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SECTION 1
HUMAN MOVEMENT ANATOMY AND FUNCTION
Trainers are in the business of creating fitness. They apply exercises to their clients in order to induce adaptations in structure and function that lead to improved fitness. In order to do this effectively, reliably and safely, the working trainer must understand the structures they are stressing with exercise to produce the functional change that is fitness. This is the primary reason we learn anatomy and physiology. We need to understand the materials we work with in our occupation in order to accomplish the job we are expected to deliver.

Secondarily, there are communication reasons for learning structures and functions. Telling a trainee that he might have “pulled his thyroid” as rationale for hamstring pain, or telling a fellow professional about a client’s “ver-tee-byou-lar” (not the correct ver-te-bral) issues, does little to portray one as a knowledgeable professional. Anatomy and physiology form a common language that connects the trainer, the therapist, the clinician and the client. We must learn our profession inside out to earn the respect and recognition due an expert.

Understanding how something is built will help you understand how it works. The biological and engineering adage “form equals function” is quite important in exercise and fitness. When we want to get better at an exercise we need to understand how the structure of the body contributes to its performance. When bones come together at moveable joints, they create levers. Levers are simple machines, and this fact makes the bone-muscle-joint assembly a biological machine that produces every movement we make in exercise and life. Anatomical technical errors—something as small as too narrow of a bench-press grip, starting a pull with the bar too far away from the shins or running with the elbows held away from the body—can significantly reduce performance. Simple changes in structure can also change function; adding mass to a muscle makes the angle of pull closer to the optimal 90 degrees for force application, and losing mass reduces the number of muscle protein-interactions that can produce force, the good and the bad. This is anatomy and physiology in action.

The best place to start in the study of what makes a human move is learning the bones and muscles that actually allow human movement—in that order. The bones form a moveable scaffolding that allows the muscles attached to it to create gross (large scale) and fine (small scale) movement. Everything we do in the gym, at work, at play and in daily life is dependent on the ability of the muscles to move two or more bones through their range of motion.

SKELETON

The skeleton is comprised of 206 individual bones. It is a relatively easy learning task to memorize the names of all the bones, as there are lots of duplicates, such as right femur-left femur, right 12 ribs-left 12 ribs, etc. Furthermore, some of the 22 bones of the skull are not specifically relevant to exercise, so we can trim a little bit more of the memorization task. But before we begin naming names, we need to examine the basics of how bones are built.
SIMPLE BONE STRUCTURE
The bones most closely associated with movement are the long bones—the femur of the thigh, the humerus of the upper arm, the tibia and fibula of the shin, for example. These long bones form the levers that make movement possible. But there are other types of bones, too, and each of the four types has a characteristic structure: long bones, short bones, flat bones, irregular bones.

We will focus on the structure of bones that most directly affect movement: the long bones. They are named “long bones” simply because they are the longest bones in the body. They have a characteristic anatomical structure (Figure 1). There are eight areas of anatomical importance in long bones, with likely the most familiar being the epiphysis and the epiphyseal plate (commonly known as the “growth plate”).

**Epiphysis**—The term epiphysis describes the end of a long bone. There will be two of them.

**Epiphyseal Plate**—The epiphyseal plate is a thin but diffuse layer of cartilaginous tissue located between the epiphysis and diaphysis. There are many active cells that divide to produce bone growth. At full maturity, the cells cease activity and the plate fuses with the existing bone. At that point, longitudinal growth (gain in height) stops. Contrary to many misdirected and uninformed opinions, weighted exercise does nothing to harm these plates.

**Diaphysis**—The term diaphysis is used in reference to the shaft of the bone.

**Cancellous Bone**—This feature of long bones has also been called “spongy bone” as it appears similar to the texture of a very tightly pored and rigid sponge. Cancellous bone is usually found closer to the epiphysis and functions to help absorb external mechanical stress.

**Compact Bone**—Compact bone is found in the shaft of the bone and is comprised of mineralized connective tissue. It gives the skeleton much of its ability to support and protect.

**Cartilage**—This is a fibrous tissue that lines joint capsules where bone meets bone. The more a bone end is exposed to physical stress, the thicker the cartilage will be. So systematically loading bones in exercise is a healthy undertaking for bone and joint health.

**Medullary Cavity**—This central cavity is the site of bone-marrow genesis. Marrow is responsible for blood-cell production.

**Periosteum**—The periosteum appears as a fibrous sheath surrounding the bone.
BONE IDENTIFICATION

The next step is to learn the names and locations of the bones of the human body and how they are arranged to form the scaffold of movement. At this point in our learning, we are associating the names of the bones with locations in the body. A later and separate step toward expertise would be to learn all of the small bumps, ridges, dips and holes on and in individual bones that provide attachments for muscles or create other functional outcomes.

We will divide the skeleton up into parts or regions: the axial skeleton and the appendicular skeleton. The axial skeleton is so named as it creates the longitudinal (vertical) axis of the body. It runs from the skull down to the tailbone and includes the skull, vertebral column, ribs, collarbones and sternum. The appendicular skeleton is comprised of the appendages: the bones of the arms, legs, hands and feet.
Table 1.

<table>
<thead>
<tr>
<th>Skull</th>
<th>Vertebral Column</th>
<th>Chest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal (1)</td>
<td>Cervical vertebrae (7)</td>
<td>Sternum (1)</td>
</tr>
<tr>
<td>Temporal (2)</td>
<td>Thoracic vertebrae (12)</td>
<td>Ribs (24)</td>
</tr>
<tr>
<td>Parietal (2)</td>
<td>Lumbar vertebrae (5)</td>
<td></td>
</tr>
<tr>
<td>Occipital (1)</td>
<td>Sacrum (1)</td>
<td></td>
</tr>
<tr>
<td>Zygomatic (2)</td>
<td>Coccyx (1)</td>
<td></td>
</tr>
<tr>
<td>Nasal (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maxilla (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mandible (1)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Bones not listed = 20

Bones of the axial skeleton. Numbers in parentheses (x) are total number of that bonetype in the body.

