ACE’s Essentials of Exercise Science for Fitness Professionals
Chapter 5:
Physiology of Training
Learning Objectives

- This session, which is based on Chapter 5 of ACE’s Essentials of Exercise Science for Fitness Professionals, presents the acute and chronic adaptations to cardiorespiratory, resistance, and flexibility training, as well as principles that should guide the design and progression of an exercise program.

- After completing this session, you will have a better understanding of:
  - The acute and chronic adaptations of the cardiovascular and respiratory system in response to exercise
  - Fuel use during exercise
  - Acute adaptations of the muscular system, including muscle contractility and fatigue
  - Muscle growth and muscle-fiber adaptations in response to a progressive training program
  - General training principles: specificity, progressive overload, diminishing returns, and reversibility
  - The acute and chronic adaptations to flexibility training
Going from rest to exercise requires the circulatory and respiratory systems to increase oxygen delivery.

- To meet the increased demands of the muscles, two major adjustments in blood flow occur:
  - Redistribution of blood flow from the inactive organs to the active skeletal muscles
  - Increased cardiac output ($Q = SV \times HR$)
- Regulation of heart rate is controlled:
  - Intrinsically by the sinoatrial node (SA node)
  - Extrinsically by the nervous and endocrine systems
  - Changes in heart rate are influenced by the parasympathetic and sympathetic divisions of the autonomic nervous system (ANS).
Because of its specialized capacity, cardiac muscle is able to maintain its own rhythm.

The inherent rhythm of the heart is about 100 bpm and occurs because it can be innately stimulated via spontaneous depolarization and repolarization of the SA node.

- Impulses that originate at the SA node spread to the atrioventricular node (AV node), causing the atria to contract together, then the ventricles to contract together.
Parasympathetic Regulation

- Parasympathetic fibers reach the heart via the vagus nerves, which will make contact at both the SA node and AV node.

- When stimulated, vagus nerve endings will release acetylcholine, decreasing SA and AV node activity and reducing heart rate.
Sympathetic Regulation

- Sympathetic fibers connect to the heart via the cardiac accelerator nerves, which innervate the SA node and ventricles.

- When stimulated, epinephrine and norepinephrine are released, accelerating depolarization of the SA node and increasing heart rate and contractility.

- At the onset of exercise, the initial increase in heart rate (up to 100 bpm) is due to the withdrawal of parasympathetic tone.
Sympathetic Regulation (cont.)

- Increased cardiac output is influenced by:
  - An increase in end diastolic volume, which causes a stretch in cardiac fibers, thereby creating a stronger force of contraction
  - Stronger contractility results in more blood pumped per beat (i.e., a greater stroke volume)

- Increased blood flow to the working muscles is a result of:
  - Vasoconstriction of the vessels supplying non-working muscles (except the heart) and vasodilation of the vessels supplying the working muscles
  - Autoregulation, which is the monitoring system of the effectiveness of blood flow in response to the accumulation of metabolites
  - Vasoconstriction of the arteries supplying the viscera, which shifts blood from the abdominal organs to the working muscles
Systolic blood pressure has a much higher increase during exercise than diastolic blood pressure due to:

- Increased contractility of the heart
- Increased stroke volume
- The muscular need for greater force and pressure to deliver blood to the exercising muscles
- Vasodilation within the exercising muscle, which results in more blood draining from the arteries, through the arterioles, and into muscle capillaries, minimizing the change in diastolic pressure

Normal responses to blood pressure during exercise
Blood Distribution During Exercise

- Exercise affects the blood flow to various organ systems differently.
  - For example, skeletal muscle receives about 15–20% of total cardiac output during rest, but 80–85% during maximal exercise.

