework assignment relates to the laboratory session, Exercise Physiology, (40% of total unit mark). Further details of the assignment will be available in the practical class.

The deadline for handing in the assignment is Thursday 11th December 2008.
Submit to Mike Hibbs via the Faculty Office.

Failure to acknowledge the work of others will be regarded as plagiarism, as will the direct copying of text from published sources. Students suspected of plagiarism will be required to submit an electronic version of their work for analysis by the plagiarism-detecting software, TurnitinUK

It is the intention that TurnitinUK will be available on the unit Blackboard site for students to use prior to submission of work. A help sheet for students explaining how to use TurnitinUK is available from

<https://www.lsbu.ac.uk/bb/downloads/TurnitinBBstudent.pdf>

Students must keep a copy of all course work submitted and must obtain a receipt when handing work in.

Compulsory Examination Question

The following question will appear on the examination paper and will be worth 25% of the exam mark.

Demonstrate your understanding of homeostasis and negative feedback control with reference to three examples. One example is to be obtained from renal physiology, one example is to be obtained from respiratory physiology and one example is to be obtained from cardiovascular physiology.

Award of marks

As a general guide, marks are awarded for the following levels of achievement:

- | | |
|------------------|---|
| >70% | Comprehensive and competent answer. Well communicated. Evidence of additional reading and original thinking. Good analysis of the problem and logical solutions. Factually correct. |
| 60 - 70 % | Overall competent and logical insight into the problem. Largely factually correct. Coverage not extensive but original thinking. |
| 40 - 60% | Generally competent. Some factual errors. Overall understanding but lack of convincing answer |
| below 40% | A lack of understanding of the problem. Superficial answer. Factual errors. Poor communication skills. |

Criteria used in assessing essay assignments and essay-type examination questions

Guidance only.

A more specific guide to the assessment of essay assignments and essay-type examination questions is given below. These criteria are intended to provide a benchmark against which marks allocated to an essay-type question can be compared, in order to see if they give a reasonable assessment of the quality of an answer.

1st Class (70-100%)

An excellent answer displaying complete understanding of the question. It presents all, or virtually all, the relevant 'given' information. Furthermore it will normally, where relevant, contain significant '**non-given**' (not presented within the taught unit programme) information displaying evidence of wider reading and an ability to synthesise information from diverse sources. The answer will be logically organised and well presented, it should be substantially error-free. It may well, if appropriate, show originality of thought or approach and will display insight.

2(i) - Upper 2nd Class (60-69%)

A very good answer showing a sound understanding of the question. It will contain all, or nearly all, the relevant 'given' information. It should normally display evidence of wider

reading or contain 'non-given' information. It will have a low error level and will not contain any serious errors. The answer should be logically presented. Better answers in this category may display originality or 'synthetic' ability.

2(ii) - Lower 2nd Class (50-59%)

A sound satisfactory answer, containing most of the 'given' information but will probably display little or no evidence of wider reading or originality. It will normally have a low error level. Some answers in this category may display some attributes of a 2(i) answer but with a higher level of error, however, and less logical presentation.

3rd Class (40-49%)

Although displaying some understanding of the question the answer will be incomplete and show a poor appreciation of the subject. It will contain relevant 'given' information but may have a high level of errors or irrelevancies. Important points will not be addressed.

Presentation may be poor.

FAIL (<40%)

An inadequate answer lacking substance and understanding, it may not represent a serious attempt. Where the question has been understood the answer will be very limited and probably contain many errors. Where the student has answered the wrong question, marks may still be given if relevant information is presented.

ACADEMIC MISCONDUCT

Students are referred to the University's Student Handbook Section 10.12 Academic misconduct, which summarises Chapter 13 of the academic regulations. The full version of the regulations is available from the registry (situated in Technopark building).

Sections taken from 10.12 academic misconduct section of the student handbook:

Academic misconduct is defined as 'any attempt to gain unfair advantage in assessment, or to help another student gain unfair advantage, by deception or fraudulent means.'

Some examples of academic misconduct:

Assisting another student to gain unfair advantage – for example by allowing another student to copy your work, or use an electronic copy of your work.

Syndication: The submission of pieces of work, which are substantially similar by two or more students. This may apply within the same institution or in a number of institutions, either at the same time or different times.

Plagiarism: To 'take and use another person's thoughts, writings, inventions as one's own'. Representing another person's work as your own, without acknowledging the source. Examples of this are provided in your student handbook (10.12d).

Collusion: Representing as your own piece of work which two or more students have undertaken together, without permission to do so.

Bribery: Offering payment or other inducement to another person in order to gain improper advantage in assessment or to falsify the result of assessment.

Commission: Commissioning another person to undertake all or part of an assignment presented as your own work, or knowingly undertaking work for another student to present his or her own work.