Figure 2.
The observable bones of the skull.
The skull, vertebral column, sacrum, coccyx and ribs as they appear in the human skeleton. The cervical, thoracic and the lumbar vertebrae are generally referred to in an abbreviated form: C = cervical, T = thoracic and L = lumbar, followed by the sequential number of the bone when counted from the top of the section. For example, C3 refers to the third vertebrae below the skull, T7 refers to the seventh thoracic vertebrae (all thoracic vertebrae attach to ribs), and L1 refers to the first lumbar vertebrae (which is immediately below the 12th thoracic vertebrae, T12).
Figure 4.
The “Rib Cage” is part of the axial skeleton and attaches the vertebral column to the posterior. Anterior view—left. Superior view—right.

Table 2.

<table>
<thead>
<tr>
<th>Shoulder</th>
<th>Arm</th>
<th>Wrist *</th>
<th>Hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clavicle (2)</td>
<td>Humerus (2)</td>
<td>Pisiform (2)</td>
<td>Metacarpals (10)</td>
</tr>
<tr>
<td>Scapula (2)</td>
<td>Radius (2)</td>
<td>Lunate (2)</td>
<td>Proximal phalange (10)</td>
</tr>
<tr>
<td></td>
<td>Ulna (2)</td>
<td>Scaphoid (2)</td>
<td>Middle phalange (8)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trapezium (2)</td>
<td>Distal phalange (10)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Triquetral (2)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hamate (2)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Capitate (2)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trapezoid (2)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>*Collectively known as the carpals</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Bones of the upper appendicular skeleton. Numbers in parentheses (x) are the total number of that bone type in the body.
Figure 5.
The bones of the shoulder—the scapula and clavicle—are part of the appendicular skeleton and articulate with the bone of the upper arm: the humerus.
Figure 6.
The upper and lower arm bones of the appendicular skeleton. Note that the radius is on the thumb-side of the arm in all orientations.
Figure 7.
The upper appendicular skeleton is a key element of effectively transferring force to any exercise apparatus held by the hands.

Figure 8.
The bones of the wrist and hand are the terminal pieces of the upper appendicular skeleton. Here we are grouping eight bones of the right and left wrists into one group called the carpals. This is sufficient detail for early anatomy learning and memorization. 1 = radius, 2 through 9 = carpals, 10 through 14 = metacarpals, 15 through 19 = proximal phalanges, 20 through 23 = middle phalanges, 24 through 28 = distal phalanges.
Table 3.

LOWER APPENDICULAR SKELETON

<table>
<thead>
<tr>
<th>Leg</th>
<th>Ankle and Rear Foot*</th>
<th>Mid- and Forefoot</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femur (2)</td>
<td>Calcaneus (2)</td>
<td>Metacarpals (10)</td>
</tr>
<tr>
<td>Tibia (2)</td>
<td>Talus (2)</td>
<td>Proximal phalanges (10)</td>
</tr>
<tr>
<td>Fibula (2)</td>
<td>Navicular (2)</td>
<td>Middle phalanges (8)</td>
</tr>
<tr>
<td>Patella (2)</td>
<td>Cuboid (2)</td>
<td>Distal phalanges (10)</td>
</tr>
<tr>
<td></td>
<td>Cuneiform (6)</td>
<td></td>
</tr>
</tbody>
</table>

*Collectively called the tarsals

The bones of the hip form the transition and connection of the axial skeleton and the lower appendicular skeleton and are key elements of human movement during exercise. The ilium, ischium and pubis bones join to form the acetabulum, the point of articulation for the lower leg at the hip.
Figure 10.
The upper and lower leg, bounded by the hip above and the ankle below is composed of four bones. The femur articulates with the tibia and fibula to extend the lower appendicular downward to the terminal element, the ankle.

Figure 11.
The hips, knees and ankles form the basis of almost all functional exercises. Understanding how they are built and function is important for every fitness professional.
Figure 12.
The ankle and foot complete the lower portion of the axial skeleton. As we did with the wrist, we are grouping bones of the right and left ankle and rear foot into one group called the tarsals. This is sufficient detail for early anatomy learning and memorization.
MUSCLE

Everyone who eats meat is already familiar with the basic physical appearance of muscle—its texture, its color, its weight. But seeing these little slices of muscle do not provide us with an appreciation of the overall structure of an intact muscle doing what it was designed to do: be the engine of movement. Composed of thousands and thousands of individual muscle cells, these engines act on the levers formed by the bones and joints. Together they create movement.

It is the unique structure of muscle at both the microscopic (small scale) and the gross (large scale) that enables contraction and force generation. We will briefly examine both of these levels of structure in order to understand the basic anatomical and physiological nature of muscle cells and whole muscles.

MICROSCOPIC STRUCTURE

A muscle cell is an animal cell and contains the same basic cellular organelles and parts you learned in junior high and high school.

Cell Membrane—The lipid bi-layer that separates individual cells. The membrane also allows certain substances to pass in and out of the cell. This characteristic is important in metabolism and during the electrical events leading to contraction.

Cytoplasm—Called the “sarcoplasm” in muscle cells, this is a water-based suspension containing other cellular substructures and metabolic substances. This is also the site of anaerobic (non-oxygen requiring) metabolism that supports high-intensity work.

Nucleus—The nucleus is the largest cellular organelle in mammalian cells and contains nearly all of the cell’s genetic material or DNA (deoxyribonucleic acid).