- Blood volume is affected by the hydrostatic pressure of a muscle contraction, accumulation of metabolites, and sweat.
  - The body preserves blood volume during exercise by several means.
    - Offsetting the small decrease in stroke volume by increasing heart rate (during steady state exercise)
    - Increasing vasoconstriction in non-working muscles to maintain peripheral resistance and blood pressure
    - Releasing vasopressin and aldosterone to help reduce water and sodium loss
  - During resistance training, the working muscles experience a temporary increase in fluid accumulation, which results in a feeling of fullness in the muscle (transient hypertrophy).
Ventilatory Regulation

- Aerobic exercise results in an increase of oxygen to the working tissues, increased return of carbon dioxide to the lungs, and an increase in the volume of air breathed per minute (minute ventilation—$V_E$).

- During submaximal exercise, ventilation increases proportionately with increased oxygen consumption and carbon dioxide production. As intensity increases to near maximal, the minute ventilation increases disproportionately to oxygen consumption.

- Ventilatory response to exercise increases with the exception of two distinct points at the first and second thresholds (VT1 and VT2).
  - VT1 represents the increased respiratory response to remove extra CO$_2$ produced by the buffering of lactate as it begins to accumulate in the blood.
  - VT2 represents the blood buffering systems becoming overwhelmed by rapidly increasing blood lactate.

*Note: VT1 = First ventilatory threshold; VT2 = Second ventilatory threshold; $\dot{V}_E$ = Minute ventilation
Ventilatory effects during aerobic exercise
Fast-acting Hormones

- Hormones that ensure blood glucose maintenance during exercise and quickly return blood glucose concentrations back to normal after exercise include:
  - Catecholamines (epinephrine and norepinephrine)
    - Increase cardiac contractility, leading to increased cardiac output
    - Vasoconstriction of non-working muscles increases total peripheral resistance, causing an increase in systolic blood pressure (SBP)
    - Epinephrine only:
      - Dilates respiratory passages and reduces digestive activity and bladder emptying
      - Stimulates the mobilization of stored carbohydrates and fats, the production and release of glycogen, and glycogenolysis in skeletal muscle
      - Promotes lipolysis
      - Alerts the central nervous system (CNS) of impending stressors
Insulin and Glucagon

- Activation of the sympathetic system during exercise suppresses the release of insulin from the pancreas.
  - Insulin sensitivity increases, requiring less insulin for the same effect.
  - Glucose uptake by the skeletal muscle occurs at a higher rate.

- Glucagon, also released from the pancreas, stimulates an almost immediate release of glucose from the liver.
  - Facilitates an increase in blood glucose levels in response to low levels (negative feedback loop)
  - This reaction takes effect as exercise progresses and glycogen stores deplete.
Cortisol, released by the adrenal cortex, stimulates the mobilization of free fatty acids (FFA) from adipose tissue, mobilizes glucose synthesis in the liver, and decreases the rate of glucose utilization in the cells.

- Increases with intensity and stress on the body
- Prolonged elevated levels have been linked to excessive protein breakdown, tissue wasting, negative nitrogen balance, and abdominal obesity.

Growth hormone, released by the anterior pituitary gland, supports the action of cortisol and plays a role in protein synthesis.

- Dramatic increase during short-term physical activity
Carbohydrates are the major food macronutrient for the metabolic production of adenosine triphosphate (ATP) and the only one whose stored energy can produce ATP anaerobically.

- Stored as glycogen in the muscle and liver
  - Glycogenolysis (breakdown of liver glycogen to glucose) is the primary regulator of blood glucose.

- Carbohydrates used during exercise come from both glycogen stores in muscle tissue and blood glucose.
  - The relative contribution of muscle glycogen and blood glucose used during exercise is determined by intensity and duration.
  - After the first hour of submaximal exercise, carbohydrate metabolism shifts from muscle glycogen to glycogenolysis in the liver.
Fuel Use During Exercise: Fats

- Fats are mainly stored as triglycerides in adipocytes, which must be broken down into FFAs and glycerol.
  - During low-intensity exercise, circulating FFAs from adipocytes are the primary energy source from fat, but during higher intensities, muscle triglyceride metabolism increases.
  - As duration increases, the role of plasma FFAs as a fuel source increases.
Fuel Use During Exercise: Protein

- Protein plays a small role in the fueling of exercise. It must be broken down into amino acids, which can be supplied to the muscle tissue from the blood and from the muscle fiber itself.
  - Skeletal muscle can directly metabolize certain amino acids to produce ATP.
  - During exercise, glucose stored in a non-exercising muscle can be delivered indirectly to the exercising muscle via the glucose-alanine pathway.