6.0 INTRODUCTION TO STUDYING THE UNIT

6.1 Overview of the Main Content

Circulatory System

Functional anatomy. Heart; anatomy, mechanical and electrical events of cardiac cycle, cardiac output and its control. Vascular system; pressure / flow / resistance and other functional characteristics of different blood vessels. Regulation of systemic arterial pressure. Normal and abnormal patterns eg. posture, exercise, hypertension. Mode of action of commonly prescribed drugs.

Respiratory System

Functional anatomy. Ventilation and lung mechanics. Exchanges of gases in alveoli and tissues. Transport of respiratory gases. Control of respiration. Normal and abnormal patterns eg. hypoxia, exercise, respiratory acidosis and alkalosis. Mode of action of commonly prescribed drugs.

Renal System

Functional anatomy. Renal processes; filtration, reabsorption, secretion. Regulation of extracellular fluid; sodium and water balance, regulation of pH. Normal and abnormal patterns eg. exercise, haemorrhage, acidosis, alkalosis. Mode of action of commonly prescribed drugs.

Integration of Systemic Function

Examples will include the physiology of exercise and thermoregulation.

6.2 Overview of Types of Classes

This guide is designed to help you structure your learning by providing an indicative structure and content for the unit. It is a guide and not a definitive statement of what you will be taught. We will try to follow the published schedule as far as possible, but there may be some variation as the unit develops and as we try to match the pace and content of the teaching to student needs.

Lectures

Tuesdays at 2.00 and 3.20. All lectures with Mike Hibbs.

Tutorials

Remedial tutorials will be held on Tuesdays at 4.15. Please do not hesitate to bring any queries from the lectures, practicals or your private study to these sessions.

Laboratory Sessions

There are four group-based sessions which offer opportunities for making physiological measurements, observing demonstrations, carrying out data handling and discussing features of scientific method, experimental design etc.

Your individual timetable will confirm the time and place of the laboratory sessions.

Laboratory Sessions for Students on Bioscience, Nutrition etc

- Week 3:** Cardiovascular function (including arterial blood pressure and ECG)
- Week 4:** Lung function and ventilation
- Week 5:** Exercise Physiology
- Week 6:** Review of exercise physiology and preparation for assignment.

Laboratory Sessions for Students on Sport & Exercise Science

- Week 7:** Exercise Physiology
- Week 8:** Review of exercise physiology and preparation for assignment.
- Week 9:** Cardiovascular function (including arterial blood pressure and ECG)
- Week 10:** Lung function and ventilation

6.3 Importance of Student Self-Managed Learning Time

Students are expected to carry out approximately 108 hours of private study as part of this unit. This is the most substantial component of the unit time and it is the part which is often neglected. **At the very least** you should do the directed reading week by week throughout the semester. Lectures will provide overviews and key points - your learning must also come from your reading. You are advised to purchase the core text. **Success in the unit depends on your constructive use of private study time.**

7.0 THE PROGRAMME OF TEACHING, LEARNING AND ASSESSMENT

Week	Topic	Ref. Vander's Human Physiol. 10th edition
1	Introduction and Overview Autonomic Nervous System	Chapter 1
2	Cardiovascular System, Functional Anatomy The Heart	388-399
3	The Heart continued	403-414
4	Blood Vessels and the Circulation	414-436
5	The Control of Circulation Transport of Respiratory Gases	436-451 500-506
6	Respiratory System, Functional Anatomy Ventilation and the Mechanics of Breathing	478-499
7	Respiratory Gas Exchange Control of Respiratory Function	478-483 507-515
8	Urinary System, Functional Anatomy Filtration, Reabsorption and Secretion	526-536
9	Water and Sodium Balance	541-555
10	Water and Sodium Balance continued Acid-Base Balance, Diuretics	541-555 563-570
11	Review Control and Integration Thermoregulation	640-646
12	Revision Class	

End of Unit Review – your feedback is valuable.

It is University policy to obtain student feedback at the end of each unit. This is carried out using a standard form that should be completed and returned in a way that should maintain your anonymity. The results of the tick-box responses and any additional commentary are presented and discussed at the Subject Area Review & Planning Meetings that are held twice per year. Should it be required, members of staff at these meetings agree to modify aspects of unit delivery and assessment for the next time that the unit will be presented (usually for the following year).

8.0 LEARNING RESOURCES

8.1 Core Materials

Widmaier E., Raff H., Strang K. *Vander's Human Physiology: The Mechanisms of Body Function, 10th. ed.*, McGraw Hill (2006)

8.2 Optional Materials

Other Useful Texts

The University library has a good stock of Human Physiology texts filed under 612. Below is a small selection.

Silverthorn D. *Human Physiology, an integrated approach. 4th ed* Pearson (2007)

Marieb E.& Hoehn K. *Human Anatomy & Physiology, 7th ed.*, Pearson (2007)

Sherwood L. *Human Physiology: From Cells to Systems, 6th ed.*, Thomson (2007)

The following online text is a useful resource

www.ultranet.com/~jkimball/BiologyPages

Past examination paper

BSc (Hons) Human Biology; Applied Biology; Food, Nutrition & Health Schemes
BSc (Hons) Sport & Exercise Science

Two Hours

HUMAN PHYSIOLOGY (Systems)

Instructions to Candidates

You must answer Question 1 (Section A)

You must answer 2 questions from Section B

You must answer all the questions in Section C

You are required to answer Section C in your question book

SECTION A (25 marks)

1. Give an account of how the cardiovascular and respiratory systems respond to the disturbance in homeostasis caused by muscular exercise.

