

Midterm Exam 2

The exam is not cumulative: therefore, you only need to cover the metabolism/metabolic rate, electrocardiography and cardiovascular response to exercise material. I am always available for quick questions via e-mail.

Possibly useful websites:

Leif - <http://stripe.colorado.edu/~saul/physiology/>
Mike - <http://ucsu.colorado.edu/~pascoe/physiology/index.html>
IPHY - <http://www.colorado.edu/intphys/iphy3435/index.html>

Prioritize in this order:

1. Read the manual and notes: understand all the expected outcomes from the experiments and re-read closely to pick up some of the obscure information for tricky questions.
2. Be prepared to use Chart and Scope to replicate parts of the lab sessions.
3. Cover all the questions we had on the quizzes and homeworks.
4. Ask questions if you don't understand something.

Metabolism and Metabolic Rate

Big picture: Metabolic rate, as assessed by oxygen/carbon dioxide, should increase linearly with external work intensity. RER (also called RQ when between 0.7 and 1.0) is indicative of substrate utilization, which changes from fat (0.7) to carbohydrates (1.0) as exercise intensity increases. Not all metabolic energy is converted directly to mechanical work - lost as heat etc. (economy).

- Ventilation increases with exercise intensity to supply a greater volume of **air**. There is now more *available* O₂, but it is still up to the tissues to extract it. For example, if you breathed much more deeply and faster *without exercising*, your VE would increase greatly but VO₂ would not change much because you are still at rest i.e. no metabolic demand for more oxygen.
- VO₂ increases with exercise intensity because as intensity increases, more O₂ is required for oxidative metabolism to provide adequate energy production as efficiently as possible (oxidative is more efficient than anaerobic energy production).
- VCO₂ increases since CO₂ is a product of substrate oxidation and also because of bicarbonate buffering and hyperventilation at higher workloads - which also explains why the RER can increase beyond 1 at very high workloads. *Recent* meals can also influence RER since the body will preferentially metabolize recently eaten food.
- Theoretically, the increase in RER with increasing exercise intensity corresponds to a shift from fat utilization to carbohydrate because ATP is needed at a much greater rate. Anaerobic metabolism of carbohydrate re-synthesizes ATP faster than oxidative metabolism of fat. Anaerobic metabolism only uses carbohydrates.
- What does 'economy' mean in the context of this experiment?
- Know the abbreviations included in my notes' bonus slides.
- What is BMR and what controls are typically used to control metabolic rate to measure BMR accurately? How is a MET related to BMR?
- What is 'steady state exercise' and what are its typical characteristics?
- Calculate metabolic rate (kcal/min) if you are given RQ, VO₂, and table 3 on p. 92.
- Distinguish between Ve_{ATP} and Ve_{STP}. STP = standard temp and pressure; 0°C, 760mmHg. ATP = ambient temp and pressure i.e. whatever the local conditions are.
- What is the equation for the oxidation of foodstuffs (p. 86)? Note that it is not stoichiometric.
- Features of direct vs. indirect calorimetry (technique, advantages/disadvantages)
- All that other stuff in the lab manual - scaling/allometry/anabolism/catabolism/calorie etc.
- Use of Chart for interpreting any of the data we recorded in class:
 - o Tidal volume
 - o Breathing rate
 - o VE_{ATP}

Cardiovascular Response to Exercise

Big Picture: As exercise intensity increases, the demand for O_2 , and therefore blood flow, in working tissues also increases. Q increases to meet the demand (HR and stroke volume increase). With this rise in Q , systolic pressure goes up. TPR decreases to accommodate increased Q . The reduction in TPR allows systolic BP to increase within a safe range. Diastolic BP largely unaffected.