The glucose-alanine cycle

The table below provides a general review of the relative contributions of the three energy systems during exercise.

<table>
<thead>
<tr>
<th>System</th>
<th>Rate of ATP Production</th>
<th>Substrate</th>
<th>System Capacity</th>
<th>Utilization</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phosphagen</td>
<td>Very rapid</td>
<td>Creatine phosphate, ATP</td>
<td>Very Limited</td>
<td>High-intensity, very short-duration activities</td>
<td>Limited energy supply</td>
</tr>
<tr>
<td>Anaerobic Glycolysis</td>
<td>Rapid</td>
<td>Blood/muscle glucose, glycogen</td>
<td>Limited</td>
<td>High-intensity, short-duration activities</td>
<td>Lactic acid production</td>
</tr>
<tr>
<td>Aerobic</td>
<td>Slow</td>
<td>Blood glucose, glycogen, fatty acids, proteins</td>
<td>Unlimited</td>
<td>Lower-intensity, longer-duration activities</td>
<td>Slow rate of oxygen production</td>
</tr>
</tbody>
</table>

*Note: ATP = Adenosine triphosphate*
Lactate as Fuel

- Lactate is generally thought of as a waste product of glycolysis.
  - Plays a role in glucose production in the liver (gluconeogenesis)
    - During exercise, some of the lactic acid produced by skeletal muscle is transported to the liver via the blood and converted back to glucose and released into the bloodstream, traveling back to the skeletal muscles to be used as an energy source.
  - The cycle of lactate to glucose between the muscle and the liver is called the Cori cycle.
  - Serves as a direct fuel source for skeletal muscle and the heart
Muscle contractility depends on maximal force production, speed of contraction, and muscle fiber efficiency.

- Fast-twitch muscle fibers contain more myosin cross-bridges per cross-sectional area of fiber and produce 10–20% more force than slow-twitch muscle fibers.
- Fast-twitch muscle fibers have a higher concentration of myosin ATPase, allowing them to contract at a higher speed.
- Slow-twitch fibers are more efficient at using oxygen to generate ATP to fuel continuous muscle contractions due to their higher concentrations of myoglobin, larger number of capillaries, and higher mitochondrial enzyme activity.
Muscle Fatigue

- Muscle fatigue is associated with an acute bout of prolonged exercise in which muscular performance declines and sensations of muscle pain occur.
  - When muscle glycogen is depleted, an increase in the use of fat for energy occurs.
  - Fat mobilization and oxidation are much slower, resulting in a reduction of power output of the muscle.
  - Drinking a glucose and water solution near the point of fatigue may help for a short time, but glycogen will remain depleted.
  - High-carbohydrate diets (>60% of calories from carbohydrates) and carbohydrate loading can extend performance before “hitting the wall.”
Thermoregulation During Exercise in Altered Conditions

- In the heat:
  - Thermal core receptors signal the hypothalamus that core temperature is rising, which directs the nervous system to commence sweating and increase blood flow to the skin.
  - While four mechanisms are used to give off heat, evaporation is the major contributor during exercise.

<table>
<thead>
<tr>
<th>Mechanisms of Thermoregulation</th>
<th>Rest</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thermoregulatory Mechanism</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conduction and convection</td>
<td>20% of total</td>
<td>10–15% of total</td>
</tr>
<tr>
<td>Radiation</td>
<td>55–60% of total</td>
<td>5% of total</td>
</tr>
<tr>
<td>Evaporation</td>
<td>20% of total</td>
<td>80% of total</td>
</tr>
<tr>
<td>Excretion/lungs*</td>
<td>5–10% of total</td>
<td>&lt;2% of total</td>
</tr>
</tbody>
</table>

*300 mL in mucus membranes
Exercise in the Cold

- Three primary ways in which the body avoids excessive heat loss:
  - Peripheral vasoconstriction
  - Nonshivering thermogenesis
  - Shivering

- Air and water are the two major cold stressors.
  - As windchill increases, so does the risk of freezing body tissue.
  - The body loses heat four to five times faster in water than in air of the same temperature.
Regular, consistent exercise leads to several adaptations that allow the body to improve performance.