Mitochondria—Often called the “powerhouse” of the cell due to its role in aerobic (oxygen requiring) metabolism that supports lower-intensity and long-duration work. The set of reactions intimately involved in ATP (adenosine triphosphate) production—known as the citric-acid cycle (or Krebs cycle)—and the electron-transport system are located here.

Ribosomes—These organelles are the site of protein synthesis and are essential to life and exercise. Proteins in muscle create the ability to contract. Proteins in the blood carry oxygen. Proteins everywhere drive metabolism. Proteins are used to create architecture and function.

Endoplasmic Reticulum—There are two basic types of endoplasmic reticulum. You probably learned them in school biology: rough endoplasmic reticulum (having associated ribosomes) and smooth endoplasmic reticulum (having no associated ribosomes). In muscle, these organelles are involved in producing lysosomal enzymes, secreting cellular and membrane proteins, adding carbohydrate to proteins, synthesizing lipids, and metabolizing carbohydrates. The endoplasmic reticulum is also a regulatory center for calcium-ion storage. The large stores of calcium within the sarcoplasmic reticulum are
vital to muscle contraction and can be rapidly released into the sarcoplasm, which, in turn, initiates contraction in muscle cells.

**Golgi Apparatus**—This organelle is composed of membrane-bound vesicles. The Golgi apparatus works in conjunction with the endoplasmic reticulum by modifying outgoing substances for delivery to their intended destination, both inside and outside of the cell.

There are some other very interesting and functional microscopic structures that provide muscle its capabilities. These derive from the nature of the proteins that comprise the actual contractile apparatus. There are two key contractile proteins that enable muscle to contract. They are:

1. Actin
2. Myosin

These proteins form the business part of the muscle cell, where contraction and relaxation takes place. Two sets of myofilaments—one thick, the other thin—are arranged in alternating lines, with two identical sets longitudinally across from each other, separated by a central space. Each set of myofilaments thus arranged is bound to another set of proteins that make up the Z-lines—one at each end of the assembly. One entire unit, from one Z-line to the next, is known as a sarcomere (Figure 13).

Thick myofilaments are made primarily of the protein myosin and held in place relative to other myofilaments. Thin filaments are composed of the protein actin. Other regulatory proteins are bound to actin and act as regulatory proteins that moderate the interactions between actin and myosin.

Together and when appropriate, neural input creates the appropriate chemical conditions. The interactions of actin and myosin will create contraction. Simplistically, we can say that an electric nervous impulse hits the muscle cell; mineral ions such as calcium, sodium and potassium get moved around to spread the impulse throughout the cell; and a molecular reconfiguration allows actin and myosin to interact and bind to each other. This binding causes a shortening of the muscle cell that brings the two ends of the sarcomere (the muscle if considered in total) toward the midpoint.
MUSCLE AND TENDON STRUCTURE
Muscles and tendons are often thought of as separate structures; i.e., a muscle just has tendons tacked on the end to connect it to two bones. This isn’t the actual biological reality. The connective tissue that creates a tendon is present in the body of the muscle, too; we just don’t think of it as being a “tendon.”

Immediately surrounding each muscle cell there is a layer of connective proteins (like collagen) surrounding the individual cell. The connective layer around one muscle cell merges with that of its neighboring muscle cells (think of a box of toothpicks as the muscle cells). There is also a second layer of connective proteins that surround groups of between 10 and 100 muscle cells, then this layer further merges with the layer of connective tissue that covers the entire outside of the muscle. These layers of connective proteins are important, as they will have both structural and functional effects for tendons.

At the belly of the muscle, the ratio of muscle to connective tissue is quite high, with the volume of muscle being large compared to the volume of connective tissue. As you
near the ends of the muscle, the number of muscle cells present tapers off and the ratio of muscle volume to connective-tissue volume flips. When there are virtually no more muscle cells present, a tendon proper is present. Note that the length of transition from muscle to tendon can be quite different between muscles, as can be the length of tendons. The muscles of the skull have very short tendons, and the tendons of the tensor fascia latae run virtually the entire length of the lateral thigh.

This particular architectural arrangement between muscle and tendon allows muscle to generate force through the shortening of muscle cells and to transmit that force to the relatively non-stretchable connective-tissue network. When a muscle cell contracts, the shortening of its cell body length pulls the connective tissue attached to it along with it and toward the middle of the cell. This creates tension and a change in length that is transferred through the connective-tissue network and ultimately ends at the tendon-bone interface, where the bones are pulled closer together to produce body movement (a joint will be involved).

MUSCLE IDENTIFICATION

At this initial level of learning, we are forgoing the traditional meme of learning the muscles: learn the origin (the name of the bone and bony feature the muscle attaches to at its proximal end or ends), learn the insertion (the name of the bone and bony feature the muscle attaches to at its distal end or ends), and then learn the muscle’s function or functions (when it contracts and what parts of the body are moved).

Rather, here we are learning the muscle names and their geographic locations on the body. As you learn muscle names and locations, simply locate the muscle on your body, put your hand on it to reinforce your understanding of its location and then move the bits of the body around the muscle. By doing so, you’ll get a basic idea of what it does. Keep it simple for now. We would, however, recommend that later you more formally study the anatomy of the body to learn the nuances necessary for a functional mastery.