SECTION B (40 marks. Answer 2 questions each worth 20 marks)

2. Write short notes on the autonomic nervous system and make comparisons between the sympathetic and parasympathetic divisions.
3. Give an account of the transfer of an oxygen molecule from room air to an exercising muscle cell. Consider the driving forces, the movement across epithelia and oxygen transport in the blood.
4. Explain how we are able to produce urine of different osmolarities depending on our state of hydration/dehydration.
5. Give an account of the control of breathing.
6. Give an account of the physiological factors contributing to thermoregulation whilst doing physical work in a hot environment.
- 7.a). Outline the process that results in the formation of interstitial fluid at the capillaries. (10 marks) AND
- 7.b). Give an account of the various imbalances that can occur which result in excessive interstitial fluid (oedema). (10 marks)

8. Demonstrate your understanding of negative feedback control with reference to the short-term reflex control of arterial blood pressure and compare this with the long-term control of arterial blood pressure.

SECTION C (35 marks. Answer all questions)

9. Person X is prone to occasional premature heartbeats. Explain why the stroke volume ejected on the next heart beat after a premature beat is larger than normal. NB At a given heart rate, the time interval between a premature beat and the next normal beat is longer than the interval between two normal beats. (4 marks)

10. If a person's arterial blood pressure recording is described as 130/76 mm Hg.

- a) What is the systolic pressure?
- b). What is the diastolic pressure?
- c). What is pulse pressure?
- d). What is mean arterial pressure?

Questions e), f). and g). refer to the determination of arterial blood pressure by sphygmomanometry

- e). Would any sound be heard at the brachial artery if the pressure in an external cuff was 140 mm Hg? Yes or No?
- f). Would any sound be heard at the brachial artery if the pressure in an external cuff was 125 mm Hg? Yes or No?
- g). Would any sound be heard at the brachial artery if the pressure in an external cuff was 70 mm Hg? Yes or No?

(7 marks)

11. Answer True or False (6 marks)

- a). Respiratory Acidosis is generally caused by hyperventilation.
- b). Metabolic Alkalosis could be due to prolonged vomiting.
- c). Haemoglobin contributes to H^+ ion buffering capacity of the body.
- d). Pulmonary arterioles constrict when the local pO_2 rises.
- e). Arterioles in skeletal muscle constrict when the local pCO_2 rises.
- f). Flow = Pressure x Resistance

12. With reference to the oxygen-haemoglobin saturation curve answer left or right. (5 marks)

- a). The curve will shift to the with an increase in temperature.
- b). The curve will shift to the with an increase in local DPG concentration.
- c). The curve will shift to the with a fall in pH.
- d). A shift to the favours the loading of oxygen onto the haemoglobin molecule.
- e). If the local pCO_2 is high, the curve will shift to the

13. Most of the sodium is reabsorbed in the tubules of the nephron, whilst the fine regulation of reabsorption takes place in the tubule. Aldosterone promotes the of sodium from the nephron. Atrial Natriuretic Factor promotes the of sodium from the nephron. Antidiuretic Hormone promotes the of water from the nephron. (5 marks)

14. (2 marks each)

- a).** If the heart rate is 66 beats per minute and the cardiac output is 5.0 litres per minute, what is the value of the stroke volume?
- b).** If aortic blood flow is 4.8 litres per minute, what is the blood flow in the pulmonary vein?
- c).** If a man has a tidal volume of 0.6 litre, a breathing frequency of 12 breaths per minute and a dead space volume of 0.16 litre, what is his alveolar ventilation?
- d).** Examination of an electrocardiogram shows that successive QRS complexes are, on average, 0.8 seconds apart. What is the heart rate in beats per minute?

SUMMARY NOTES

CARDIOVASCULAR SYSTEM, CIRCULATION AND BLOOD

PRESSURE, FLOW, AND RESISTANCE

1. The cardiovascular system consists of two circuits: the pulmonary circulation, from the right ventricle to the lungs and then to the left atrium; and the systemic circulation, from the left ventricle to all peripheral organs and tissues and then to the right atrium.
2. Arteries carry blood away from the heart, and veins carry blood toward the heart.
 - a. In the systemic circuit, the large artery leaving the left heart is the aorta, and the large veins emptying into the right heart are the superior vena cava and inferior vena cava. The analogous vessels in the pulmonary circulation are the pulmonary artery and the pulmonary veins.
 - b. The microcirculation consists of the vessels between arteries and veins: the arterioles, capillaries, and venules.
3. Flow between two points in the cardiovascular system is directly proportional to the pressure difference between the points and inversely proportional to the resistance: $F \propto \Delta P/R$
4. Resistance is directly proportional to the viscosity of a fluid and to the length of the tube. It is inversely proportional to the fourth power of the tube's radius, which is the major variable controlling changes in resistance.
5. Poiseuille's Law.