Cardiorespiratory changes

- Cardiorespiratory endurance capacity is determined by the ability of the cardiovascular and respiratory systems to deliver oxygen to active tissues, and the ability of those tissues to extract and use the oxygen during prolonged bouts of exercise.

<table>
<thead>
<tr>
<th>Summary of Adaptations to Cardiovascular Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Output Factors</td>
</tr>
<tr>
<td>------------------------</td>
</tr>
<tr>
<td>Decreased HR at any submaximal effort, including rest</td>
</tr>
<tr>
<td>Increased SV at rest, and at all intensities</td>
</tr>
<tr>
<td>Increased maximum cardiac output</td>
</tr>
</tbody>
</table>

*Note: HR = Heart rate; SV = Stroke volume*
Cardiorespiratory Changes: Blood Volume

- Increase in blood volume
  - An initial, rapid adaptation to exercise
  - Increase is due primarily to plasma and, to a lesser extent, red blood cells
    - Plasma volume can increase 12 to 20% after three to six aerobic-training workouts.
    - The number of red blood cells may increase, but the ratio of red blood cell volume to total blood volume may decrease.
Cardiorespiratory Changes: Heart Size and Volume

- Heart size and volume
  - Increase as an adaptation to increased work demand, but return to pre-training levels within several weeks if training ceases
  - Characterized by an increase of the left ventricular cavity and slight thickening of the walls.
  - Increase in size is due to endurance training and an increase in blood volume
  - These adaptations lead to an increase in cardiac force and the amount of blood pumped per beat.
  - Decreased resting heart rate (RHR) and exercise heart rate for a given intensity allow for longer diastolic filling and a reduced work requirement for the heart.
  - Improved maximal oxygen uptake (VO₂max) and decreased cardiac stress
Fick Equation

- Used to determine the rate at which oxygen is being used during physical activity
  
  \[ \text{VO}_2 = Q \times \text{a-vO}_2 \text{ difference} \]
  
  - Q = Cardiac output (HR x SV) = oxygen delivery
  - a-vO\(_2\) difference = oxygen extraction

### Stroke Volumes (SV) for Different States of Training

<table>
<thead>
<tr>
<th>Subjects</th>
<th>SV\text{rest} (mL/beat)</th>
<th>SV\text{max} (mL/beat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untrained</td>
<td>50–70</td>
<td>80–110</td>
</tr>
<tr>
<td>Trained</td>
<td>70–90</td>
<td>110–150</td>
</tr>
<tr>
<td>Highly Trained</td>
<td>90–110</td>
<td>150–220+</td>
</tr>
</tbody>
</table>

Improvements in VO$_2$max are due to increases in one or more of the following variables:

- Stroke volume
  - Increases at rest and during exercise result from regular training

- Heart rate
  - Regular training typically yields:
    - Decreased RHR of more than 10 bpm
    - Decreased submaximal heart rate of 10–20 bpm

- a-vO$_2$ difference
  - Increases with training, particularly at maximal exercise
  - Reflective of greater oxygen extraction at the tissue level and more effective distribution of blood flood to active tissue
Blood flow

- Increased blood flow to working muscles is enhanced through regular endurance training due to:
  - Increased capillarization of trained muscles
  - Greater recruitment of existing capillaries in trained muscles
  - More effective blood flow redistribution from inactive areas to active tissues
  - Increased blood volume

Blood pressure

- In response to regular endurance training, a decrease in resting SBP and DBP is noted only for borderline or moderately hypertensive individuals
  - Resistance training may also reduce SBP
Oxidative enzymes

- Responses to regular endurance training include:
  
  - Increase in the size and number of mitochondria in skeletal muscle
    - Enhances the muscle’s ability to use oxygen and produce ATP via oxidation
  
  - Increase in the activity of the mitochondrial oxidative enzymes
    - Slower rate of muscle glycogen utilization
    - Enhanced reliance on fat as fuel at any given exercise intensity
Neural Changes

- More significant as a result of resistance training than aerobic training
  - Occur in the early part of a strength-training program (1–3 weeks) before muscle hypertrophy occurs

- Motor-unit recruitment and synchronization
  - All-or-none principle: When activated, all muscle fibers in a motor unit contract maximally.
Neural Changes (cont.)