As a coach, you will never get to see the inside of the body to identify muscles. You have to rely on your ability to associate areas of the body with the muscles residing there. You will need to “see” the muscles without actually seeing them. Figure 16 illustrates this basic idea.
Figure 14.
Schematic of muscle-cell structure and contraction.
Figure 15.
Posterior muscle names and locations.
JOINTS AND MOVEMENT

If you look at a construction or painter’s scaffold, you’ll see that something connective is added everywhere two pieces come together at joints. Just like scaffolding elements, bones don’t and can’t do their job without some means of connection. Bones will invariably connect to one bone or several bones through a joint, and in some instances through multiple joints. The elements of a joint are the bones joined, the tendons of the muscles that cross the joint and the ligaments attached to the bones.
Figure 17.
The many movements possible at the shoulder.
Hip Circumduction

Abduction
Adduction
Internal Rotation

Extension
Flexion
External Rotation

**Figure 18.**
The multitude of movements possible at the hip.
It’s the elements of a joint that establish range of motion or flexibility. The majority of range of motion relies on the ability of muscle to stretch. We don’t want stretchy tendons, as that would inhibit the ability of muscle to transfer force to bones. Think of it as trying to pull a weight with a bungee cord versus a rope. Which is more efficient? We also don’t want stretchy ligaments, those dense collagen bundles connecting bone to bone. Ligaments are there to limit the maximum distance the bones of a joint can be from each other. They are safeguards from dislocation. In normal exercises utilizing normal flexion and extension of joints through complete ranges of motion, the risk of ligament damage and dislocation is low.

Some joint movements are easy to describe and understand, such as flexion and extension of the elbow, hip and knee. Extension opens the angle of the joint like opening a book. Flexion closes the angle of the joint like closing a book. But there are numerous other names for movements of the body beyond flexion and extension, such as opposition—the movement of your fingers to touch your thumb. Some terms can be confusing, as in the case of dorsiflexion and plantar flexion of the ankle and foot. Two flexions of the same anatomical structure? Sometimes you have to come up with clever ways to remember each. “Dorsi” refers to the back, so dorsiflexion is movement of the foot toward the back (lift your toes off the floor while keeping the heel on the floor). Plantar flexion is movement of the forefoot toward the ground (moving the heel upward while keeping the toes pointed down—like pushing down on a shovel or digging a hole to plant something). The following figures present common movements of the shoulder and hip.
SECTION 2
CARDIORESPIRATORY ANATOMY AND FUNCTION
The cardiovascular system is as essential to exercise as the muscle and bones. Without it, energy cannot be delivered, nutrients cannot be delivered, waste cannot be removed, body heat cannot be controlled, and more. Like the muscles and bones, the heart is adaptable, meaning that its structure can be changed with exercise for a performance or health benefit. Conversely, being sedentary can change its structure, and this can yield performance and health decrements.

We know exercising the heart is beneficial, but we also need to understand the heart in three perspectives:

1. How is the heart structured?
2. How does the heart function to support exercise?
3. How does exercise affect the heart?

HEART STRUCTURE

Everyone possesses a physically functioning heart that is architecturally the same, with only normal and minor variations between individuals. The majority of the heart is composed of muscle that encloses spaces. Essentially, these exist as chambers wrapped in muscle. The arrangement of the muscles around these chambers is important as the heart is a biological fluid pump, pumping blood to all points of the body (that have blood vessels). It pumps blood constantly from birth to death. If we consider the present average lifespan to be 76 years, that works out to 2,796,192,000 pumping actions (beats) over the lifespan. That is a lot of work. The energy required to drive blood through the vessels has been calculated to be 0.0023 calories (kcal) per each heartbeat. This equates to roughly 6,518,900 calories used over the lifespan just to pump blood. That’s about 235 calories per day spent on driving blood through the arteries and veins.

The human heart tips the scales at between half a pound and three-fourths of a pound (250 to 350 g). In general, the size of the heart is related to the size of the body containing it. A small body has a heart on the smaller end of the scale of norms, and a large body has a heart on the larger end of the scale. You can see examples of this in the animal world: some animals have hearts about the size of the end of your pinky, while an elephant heart is bigger than a basketball. A very quick and easy and somewhat accurate way to visualize the size of your heart is to simply make a fist. The fist is approximately the size of your heart. This is not an absolute and accurate measure but a ballpark estimation.

The human heart lies at about a 20-degree angle (downward, your right to left) just posterior to the sternum, somewhat centered within the ribcage (a little biased to the left). The big blood vessels coming out of its top will be toward the right and the narrowed tip of the heart will be toward the left. Its lowest excursion is above the level of the bottom of the sternum. Further, it is nestled in between the lungs, liver and diaphragm and endowed with some shock-absorbing and metabolically available adipose deposits (fat). It is a very well-protected organ.
CHAMBER ORGANIZATION

The heart is divided first into superior (upper) and inferior (lower) segments. The term “segment” is used here as the heart is not symmetrical and the term “halves” is not appropriate. The upper segment contains two small chambers called atria (singular = atrium). Each atrium has a superior entry opening and an inferior exit opening. The atria are fairly thin and look reminiscent of very small and partially deflated whoopee cushions. The function of the atria is to accept blood from the circulatory vessels and then move it with pressure into the lower segment of the heart. The short distances and large-diameter openings through which it moves blood allow for the light musculature present to function quite well. Both atria are subject to essentially the same work stress and have very similar structure.

The lower segment contains two much larger chambers called ventricles. Ventricles also have entry and exit openings but are much more heavily muscled than the atria. The thicker musculature is necessary for the ventricles to accomplish their pumping function. Both ventricles must produce higher forces than the atria for several reasons. Higher pressures are required to move blood greater distances upon emergence, and the blood must be pumped through smaller and smaller peripheral openings and vessels.

The cardiac mass is also divided into left and right segments, with each segment containing one atria and one ventricle. This is a functional and morphological division. The right segment composed of the right atria and ventricle is considered the pulmonary side. The pulmonary side receives oxygen-depleted blood from the body and routes it to the lungs for oxygenation. The left segment, composed of the left atria and ventricle, is considered the systemic side, receiving oxygenated blood from the lungs and delivering it to the body—all systems, hence the descriptor “systemic circulation.”