THE HEART

ANATOMY

1. The atrioventricular (AV) valves prevent flow from the ventricles back into the atria.
2. The pulmonary and aortic valves prevent back flow from the pulmonary trunk into the right ventricle and from the aorta into the left ventricle.
3. Cardiac muscle cells are joined by gap junctions that permit action potentials to be conducted from cell to cell.
4. The myocardium also contains specialized muscle cells that constitute the conducting system of the heart, initiating the cardiac action potentials and speeding their spread through the heart.
5. The wall of the left ventricle is much thicker than the wall of the right ventricle.

HEARTBEAT COORDINATION

1. Cardiac muscle cells must undergo action potentials for contraction to occur.
 - a. The rapid depolarization of the action potential in atrial and ventricular cells is due mainly to an increase in sodium permeability.
 - b. Following the initial rapid depolarization, the membrane remains depolarized (the plateau phase) almost the entire duration of the contraction because of prolonged entry of calcium into the cell through slow plasma-membrane channels.
2. The SA node generates the current that leads to depolarization of all other cardiac muscle cells.
 - a. The SA node manifests a pacemaker potential, which brings its membrane potential to threshold and initiates an action potential.
 - b. The impulse spreads from the SA node throughout both atria and to the AV node, where a small delay occurs. The impulse then passes in turn into the bundle of His, right and left bundle branches, Purkinje fibers, and to all ventricular muscle fibers.
3. Calcium, mainly released from the sarcoplasmic reticulum (SR), functions as the excitation-contraction coupler in cardiac muscle, as in skeletal muscle, by combining with troponin.
 - a. The major signal for calcium release from the SR is calcium entering through voltage-gated calcium channels in the plasma membrane during the action potential.
4. Cardiac muscle cannot undergo summation of contractions because it has a very long refractory period.

MECHANICAL EVENTS OF THE CARDIAC CYCLE

1. The cardiac cycle is divided into systole (ventricular contraction) and diastole (ventricular relaxation).
 - a. At the onset of systole, ventricular pressure rapidly exceeds atrial pressure, and the AV valves close. The aortic and pulmonary valves are not yet open, however, and so no ejection occurs during this isovolumetric ventricular contraction.
 - b. When ventricular pressures exceed aortic and pulmonary trunk pressures, the aortic and pulmonary valves open, and ventricular ejection of blood occurs.

- c. When the ventricles relax at the beginning of diastole, the ventricular pressures fall significantly below those in the aorta and pulmonary trunk, and the aortic and pulmonary valves close. Because AV valves are also still closed, no change in ventricular volume occurs during this isovolumetric ventricular relaxation.
- d. When ventricular pressures fall below the pressures in the right and the left atria, the AV valves open, and the ventricular filling phase of diastole begins.
- e. Filling occurs very rapidly at first so that atrial contraction, which occurs at the very end of diastole, usually adds only a small amount of additional blood to the ventricles.
2. The amount of blood in the ventricles just before systole is the end diastolic volume. The volume remaining after ejection is the end-systolic volume, and the volume ejected is the stroke volume.
3. Pressure changes in the systemic and pulmonary circulations have similar patterns but the pulmonary pressures are much lower.
4. The first heart sound is due to the closing of the AV valves, and the second is due to the closing of the aortic and pulmonary valves.

THE CARDIAC OUTPUT

1. The cardiac output is the volume of blood pumped by each ventricle and equals the product of heart rate and stroke volume.
 - a. Heart rate is increased by stimulation of the sympathetic nerves to the heart and by adrenaline (epinephrine); it is decreased by stimulation of the parasympathetic (vagus) nerves to the heart.
 - b. Stroke volume is increased by an increase in end-diastolic volume (the Frank-Starling mechanism) and by an increase in contractility due to sympathetic-nerve stimulation or to epinephrine.

THE VASCULAR SYSTEM

ARTERIES

1. The arteries function as low-resistance tubes and as pressure reservoirs for maintaining blood flow to the tissues during ventricular relaxation.
2. The difference between maximal arterial pressure (systolic pressure) and minimal arterial pressure (diastolic pressure) during a cardiac cycle is the pulse pressure.
3. Mean arterial pressure can be estimated as diastolic pressure plus one-third pulse pressure.