- **Rate coding**
  - A motor unit produces varying levels of force depending on the frequency at which it is stimulated.
    - Twitch
    - Summation
    - Tetanus
  - May increase with resistance training, resulting in increased frequency of discharge of the motor units and allowing trained muscles to reach peak force production more quickly

- **Diminished co-contraction**
  - In opposing muscles, when maximizing force generated by the agonist, the activation of the antagonist must be diminished.
Hormonal Changes

- Generally, the hormonal response to a given exercise load declines with regular endurance training (signifying an increase in sensitivity or efficiency).

<table>
<thead>
<tr>
<th>Hormones and Their Responses to Endurance Training</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hormone</strong></td>
</tr>
<tr>
<td>Epinephrine and norepinephrine</td>
</tr>
<tr>
<td>Cortisol</td>
</tr>
<tr>
<td>Insulin</td>
</tr>
<tr>
<td>Glucagon</td>
</tr>
<tr>
<td>Growth hormone</td>
</tr>
</tbody>
</table>


- Resistance training adaptations result in increases in growth hormone and testosterone (especially in men), which are associated with strength improvement.
General Adaptation Syndrome

- General adaptation syndrome refers to the body’s predictable response to stressful events (including heavy exercise).

- Three stages
  - Shock or alarm phase (usually lasts 2 to 3 weeks)
    - The individual initially exhibits signs of fatigue, weakness, and soreness.
    - He or she soon experiences remarkable gains (attributed to neuromuscular adaptations).
  - Adaptation or resistance phase (begins around weeks 4 to 6)
    - Major muscular adaptations (biochemical, mechanical, and structural)
    - Progressive increases in muscle size and strength
  - Exhaustion phase (may occur at any time)
    - Symptoms similar to the first phase, but inadequate repair or recovery time lead to burnout, overtraining, reduction or elimination of overload, injury, illness, or lack of adherence
Overtraining often occurs during periods of intense overload in which signs and symptoms are individualized and include a combination of both physiological and emotional factors.

Some signs and symptoms include:
- A decline in physical performance with continued training
- Elevated heart rate and blood lactate levels at a fixed submaximal work rate
- Weight loss
- Sleep disturbance
- Multiple colds or sore throats
- Irritability, restlessness, excitability, and/or anxiousness
- Loss of motivation and vigor
- Lack of mental concentration and focus
- Lack of appreciation for things that are normally enjoyable

The best way to prevent overtraining is periodization.
Delayed Onset Muscle Soreness (DOMS)

- Research suggests DOMS is caused by tissue injury from excessive mechanical force, particularly eccentric force, exerted on muscle and connective tissue.
  - Generally appears 24–48 hours after strenuous exercise
  - Is thought to result from a series of events activated by strenuous exercise:
    - First, structural damage occurs as a result of strenuous eccentric muscle actions.
    - As a result, calcium is leaked out of the sarcoplasmic reticulum and collects in the mitochondria, halting ATP production.
    - The build-up of calcium activates enzymes that break down proteins.
    - The breakdown of proteins causes an inflammatory process.
    - Lastly, the accumulation of histamines, potassium, prostaglandins and edema stimulates pain receptors, leading to the sensation of DOMS.
  - Attempt to reduce DOMS by starting at a low intensity and progressing slowly through the first few weeks while minimizing eccentric actions.
General Training Principles

- **Principle of specificity**
  - The exercise response to any training program is specific to the mode and intensity of training.