The right and left ventricles share a common muscular wall termed the ventricular septum. The remaining musculature forms the exterior cardiac walls. The exterior wall of the left ventricle is much thicker than that of the right ventricle. This is a functional consequence of the left ventricle muscle having to produce more force to move blood through the systemic circulation. This means the left side of the heart is the high-pressure side, capable of creating an average arterial pressure of about 100 mmHg (the same units of measure as barometric pressure) in order to drive blood out of the heart and to all parts of the body, whereas only about 25 mmHg of pressure is required to move blood through the pulmonary circulation.
The muscle present in the heart differs anatomically and functionally from the muscles that move the body (skeletal muscle). That is because the individual muscle cells (myocytes) differ in structure. A cardiac myocyte differs from a skeletal myocyte in shape and how it communicates with its neighboring cells, and it has only one nucleus (versus many nuclei in skeletal muscle).

**ELECTRICAL EVENTS OF THE HEARTBEAT**

Within the walls of the heart, groupings of specialized function cells conduct and direct electrical impulses through the heart, from the top of the atria down to and through the ventricles. This network of conductive cells moves those impulses in an organized geographic and sequential manner. This culminates in a sequential muscle contraction—atria then ventricles—that squeezes blood through the heart without allowing backflow.

The electrical activity driving muscle contraction can be recorded and visualized—it’s the electrocardiogram (EKG or ECG) that’s part of your physical checkup or seen on your favorite medical TV show. Electrical activity must precede contraction; it is what stimulates the heart to beat.
There are seven commonly accepted components to the cardiac electro-conductive pathway: sinoatrial node, inter-nodal tract, Bachmann’s bundle, atrioventricular node, bundle of His, right and left bundle branches, and Purkinje fibers.

Figure 2.
The conductive elements of the heart depicted in respect to their locations to other structures.

In a normal and healthy heart, the stimulus to begin the cardiac cycle of contraction begins at the **sinoatrial node**. The sinoatrial node stimulates both atria to contract and its impulse passes to the **atrioventricular node**.

The atrioventricular node lies essentially between the bottom of the atrium and the top of the ventricle. It acts as a sort of anatomical timer, allowing atrial contraction to occur before sending the impulse on to the ventricles.

The atrioventricular node leads next to the **bundle of His** below it. The pathway runs from the node into the muscle between the ventricles, and then it divides into the right and left **bundle branches**, which direct themselves and their offshoots to the right and left ven-
tricles, respectively. These offshoot branches are called Purkinje fibers, and they branch and divide extensively down the length of the septum between the ventricles and then go up and around the exterior ventricular walls. This arrangement creates an enveloping conductive network that permeates the ventricular muscle. This creates a nearly instantaneous ability to spread the contractile stimulus throughout the large ventricular muscle mass, both right and left, so it can contract as a single unit.

It is this electrical organization and network that produces the sequential and separate contraction of the atria and ventricles; the blood can then be moved in a controlled and directional manner that is responsive to biological demand.

BLOOD FLOW THROUGH THE HEART

We will start the description of how blood travels through the heart on the right side—the pulmonary side. The right side of the heart receives deoxygenated blood from the body into the right atrium. The blood is delivered by the superior and inferior vena cava and collects into the coronary sinus—a sort of juncture between the superior and inferior vena cava—and passes through a one-way valve into the atrium.

After atrial pressure changes (goes up) and the valve closes to prevent regurgitation (backward blood flow rather than vomiting), the blood is pushed through contraction to another valve and into the right ventricle.

The blood from the right ventricle is destined for oxygenation in the lungs via the pulmonary artery (the pulmonary circulation). The chamber will fill, and then after ventricular contraction, the blood will exit the right ventricle through another valve and make its way to the lungs. Once the blood passes through the lungs (where it picks up oxygen and drops off metabolic waste), it returns to the heart by way of the pulmonary vein. It is worthwhile to note that the pulmonary artery and pulmonary vein are exceptions to the rule that arteries carry oxygenated blood away from the heart and veins carry deoxygenated blood to the heart. They have opposite roles to their naming convention.

The oxygenated blood exits the pulmonary vein (through another one-way valve) and enters directly into the left atrium. Once full, the pressure within the left atria rises and forces open another valve, and blood moves down into the left ventricle. Rising pressure in the full ventricle shuts the entry valve and opens the exit valve, and the blood leaves the heart through the aorta.

The aorta is the most important artery in the body in terms of blood delivery. After emerging from the heart, it arches back toward the vertebral column and becomes known as the thoracic aorta, and then the abdominal aorta as it passes through those cavities, respectively. All the important appendicular arteries (like the subclavian and iliac arteries) and visceral arteries (like the hepatic and mesenteric arteries) are derived from the aorta. The majority of the blood leaving through the aorta is destined for the systemic circulation, but a portion of it is immediately redirected from the aorta into the coronary circulation. The coronary arteries are those that feed into the cardiac muscle and keep the heart
supplied with everything it needs. These are also the arteries where heart attacks occur (through blockage).

**Figure 3.** The route of the blood through the heart.

**EXERCISE AND HEART STRUCTURE**

The heart is a muscle, and like skeletal muscle, it can adapt to being loaded; conversely, it can adapt to being unloaded. Where any muscle is concerned, one important adaptive possibility is hypertrophy, the anatomical enlargement of a cell or tissue. Historically, hypertrophy of the heart has been considered to be a negative adaptation—a pathological condition that is damaging to function and health. Modern diagnostic tools are much more informative, and we now know better that not all cardiac hypertrophy is bad.