ARTERIOLES

1. Arterioles, the dominant site of resistance to flow in the vascular system, play major roles in determining mean arterial pressure and in distributing flows to the various organs and tissues.
2. Arteriolar resistance is determined by local factors and by reflex neural and hormonal input.
 - a. Local factors that change with the degree of metabolic activity cause the arteriolar vasodilation and increased flow of active hyperemia
 - b. Flow autoregulation, a change in resistance that maintains flow constant in the face of a change in arterial blood pressure, is due to local metabolic factors and to arteriolar myogenic responses to stretch.
 - c. The sympathetic nerves are the only innervation of most arterioles and cause vasoconstriction via alpha-adrenergic receptors. In certain cases non-cholinergic, nonadrenergic neurons that release nitric oxide or other non-cholinergic vasodilators also innervate blood vessels.
 - d. Epinephrine causes vasoconstriction or vasodilation, depending on the proportion of alpha and beta-adrenergic receptors in the organ.
 - e. Angiotensin II and vasopressin cause vasoconstriction.
 - f. Some chemical inputs act by stimulating endothelial cells to release vasodilator or vasoconstrictor paracrine agents, which then act on adjacent smooth muscle. These paracrine agents include the vasodilators nitric oxide (endothelium-derived relaxing factor) and the vasoconstrictor endothelin-1.
3. Arteriolar control in specific organs varies considerably, including influences from metabolic factors, physical forces, autoregulation, and sympathetic nerves.

CAPILLARIES

1. Capillaries are the site of exchange of nutrients and waste products between blood and tissues.
2. Blood flows through the capillaries more slowly than in any other part of the vascular system because of the huge cross-sectional area of the capillaries.
3. Capillary blood flow is determined by the resistance of the arterioles supplying the capillaries and by the number of open precapillary sphincters.
4. Diffusion is the mechanism by which nutrients and metabolic end-products exchange between capillary plasma and interstitial fluid.

- a. Lipid-soluble substances move across the entire endothelial wall, whereas ions and polar molecules move through water-filled intercellular pores.
 - b. Plasma proteins move across most capillaries only very slowly, either by diffusion through water-filled channels or by vesicle transport.
 - c. The diffusion gradient for a substance across capillaries arises as a result of cell utilization or production of the substance. Increased metabolism increases the diffusion gradient and increases the rate of diffusion.
5. Bulk flow of protein-free plasma or interstitial fluid across capillaries determines the distribution of extracellular fluid between these two fluid compartments.
- a. Filtration from plasma to interstitial fluid is favoured by the hydrostatic pressure difference between the capillary and the interstitial fluid. Absorption from interstitial fluid to plasma is favoured by the plasma protein concentration difference between the plasma and the interstitial fluid.
 - b. Filtration and absorption do not change the concentrations of crystalloids in the plasma and interstitial fluid because these substances move together with water.
 - c. There is normally a small excess of filtration over absorption; this excess becomes lymph which eventually returns to the blood circulation.

VEINS

1. Veins serve as low-resistance tubes for venous return.
2. Veins are very compliant and contain most of the blood in the vascular system.
 - a. Their diameters are reflexively altered by sympathetic-mediated vasoconstriction so as to maintain venous pressure and venous return.
 - b. The skeletal-muscle pump and respiratory pump increase venous pressure locally and enhance venous return. Venous valves permit the pressure to produce only flow toward the heart.

INTEGRATION OF CARDIOVASCULAR FUNCTION – REGULATION OF SYSTEMIC ARTERIAL PRESSURE

1. Mean arterial pressure, the primary regulated variable in the cardiovascular system, equals the product of cardiac output and total peripheral resistance, $MAP = CO \times TPR$

BARORECEPTOR REFLEXES

1. The primary baroreceptors are the arterial baroreceptors--the two carotid sinuses and the aortic arch. (There are also non-arterial baroreceptors in the systemic veins, pulmonary vessels, and walls of the heart.)
2. Baroreceptors are stretch receptors and the firing rate of the arterial baroreceptors is proportional to mean arterial pressure.
3. An increase in firing of the arterial baroreceptors due to an increase in pressure causes, by way of the cardiovascular centre in the medulla, an increase in parasympathetic outflow to the heart and a decrease in sympathetic outflow to the heart, arterioles, and veins. The result is a decrease in cardiac output and a decrease in total peripheral resistance and, hence, a decrease in mean arterial pressure. The opposite occurs when the initial change is a decrease in arterial pressure. This is a good example of negative feedback control.
4. The baroreceptor reflexes are short-term regulators of arterial pressure.
5. Long-term regulation of arterial pressure reflects the kidneys' regulation of blood volume.

TRANSPORT OF OXYGEN IN THE BLOOD

1. Each litre of systemic arterial blood normally contains 200 ml of oxygen, more than 98% bound to haemoglobin and the rest dissolved.
2. The major determinant of the degree to which haemoglobin is saturated with oxygen is the partial pressure of oxygen in the blood, pO_2 .
 - a. Haemoglobin is almost 100% saturated at the normal systemic arterial pO_2 of 100 mm Hg. The fact that saturation is already more than 90% at a pO_2 of 60 mm Hg permits relatively normal uptake of oxygen by the blood even when alveolar P_{O_2} is moderately reduced.
 - b. Haemoglobin is 75% saturated at the normal systemic venous pO_2 of 40 mm Hg. Thus, only 25% of the oxygen has dissociated from haemoglobin and entered the tissues.
3. The affinity of haemoglobin for oxygen is decreased by an increase in pCO_2 , hydrogen-ion concentration, and temperature. All these conditions exist in the tissues and facilitate the dissociation of oxygen from haemoglobin.