- **Principles of overload and progression**
  - Overload involves increasing the load on the tissue or system above and beyond the normal load.
  - Progression is the systematic process of applying overload.
General Training Principles (cont.)

- **Principle of diminishing returns**
  - The rate of fitness improvement diminishes over time as an individual’s fitness approaches its ultimate genetic potential.

- **Principle of reversibility**
  - When training ceases, all gains will return to pre-training levels and may possibly decrease to the point where they are only supporting the demands of daily use.

Hypothetical relationship between training program duration and improvements in aerobic capacity for three cardiorespiratory fitness levels (in healthy individuals).
Muscle Growth

- After a prolonged period of resistance training, chronic hypertrophy is responsible for strength gains.
  - Fiber hypertrophy
    - Results from one or more of the following: increased number of myofibrils, increased number of actin and myosin filaments, more sarcoplasm, and more connective tissue
    - Increased protein synthesis
    - Eccentric actions combined with high-velocity training promote greater increases.
  - Fiber hyperplasia
    - Stress to the muscle stimulates the migration of satellite cells to the damaged region to fuel existing muscle fibers and/or produce new ones.
    - In humans, most evidence points to muscle-fiber hypertrophy as the primary cause of increased muscle size associated with resistance training.
A resistance-training program that stimulates protein synthesis (and muscle growth) increases levels of testosterone and growth hormone.

- Growth hormone increases the availability of amino acids for protein synthesis and stimulates the release of IGF-1, which works with GH to stimulate muscle growth.
- Testosterone promotes the release of GH and interacts with neuromuscular system to stimulate muscle growth.
- These responses are brought about by performing large-muscle-group, multijoint exercises at high intensities with short rest intervals (30 to 60 seconds).
Muscle Glycogen Storage

- Approximately 300–400 grams of glycogen is stored in skeletal muscles and approximately 70 to 100 grams is stored in the liver.
  - At intensities >60% VO₂max, muscle glycogen is the predominant fuel source. Liver glycogen is more important during low-intensity activity.
  - Enhancing muscle glycogen storage involves eating a carbohydrate-rich diet and consuming carbohydrates within 30 minutes of high-intensity exercise.
  - It takes about 24 hours to fully restore muscle glycogen, if nutrient needs are met post-workout and the athlete was properly fueled prior to the workout.

<table>
<thead>
<tr>
<th>Nutrition Goals for Athletes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy</td>
</tr>
<tr>
<td>Carbohydrate</td>
</tr>
<tr>
<td>Protein</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
Muscle-buffering Capacity

- Muscle-buffering capacity refers to the muscles’ ability to neutralize the lactic acid that accumulates in them during high-intensity activity.
  - Delays the onset of fatigue
  - Allows the exerciser to perform at a higher intensity and duration before “hitting the wall”

- Training at the lactate threshold will enhance buffering capacity and delay muscle fatigue for subsequent training sessions.

- Ventilatory threshold is an indirect representation of lactate threshold.
  - Endurance training improves the ability to sustain high levels of submaximal ventilation.
Heat Acclimation

- Adaptations can take place as early as 9 to 14 days.
  - Adaptations include:
    - Increased plasma volume
    - Decreased heart rate and core temperature
    - Increased sweat rate
Flexibility Training

- Tissue properties affecting flexibility training:
  - Tissue elasticity
    - Mechanical property that allows a tissue to return to its original shape or size when an applied force is removed (“temporary deformation”)
    - A tissue reaches its “elastic limit” when it is stretched beyond the point where it cannot return to its normal length when tensile (stretching) force is removed.
    - The difference between the original resting length and new resting length is called “permanent deformation.”
    - The new state of permanent elongation is called “plastic stretch.”
    - Static stretching elongates the tissue to a point where deformation remains after the tension is removed.
Flexibility Training (cont.)

- **Tissue plasticity**
  - Allows the tissue to deform when it is loaded past its elastic limit
  - Once a tissue is set past its yield point, tissue failure resulting in additional deformation may occur with small increases in force.