There are three basic types of hypertrophic alterations to cardiac anatomy: pathological hypertrophy, eccentric hypertrophy and concentric hypertrophy.

Pathological hypertrophy is characterized by an increase in the size of the heart—specif-
ically the left ventricle. In this disease process, think of the left ventricle as an uninflated balloon. Something occurs in the heart that induces the space inside the ventricular chamber to get bigger (a number of things can cause this). Now think of what happens when you blow up a balloon. As the balloon gets bigger, the walls of the balloon get thinner and thinner. In pathological hypertrophy this happens, too, only the dimensions of the heart get bigger. There is no added muscle mass, and the muscle walls thin. This is a problem as it corrupts pumping efficiency.

Eccentric hypertrophy—or runner’s heart—is a beneficial adaptation that allows the heart to more efficiently pump higher volumes of blood through the working body. In this scenario, the overall heart mass does get larger, driven largely by increased size of the left ventricular chamber (to hold and pump more blood) and the addition of the needed muscle mass to keep the ventricular wall at normal thickness.

Concentric hypertrophy is also a beneficial adaptation that allows the heart to move blood throughout the body against high pressures of resistance. High resistance pressures occur anytime there is an occlusion of blood vessels. This happens during high-intensity and high-resistance exercise and work conditions. Here, the chamber size stays essentially the same size, but there is muscle mass added to the left ventricular walls that increases their thickness.

**VASCULATURE**

The heart is connected to the vasculature: the arteries, arterioles, capillaries, venules and veins. These structures are so thoroughly connected that the term “cardiovascular system” is the standard rather than cardiac and vascular systems. Given the importance of these systems to life and movement, we need to establish an appreciation of their basic structure.

Arteries carry oxygenated blood away from the heart (note the pulmonary exception mentioned earlier). Arteries can be huge—larger in diameter than your thumb—as in the case of the aorta. There are also small arteries, down around 100 mm in diameter (such as the coronary arteries). Below that diameter, the vessels are called arterioles. Once the arterioles get as small as possible, they become capillaries. These vessels are only microns in diameter, big enough to allow passage of only one erythrocyte at a time.

The analog to an artery is a vein. Veins carry deoxygenated blood toward the heart (note the pulmonary exception mentioned earlier). Arteries occur in pairs with veins. A vein that pairs with an artery at any given location is a little larger in diameter than the artery. The larger veins mean they have a lower pressure inside them, allowing the blood from the higher-pressure systemic arteries to flow into them. The right atrium has the lowest pressure in it than all of the circulatory elements so the venous blood continues to flow down the pressure gradient into the right atria, where it begins the cardiac pressure cycle again.
Blood vessels are adaptable to stress. If there is a hypoxic stress (low oxygen content present), it results in tissue hypoxemia (low oxygen in a tissue), a cascade of local hormonal and anabolic events occur that produces new capillaries and new arterioles. This process is called angiogenesis. It can happen in the heart and it can occur in skeletal muscle. This is considered to be an endurance-friendly anatomical adaptation, leading to improved capacity to deliver oxygen to working skeletal muscle. Angiogenesis from exercise also provides a survival benefit in the heart muscle. A more expansive vascular bed from angiogenesis gives the heart muscle more tubes to draw its blood from. If one route of blood delivery is blocked, a newly created collateral vessel or vessels can deliver the needed blood to the heart tissue. Think of this as a traffic detour off an existing highway after a wreck, where the cars backed up behind the wreck get redirected to other roads but still get to the intended destination. The creation of collateral blood vessels through exercise-driven angiogenesis does not prevent disease processes from occurring. Things such as atherosclerotic progression can and do occur, but angiogenesis can explain, in part, why fit individuals experience myocardial infarctions far less frequently than sedentary individuals.

INTEGRATION WITH THE RESPIRATORY SYSTEM

Another system that is closely entwined with the cardiovascular system is the respiratory system. They are so intimately aligned in structure and function that they are often merged into a single reference term, the cardio-respiratory system. We should be familiar with a number of elements of the respiratory system.
THE LUNGS
We can’t see them, we can’t touch them, but we certainly can feel them when we are training really hard. And they are certainly important to exercise performance in training and in competition. The lungs are intimately intermeshed with the heart and vessels in an elegant system of supply and demand. This is a system that delivers a critical element of life and exercise: oxygen. In most instances when we are hanging around and going about our daily business, and even when we are following physicians’ recommendations for exercise (low intensity, long duration), this elegant system carefully matches the amount of oxygen delivered to the amount consumed—supply meets demand. Under these conditions, we are using aerobic (oxygen requiring) metabolism to supply the energy needed to power these low levels of physical activity. With aerobic exercise, ventilation rate (how many times we breathe per minute) can increase about 300 to 400 percent during sustained movement.

If we amp up our exercise intensity—running at near-sprint speeds or increasing in exertional effort—ventilation rate can exceed baseline by more than 500 percent. But even that accelerated rate of breathing cannot provide the aerobic metabolic machinery with enough oxygen to keep pace with the demand. As a result, there is a transition to anaerobic (non-oxygen requiring) metabolism. Shortly after the transition to anaerobic metabolism, the body will fatigue, and exercise will have to slow to an aerobic pace or cease completely. Anaerobic metabolism is a short-lived source of power.

So no matter what type of exercise we do—low to high intensity, long and slow to short and fast (and everything in between)—the lungs are critical anatomical structures. Of course, the lungs are important even when we are not exercising at all.