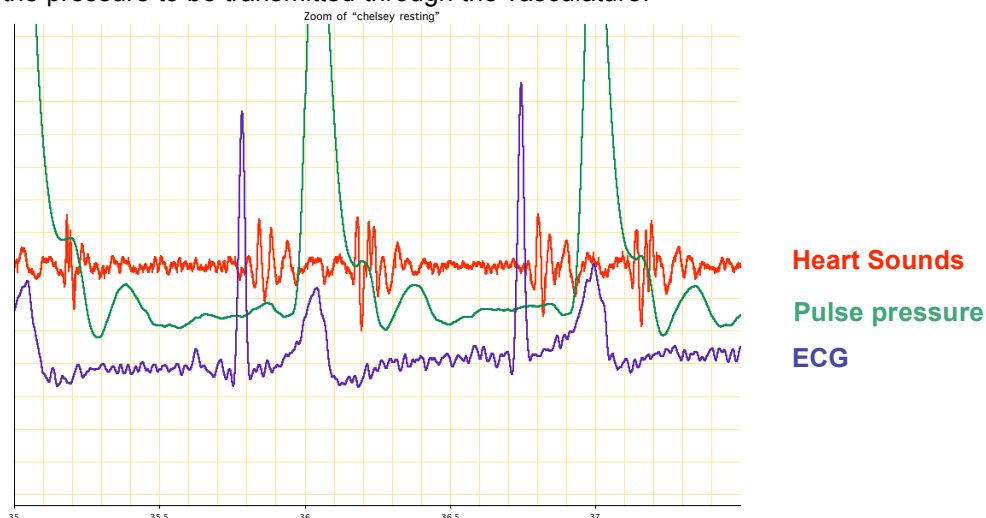
- Be able to define, calculate and know the variables that affect: systolic pressure, diastolic pressure, pulse pressure, mean arterial pressure, heart rate, cardiac output, total peripheral resistance, ventilation.
- *Cardiac Output (Q)*: volume of blood pumped from left ventricle per minute. $Q = HR \times SV$. Increases linearly at first but rate of increase slows down with higher workloads. HR increases linearly with intensity; SV increases linearly at first but plateaus early.
- *Systolic and diastolic BP*: systolic BP increases with increased Q since there will be more blood in the vessel. Diastolic BP doesn't change much if at all – a vessel at rest is a vessel at rest. Indeed, if diastolic BP does increase it suggests some kind of cardiovascular pathology.
- *Mean Arterial Pressure*: average blood pressure throughout cardiac cycle; since diastolic phase comprises approx 2/3 of the cardiac cycle, systemic pressure is closer to diastolic pressure than systemic for a longer portion of time per cardiac cycle. $MAP = DBP + 1/3(SBP-DBP)$.
- *Peripheral Resistance (TPR)* resistance to blood flow through vascular system. Blood flow (and resistance) are dependent on vessel lumen diameter and blood viscosity. TPR is primarily altered in the smaller peripheral arteries. Why is it dangerous for the subject to suddenly stop pedaling and sit still during exercise?
- *Pulse Pressure*: pressure difference throughout cardiac cycle (sBP - dBP)
- *Frank Starling Law*: increased end diastolic volume leads to increased contractile strength - (length-tension relation).
- *Ventilation/Perfusion Ratio (V_e/Q)* this measure is a comparison of the rate of air flow in the lungs to the rate of blood flow through the alveolar capillaries. Can be used to show whether ventilation (breathing) or perfusion (blood from heart) are impaired. Note that ventilation is not a limiting factor in aerobic exercise. You always have enough air, it's your ability to *extract and/or utilize* O_2 .
- Know the events of the cardiac cycle and the pathway of blood in the systemic and pulmonary circulation.
- Know the steps for obtaining sBP and dBP manually using a sphygmomanometer, and understand the physiological basis for this technique. Know how BP and MAP change as they go through the circulation i.e. large arteries to the small arteries, capillaries and veins.
- Know the role of the sympathetic and parasympathetic nervous system input and vasodilation and vasoconstriction.
- Use of Chart for interpreting any of the data we recorded in class:
 - o Tidal volume
 - o Breathing rate
 - o VE_{ATP}

Electrocardiography

Big Picture: The events of the cardiac cycle are closely related and tightly controlled. The ECG, heart sounds and peripheral pulse recording visualize the different stages of electrical activity, contraction and relaxation in the heart. In a clinical setting, these recordings can be used to identify the location and nature of heart pathologies.

- Myogenic nature of cardiac muscle - does not need direct innervation.
- Pathway of conduction: SA node - AV node - bundle of His - bundle branches - purkinje fibers + their physiological role.

- ECG waveform characteristics + what they represent. An ECG shows the electrical conduction of the heart, **not** contraction.
- Heart sounds + what they represent. Note that the sound isn't valve-closure *per se* that makes the sound, but the resultant turbulence. A murmur is any abnormal sound represented in the phonocardiogram. Typically, this is turbulent blood flow due to stenotic valves (cannot fully open) or regurgitation (valves don't fully close and blood leaks back).
- Commonalities between heart sounds (S1, S2), ECG trace and pulse pressure trace. What do the different parts represent, what should occur at the same time etc. Make sure you understand that the ECG for any particular component (e.g. ventricular depolarization) will appear first because it represents the change in electrical potential, while the other recordings will appear later because they correspond with physical events (e.g. ventricular contraction). S1 occurs at the beginning of systole (physical event); happens **after** QRS complex (electrical event). S2 occurs at the end of systole (physical event); happens **after** T-wave (electrical event). Pulse pressure (arterial pulse wave) occurs well after the QRS complex, usually close to S2 (end of systole). It takes time for the pressure to be transmitted through the vasculature.



- Pulse pressure characteristics (systolic/diastolic pressure, dicrotic notch).
- Basic ECG rhythms: normal sinus rhythm, tachycardia, bradycardia
- Abnormal ECG rhythms: ventricular fibrillation, atrial flutter
- ECG curiosities: PVC
- Limb lead [3 lead] set up/Einthoven's triangle.
- Effect of exercise? ECG trace (interval between T-wave and P-wave decreases); Heart sounds (interval between S2 and subsequent S1 decreases); Diastole is shorter; speed of contraction largely unaffected
- Use of Chart for interpreting any of the data we recorded in class.
 - o Identify parts of traces (ECG, pulse, heart sounds) and know their relation to the cardiac cycle (systole/diastole)

Final Tips:

1. Always answer questions, even if it's a guess. May be possible to get the answer by process of elimination.
2. Most answers (bar the practical questions) will be in the manual - use it as your primary resource.
3. Do NOT attempt to explain MCQ answers. I will mark you on the answer you circle regardless of anything you write in the margin.
4. ALWAYS include units in questions where units apply.