- **Tissue viscoelasticity**
  - Viscosity allows tissues to resist loads and is dependent on time and temperature.
  - With exposure to low loads, most tissues exhibit elastic behavior; exposure to higher loads causes tissue to exhibit a plastic response.
Neurological Properties of Stretching

- Autogenic inhibition: the activation of a Golgi tendon organ (GTO) inhibits muscle spindle response
  - Initially, a low-force, long-duration (static) stretch stimulates low-grade muscle spindle activity and temporary increases in muscle tension.
  - Muscle spindles become desensitized as the stretch continues (referred to as stress-relaxation).
  - After 7 to 10 seconds, the increase in muscle tension activates the GTO response, inhibiting muscle spindle activity and allowing further muscle stretching.
  - Holding the stretch beyond 10 seconds stresses the collagen fibers, causing plastic deformation and lengthening the tissue (creep).
  - When the stretch ends, muscle spindles reestablish their threshold.
  - Repeating the stretch a finite number of times produces a gradual increase in muscle extensibility.
Reciprocal inhibition

- Activating the muscle on one side of a joint (i.e., the agonist) coincides with neural inhibition of the opposing muscle on the other side of the joint (i.e., the antagonist) to facilitate movement.

- Example: While performing a supine hamstring stretch, contraction of the hip flexor muscles on the leg being stretched will produce more active hip flexion, resulting in reciprocal inhibition of the hamstring muscle group, allowing them to be stretched further.
Static Stretching

- Static stretching involves moving the joints to place the targeted muscle group in an end-range position and holding that position for up to 30 seconds.
  - Does not elicit the stretch reflex, reducing the risk of injury with proper technique
  - Active
  - Passive
Proprioceptive Neuromuscular Facilitation (PNF)

- PNF incorporates the principles of autogenic inhibition and reciprocal inhibition.
  - There are three types of PNF, all of which begin with a passive (partner) 10-second pre-stretch.
    - Hold-relax
    - Contract-relax
    - Hold-relax with agonist contraction
Dynamic and Ballistic Stretching

- **Dynamic stretching**
  - Mimics the movement pattern to be used in an upcoming workout.
  - Dynamic stretching is an effective component of a warm-up.

- **Ballistic stretching**
  - Incorporates bouncing-type movements
  - Typically triggers the stretch-reflex, which increase the risk of injury
  - Has a role in conditioning and training if done correctly
Active Isolated Stretching (AIS)

- AIS follows a design similar to a traditional strength-training program.
  - Stretches, which never last longer than 2 seconds, are performed in sets, with each movement exceeding the resistance point of the prior stretch by a few degrees.
  - Each set isolates an individual muscle.
Myofascial Release

- Myofascial release applies pressure to tight, restricted areas of fascia and underlying muscle in an attempt to relieve tension and improve flexibility
  - It is thought that sustained pressure to a tight area can inhibit tension in a muscle by stimulating the GTO to bring about autogenic inhibition.
  - Trigger points can be diminished through the application of pressure followed by static stretching of the tight area.

- In the fitness setting, a foam roller is used, allowing the exerciser to control his or her own intensity and duration of pressure.
Static Stretching and Permanent Tissue Elongation

- Static stretching
  - Low-force, long-duration stretching at elevated tissue temperatures is more likely to result in plastic lengthening (compared to high-force, short duration).
    - Dynamic stretching can be used to warm up the muscles, and be followed by static stretching.
    - Static stretching is most effective when performed during the cool-down at the end of a training session.
  - Holding the stretch for 15 to 30 seconds appears to be the most effective means of increasing range of motion.
    - Each stretch should be repeated up to three to four times.
Understanding the transition from rest to exercise and the adaptations that occur in response to regular training of the cardiorespiratory, nervous, endocrine, muscular, and thermoregulatory systems is essential for proper program design from the first session or class to the last.

This session covered:
- Acute responses to exercise
- Fast- and slow-acting hormones
- Fuel use during exercise
- The three energy systems
- Thermoregulation during exercise
- Chronic adaptations to exercise
- Cardiorespiratory, neural, and hormonal changes
- General training principles
- Muscle growth
- Flexibility training