EXTERIOR ELEMENTS
Humans have two lungs. Each is divided into segments called “lobes,” which are defined by indentations or creases intruding into the exterior surface of the lung. The left lung has two lobes and the right lung has three lobes. The lungs sit in the thoracic cavity, bounded in front, behind, to the sides and on top by the axial skeleton (ribs, sternum and vertebral column). The thoracic cavity is bound at the bottom by the diaphragm. As the diaphragm is attached to the contour of the lowest ribs, the lungs can extend no lower. The rigid structure of the axial skeleton and the moveable nature of the diaphragm enable the respiratory function of the lungs (moving air in and out).

There are other elements of the respiratory system that help move atmospheric air into the body. Air first enters through the mouth or nasal passages, passes through the larynx, goes through the trachea (windpipe), divides into the right and left primary bronchi, then it enters the lungs about one-third of the way down their length—roughly at the mid-sternum level.

INTERIOR ELEMENTS
The primary (exterior) bronchi do not feed into the lungs as most people usually visualize—as straws feeding into balloons or big hollow sacs in the chest cavity. This is most definitely not the case. The inside of the lung is filled with millions of membranous sacs
and hundreds of miles of small blood vessels. If you poke a lung, it is not like poking a balloon, it is more akin to poking a big, warm marshmallow.

The membranous air sacs within the lungs are called “alveoli.” There are about 300 million in a human lung, each sac about one-third of a millimeter in diameter. This provides an extremely large surface area, working out to be approximately the size of a tennis court if spread onto a flat surface. You can calculate this yourself with simple algebra: $300,000,000(4\pi r^2)$. Capillaries cover almost the entire alveolar surface and do so in such a complete and thorough fashion that it is almost like a sheet of blood covering the surface of each alveolus. An extremely thin barrier (0.3 μm or 1/84,667th of an inch) lies between these alveoli and the capillary blood; it is known as the “blood-gas barrier.”

The chemicals present in the barrier allow oxygen to easily diffuse into and carbon dioxide to easily diffuse out of the capillary blood stream. This is important because the heart pushes all our entire cardiac output through the pulmonary capillaries and past these alveoli, where the blood must upload oxygen and offload carbon dioxide.

After a primary bronchus enters a lung, it branches extensively, with the tubes getting progressively smaller—macroscopic to microscopic—with every sequential division. There are over 1,000 miles of air-conducting tubes in the lungs. Think of them as the piping to move air in and out.

The nuts and bolts of all gas exchange can be found near the end of the divisions of the respiratory tree. Ninety percent of the exchange occurs at the alveoli, clearly demonstrating their importance. The remainder of the exchange occurs in the respiratory bronchioles and alveolar ducts, the last tube-like structures before the alveolar sacs.

ADAPTATIONS

Most exercise programs create respiratory adaptations that allow us to do more work or to work faster. One ubiquitous adaptation is in the form of improved vital capacity of the lungs.

Increased vital capacity results from virtually every exercise system, from yoga to running to weightlifting and more. The additional respiratory work done during training increases the efficiency of breathing so you can draw more air into the lungs and move more air out of the lungs (vital capacity is the maximal amount of air the lungs receive in and then push out in a respiratory cycle). The inspiratory muscles get better at pulling in air, and the expiratory muscles get better at pushing air out, so a larger percentage of the total space in the lungs gets filled up and then emptied—residual volume of air in the lungs after exhalation goes down. The more demanding the exercise system used, the larger the gains will generally be, but there is an anatomical limit of lung volume in every individual—a top end of improvement that cannot be exceeded. Some systems just enable you to reach that anatomical plateau sooner.

Refer to The Paradox of Aerobic Fitness Prescription for a larger consideration of cardiovascular and cardiopulmonary fitness.
SECTION 3
KIDNEY ANATOMY AND FUNCTION
Everyone sweats. Everyone pees. If not, they are in a world of hurt.

It may not be readily apparent, but there is commonality in the two watery products above: The kidneys control them through their abilities to regulate the amount of water in the body. The kidneys work to control the “purity” (for lack of a better word), the dissolved constituents in body fluids and the consistency of fluid volume within the body (fluid homeostasis). To accomplish these important duties, the kidneys filter gallons of fluid from the bloodstream, rid the body of metabolic wastes and eliminate excess mineral ions produced as a result of physiological processes. They also create, recycle and return needed substances to the circulation. In the average person, this means the kidneys will process and filter about 50 gallons of blood a day (about 190 L) and remove about half a gallon (1.9 L) in the form of urine.

It is important for fitness professionals to understand the basic functions of the kidney and know some detail of the concepts of fluid balance and hydration.

ANATOMY OF THE KIDNEY AND RELATED STRUCTURES

The kidney is part of the urinary system. This system is composed of the kidneys, ureters, urinary bladder and urethra.

**Kidneys**—The kidneys are two bean-shaped organs (ever heard of kidney beans?) that act as filters of blood. They also produce urine. They lie to the right and left of the vertebral column in the abdominal, cavity approximately just below the 11th and 12th thoracic vertebra and under the diaphragm. The kidney itself is divided into two layers: the outer renal cortex and the inner renal medulla. The cortex contains the beginnings of about a half million tiny tubular and vascular units of filtration: the glomeruli. The remaining portion of the glomeruli penetrates deep into the medulla, which essentially functions to collect the filtrate into the collecting duct, which exits the kidney to the ureters.

**Ureters**—About 10-12 inches long, these are thin tubes leading from the kidneys down along and flanking the vertebral column to the urinary bladder. The ureter walls are lined with smooth muscle, and the action of the smooth muscle actively conducts urine produced in the kidneys to its temporary storage in the urinary bladder.

**Urinary Bladder**—The urinary bladder is the point of urinary collection before urine is disposed of by urination. The bladder is a hollow muscular sac. It is quite distensible and can hold approximately 350-500 ml of urine (12-16 oz. or a standard beverage can). The bladder sits on the pelvic floor, generally lying between the rectum and the pubic symphysis. Before urine can be expelled, two sphincter muscles—the involuntarily controlled internal sphincter and the voluntarily controlled external sphincter—must be relaxed. Once the sphincters have relaxed, urine exits the bladder to the urethra.