4. The affinity of haemoglobin for oxygen is also decreased by binding with diphosphoglycerate, DPG which is synthesized by the erythrocytes. DPG increases in situations associated with inadequate oxygen supply and helps maintain oxygen release in the tissues.

TRANSPORT OF CARBON DIOXIDE IN THE BLOOD

1. When carbon dioxide molecules diffuse from the tissues into the blood, 10% remains dissolved in plasma and erythrocytes, 30% combines in the erythrocytes with deoxyhaemoglobin to form carbamino compounds, and 60% combines in the erythrocytes with water to form carbonic acid, which then dissociates to yield bicarbonate and hydrogen ions. Most of the bicarbonate then moves out of the erythrocytes into the plasma in exchange for chloride ions.
2. As venous blood flows through lung capillaries, $p\text{CO}_2$ decreases because of diffusion of carbon dioxide out of the blood into the alveoli, and the above reactions are reversed.

adapted from Human Physiology by Vander, Sherman & Luciano

SUMMARY NOTES – RENAL PHYSIOLOGY

SECTION A: BASIC PRINCIPLES OF RENAL PHYSIOLOGY

FUNCTIONS AND STRUCTURE OF THE KIDNEYS AND URINARY SYSTEM

1. The kidneys regulate the water and ionic composition of the body, excrete waste products, excrete foreign chemicals and secrete the hormones renin and erythropoietin. The first three functions are accomplished by continuous processing of the plasma.
2. Each nephron in the kidneys consists of a renal corpuscle and a tubule.
 - a. Each renal corpuscle comprises a capillary tuft, termed a glomerulus, and a Bowman's capsule, into which the tuft protrudes.
 - b. The tubule extends out from Bowman's capsule and is subdivided into many segments, which can be combined for reference purposes into four: proximal tubule, loop of Henle, distal tubule, and collecting duct. Multiple tubules join, beginning at the level of the collecting ducts, and empty into the renal pelvis, from which urine flows through ureters to the bladder.
 - c. Each glomerulus is supplied by an afferent arteriole, and an efferent arteriole leaves the glomerulus to branch into peritubular capillaries, which surround the tubule.

BASIC RENAL PROCESSES

1. The three basic renal processes are glomerular filtration, tubular reabsorption, and tubular secretion. The excretion of a substance is equal to the amount filtered plus the amount secreted minus the amount reabsorbed.
2. Urine formation begins with glomerular filtration (approximately 180 L/day) of protein-free plasma into Bowman's capsule.
 - a. Glomerular filtrate contains all plasma substances, other than proteins and substances bound to proteins, in virtually the same concentrations as in plasma.
 - b. Glomerular filtration is driven by the hydrostatic pressure in the glomerular capillaries.
3. As the filtrate moves through the tubules, certain substances are reabsorbed into the peritubular capillaries.

Reabsorption may occur by diffusion or by mediated transport.
Many of the mediated-transport systems manifest transport maximums, so that when the filtered load of a substance exceeds the transport maximum, large amounts may appear in the urine.
4. Tubular secretion (movement from the peritubular capillaries into the tubules) is a pathway in addition to glomerular filtration for a substance to gain entry to the tubule.

SECTION B: RENAL REGULATION OF SODIUM, WATER

TOTAL-BODY BALANCE OF SODIUM AND WATER

1. The body gains sodium and chloride by ingestion and loses them via the skin (in sweat), gastrointestinal tract, and urine.
2. The body gains water via ingestion and internal production, and it loses water via urine, the gastrointestinal tract, and evaporation from the skin and respiratory tract (as insensible loss and sweat).
3. For both water and sodium, the major homeostatic control point for maintaining stable balance is renal excretion.
4. The control of blood volume (hence the long term control of arterial blood pressure) depends upon the regulation of body sodium.

BASIC RENAL PROCESSES FOR SODIUM AND WATER

1. Sodium is freely filterable at the glomerulus, and its reabsorption is a primary active process dependent upon sodium pumps in the membranes of the tubular epithelium.
2. Sodium entry into the cell from the tubular lumen is always passive. Depending on the tubular segment it is either through channels or by cotransport with other substances.
3. Sodium reabsorption creates an osmotic difference across the tubule, which drives water reabsorption.
4. The posterior pituitary hormone ADH acts on the collecting ducts, the water-permeability of the collecting ducts is increased by this hormone. A large volume of dilute urine is produced when plasma ADH concentration, and hence water reabsorption by the collecting ducts, is low.
5. A small volume of concentrated urine is produced when plasma ADH concentration is high.
6. Countercurrent multiplication in the Loop of Henle creates interstitial fluid with a high osmolarity in the inner medulla.

RENAL SODIUM REGULATION

1. Sodium excretion is the difference between the amount of sodium filtered and the amount reabsorbed.
2. GFR, and hence the filtered load of sodium, are controlled by baroreceptor reflexes. Decreased vascular pressures cause decreased baroreceptor firing and hence increased sympathetic output to the renal arterioles, resulting in vasoconstriction and decreased GFR. These changes are generally relatively small under most physiological conditions.
3. The major control of tubular sodium reabsorption is the adrenal cortical hormone aldosterone, which stimulates sodium reabsorption in the distal tubules and collecting ducts.
4. The renin-angiotensin system is the major controller of aldosterone secretion. When extracellular volume decreases, renin secretion is stimulated by three inputs: (1) stimulation of the renal sympathetic nerves to the juxtaglomerular cells by baroreceptor reflexes; (2) pressure decreases sensed by the juxtaglomerular cells, themselves acting as intrarenal baroreceptors; and (3) a signal generated by low sodium or chloride concentration in the lumen of the macula densa.
5. Many other factors influence sodium reabsorption. One of these, atrial natriuretic factor, is secreted by cells in the atria in response to atrial distention; it inhibits sodium reabsorption.

RENAL WATER REGULATION

1. Water excretion is the difference between the amount of water filtered and the amount reabsorbed.
2. GFR regulation via the baroreceptor reflexes plays some role in regulating water excretion, but the major control is via ADH-mediated control of water reabsorption.
3. ADH secretion by the posterior pituitary is controlled by cardiovascular baroreceptors and by osmoreceptors in the hypothalamus.
 - a. Via the baroreceptor reflexes, a low extracellular volume stimulates ADH secretion and a high extracellular volume inhibits it.
 - b. Via the osmoreceptors, a high body-fluid osmolality stimulates ADH secretion and a low osmolality inhibits it.

SECTION C: HYDROGEN-ION REGULATION

SOURCES OF HYDROGEN-ION GAIN OR LOSS

1. Total-body balance of hydrogen ions is the result of both metabolic production of these ions and of metabolic gains or losses via the gastrointestinal tract and urine.
2. A stable balance is achieved by regulation of urinary losses.
3. The kidneys and the respiratory system are the homeostatic regulators of plasma hydrogen-ion concentration.

BUFFERING OF HYDROGEN IONS IN THE BODY

1. Buffering is a means of minimizing changes in hydrogen-ion concentration by combining these ions reversibly with anions such as bicarbonate and intracellular proteins.
2. The major extracellular buffering system is the carbon dioxide / bicarbonate system, and the major intracellular buffers are proteins and phosphates.

RESPIRATORY MECHANISMS

1. A decrease in arterial plasma hydrogen-ion concentration causes reflex hypoventilation, which raises arterial $p\text{CO}_2$ and, hence, raises plasma hydrogen-ion concentration toward normal. An increase in plasma hydrogen ion concentration causes reflex hyperventilation, which lowers arterial $p\text{CO}_2$ and, hence, lowers hydrogen ion concentration toward normal.

RENAL MECHANISMS

1. Tubular secretion of hydrogen ions, a process which is directly linked with absorption of bicarbonate, helps to maintain a normal pH of body fluids in the face of metabolic acid production.
2. Hydrogen and bicarbonate ions are generated within tubular epithelial cells under the influence of the enzyme carbonic anhydrase.
3. Hydrogen ions are actively transported into the tubular lumen.

CLASSIFICATION OF ACIDOSIS AND ALKALOSIS

1. Acid-base disorders are categorized as respiratory or metabolic.
 - a. Respiratory acidosis is due to retention of carbon dioxide, and respiratory alkalosis is due to excessive elimination of carbon dioxide.
 - b. All other causes of acidosis or alkalosis are termed metabolic and reflect gain or loss, respectively, of hydrogen ions from a source other than carbon dioxide.

Adapted from Human Physiology by Vander, Sherman & Luciano

SUMMARY NOTES ON RESPIRATION

ORGANIZATION OF THE RESPIRATORY SYSTEM

1. The respiratory system comprises the lungs, the airways leading to them, and the chest structures responsible for movement of air into and out of them.
 - a. The conducting zone of the airways consists of the trachea, bronchi, and terminal bronchioles.
 - b. The respiratory zone of the airways consists of the alveoli, which are the sites of gas exchange, and those airways to which alveoli are attached.
 - c. The alveoli are lined by type I cells with some type II cells, which produce surfactant.
 - d. The lungs and interior of the thorax are covered by pleura between the surfaces of which is an extremely thin layer of intrapleural fluid.

SUMMARY OF STEPS INVOLVED IN RESPIRATION

1. The steps involved in respiration are (1) bulk flow of air ie. ventilation, (2) gas exchange between alveolar air and lung capillaries, (3) bulk flow of blood ie. the circulation, (4) gas exchange between capillaries and tissue cells, and (5) cellular utilization and production of gases. In the steady state, the net volumes of oxygen and carbon dioxide exchanged in the lungs per unit time are equal to the net volumes exchanged in the tissues.

VENTILATION AND LUNG MECHANICS

1. Bulk flow of air between the atmosphere and alveoli is proportional to the difference between the atmospheric and alveolar pressures and inversely proportional to the airway resistance:
$$F = (P_{atm} - P_{alv}) / R$$
2. Between breaths at the end of an unforced expiration $P_{atm} = P_{alv}$, no air is flowing, and the dimensions of the lungs and thoracic cage are stable as the result of opposing elastic forces. The lungs are stretched and are attempting to recoil, whereas the chest wall is attempting to move outward. This creates a subatmospheric intrapleural pressure.
3. During inspiration, the contractions of the diaphragm and inspiratory intercostal muscles increase the volume of the thoracic cage.
 - a. This makes intrapleural pressure more subatmospheric and causes the lungs to expand to a greater degree than between breaths.
 - b. This expansion initially makes alveolar pressure subatmospheric, which creates the pressure difference between atmosphere and alveoli to drive air flow into the lungs.
4. During expiration, the inspiratory muscles cease contracting, allowing the elastic recoil of the chest wall and lungs to return them to their original between-breath size.
 - a. This initially compresses the alveolar air, raising alveolar pressure above atmospheric pressure and drives air out of the lungs.
 - b. In forced expirations, the contraction of expiratory intercostal muscles and abdominal muscles actively decreases thoracic dimensions.
5. Lung compliance is determined by the elastic connective tissues of the lungs and the surface tension of the fluid lining the alveoli. The latter is greatly reduced, and compliance increased, by surfactant, produced by the type II cells of the alveoli.
6. Airway resistance determines how much air flows into the lungs at any given pressure difference between atmosphere and alveoli. The major determinant of airway resistance is the radius of the airways.

EXCHANGE OF GASES IN ALVEOLI AND TISSUES

1. Exchange of gases in lungs and tissues is by diffusion, as a result of differences in partial pressures. Gases diffuse from a region of higher partial pressure to one of lower partial pressure.
2. Normal alveolar gas pressure for oxygen is 105 mm Hg and for carbon dioxide 40 mm Hg.
3. The average value at rest for systemic venous PO_2 is 40 mm Hg and for PCO_2 is 46 mm Hg.
4. As systemic venous blood flows through the pulmonary capillaries, there is net diffusion of oxygen from alveoli to blood and of carbon dioxide from blood to alveoli. By the end of the pulmonary capillaries, the blood gas pressures have become equal to those in the alveoli.
5. Inadequate gas exchange between alveoli and pulmonary capillaries may occur when the alveolar-capillary surface area is decreased, when the alveolar walls thicken or when there are ventilation-perfusion inequalities.

6. Significant ventilation-perfusion inequalities cause the systemic arterial PO_2 to be reduced. An important mechanism for opposing mismatching is that a low local PO_2 causes local vasoconstriction of pulmonary arterioles thereby shunting blood away from poorly ventilated areas. (NB low local pO_2 causes systemic arterioles to dilate.)
7. In the tissues, net diffusion of oxygen occurs from blood to cells, and net diffusion of carbon dioxide from cells to blood.

CONTROL OF RESPIRATION

1. Breathing depends upon cyclical inspiratory muscle excitation by the nerves to the diaphragm and intercostal muscles. This neural activity is triggered by the medullary inspiratory neurons.
2. The most important inputs to the medullary inspiratory neurons for the involuntary control of ventilation are from the peripheral chemoreceptors (the carotid and aortic bodies) and the central chemoreceptors.
3. Ventilation is stimulated, via the peripheral chemoreceptors, by a decrease in arterial PO_2 , but only when the decrease is large.
4. Ventilation is stimulated, via both the peripheral and central chemoreceptors, when the arterial PCO_2 goes up even a slight amount. The stimulus for this reflex is not the increased PCO_2 itself, but the concomitant increased hydrogen-ion concentration in arterial blood and brain extracellular fluid.
5. Ventilation is also stimulated, mainly via the peripheral chemoreceptors, by an increase in arterial hydrogen-ion concentration resulting from causes other than an increase in PCO_2 . The result of this reflex is to restore hydrogen ion concentration toward normal by lowering PCO_2 .
6. Ventilation is inhibited by an increase in arterial PO_2 and by a decrease in arterial PCO_2 or hydrogen-ion concentration.
7. During moderate exercise, ventilation increases in exact proportion to metabolism, but the signals causing this are not known.
 - a. The proportional increases in ventilation and metabolism during moderate exercise cause the arterial PO_2 , PCO_2 , and hydrogen-ion concentration to remain unchanged.
 - b. Arterial hydrogen-ion concentration increases during very strenuous exercise because of increased lactic acid production. This accounts for some of the hyperventilation seen in that situation.
8. Ventilation is also controlled by reflexes originating in the airways and thorax eg. feedback signals from stretch receptors also NB irritant receptors.
9. Ventilation pattern can be modified by conscious processes originating from higher centres of the brain.

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