**Urethra**—The urethra is a tube that conducts urine from the urinary bladder to the external environment. It is approximately 2 in. (5 cm) in length in females and approximately 8 in. (20 cm) in length in males. There is a urethral sphincter that allows further voluntary control over urination.
MAJOR FUNCTIONS OF THE KIDNEY
The kidneys serve several essential regulatory functions in humans:

1. Elimination of nitrogenous wastes (nitrogen-containing molecules such as ammonia and urea) and other toxic materials from the body.
2. Reabsorption of glucose and amino acids for reuse within the body.
3. Production of hormones—erythropoietin (red-blood-cell production), renin (blood-pressure regulation), calcitrol (vitamin D metabolism).

WATER IN, WATER OUT
We’ll consider part of the fourth major function—maintenance of water balance—in a bit of detail. The regulation of fluid balance begins in the glomerulus. These tiny units are the heart of filtration and urine formation. The basic parts of the glomerular apparatus and
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their part in the process are:

1. Glomerulus: a tuft of capillaries that perform filtration.
2. Bowman’s capsule: an enlarged, cup-shaped and incomplete capsule surrounding the glomerulus.
4. Loop of Henle: sodium and water balance.
5. Distal convoluted tubule: tubular secretion.

Glomerular filtration is the first stage of urine formation. It is a passive and nonselective process where fluids and small dissolved materials are pushed through a membrane by hydrostatic pressure—the pressure inside the glomerulus is higher than outside the glomerulus. The net effect is that water and solutes (ions and small molecules) can pass out of circulation for inclusion in urine. Larger blood-borne materials such as erythrocytes, leukocytes, platelets and plasma proteins cannot pass through the exclusionary membrane and are retained in circulation. An increase in blood pressure increases the rate of filtration, and dehydration slows the rate of filtration and urine formation. So, exercise per se increases the immediate rate of urine formation through exercise-induced hypertension unless significant dehydration occurs as a result of sweat loss over a long exercise bout during which environmental conditions favor sweat production. This latter occurrence would act to counter the exercise effects and potentially reduce filtration rate and urine formation.

The kidney is often thought to have an auto-regulatory mechanism in operation that maintains a constant glomerular filtration rate in spite of fluctuating arterial blood pressures. At rest and with very low levels of work, this is correct. However, with continued heavier levels of sport training, it has been noted that resting glomerular filtration rate is affected through a statistically significant reduction.

There are two processes active in urine formation:

**Active Tubular Reabsorption**—This is a process that reclaims molecules such as glucose, vitamins, ions (especially sodium ions) and amino acids for circulation in blood. It acts against the concentration gradient present in the glomerulus. The presence of carrier or receptor molecules with high affinities for filtered molecules drive the process, but once those molecules are saturated (can’t bind to any more solutes), any excesses will be excreted in urine.

**Passive Tubular Reabsorption** – Sodium concentration differences created by active tubular reabsorption create a strong osmotic gradient, and this causes water to move out of the filtrate and back into the blood. When water leaves the tubules, the relative concentration of any substances still present in the fluid inside the tubule increases. If the concentration gradient (difference in concentration inside and outside) is large enough, dissolved materials will follow the gradients out of the tubule.

The loop of Henle is one particular part of the glomerular tubule that is important to fluid balance. The descending part of the loop is impermeable to sodium; as water exits,
relative sodium-ion concentrations inside the tubule increase. In the ascending part of the
loop, the tubule is permeable to sodium but impermeable to water. In this portion of the
loop, sodium leaves the tubule by diffusion, and the remaining fluid inside of the tubule
becomes more diluted. Minor hormonal and electrolyte actions regulate how much/
many mineral ions are inside and outside the loop and thereby control how much water is
retained or eliminated from the body.

Not every substance dissolved in blood or that is filtered out ends up being reabsorbed.
Many are not reabsorbed or are only partially reabsorbed. These would include metabolic
and nitrogenous products of protein and nucleic-acid metabolism, such as urea, creatinine
and uric acid. Potassium ions are also eliminated if they are present in excess amounts. If
the body’s pH is too low (too acidic), hydrogen ions are included in excretions.

WATER BALANCE

Water balance is simply homeostasis. The amount of water consumed in food and drink
and generated by metabolism must equal the amount of water excreted in order to
maintain healthy function. There are biological and behavioral influences on how much
water we consume (thirst) and how much salt we consume (salt craving). The kidneys have
a role in maintaining this balance. In broad, basic strokes, they do this by conserving water
(ensuring less is removed from the body), producing urine that is concentrated (less water,
more solutes) relative to blood plasma, or by removing water from the body by producing
urine that is dilute (more water, less solutes) relative to blood plasma.

The major biochemical player in this process is arginine vasopressin (AVP, also known as
anti-diuretic hormone or ADH), which, when present, induces water reabsorption. When
the hypothalamus in the brain senses blood is too concentrated, AVP is released. This pulls
water back into circulation. In opposition, when there is a large returning fluid load to the
heart, AVP production is inhibited. This allows water to be removed from the system.

Sodium (of sodium-chloride fame) is the single largest solute in extracellular fluids, so
this single molecule determines the amount of solutes per unit volume of body fluids
(increases salt concentration in the blood). This ratio is tightly controlled, as it is critical
for most life functions (think about the importance of sodium in propagating the electro-
ical signal in muscle contraction, for example). This implies that sodium balance must be
closely regulated to facilitate function or prevent destruction of cellular structures and
disruption of cell and tissue functions.

In practice, when you train long and hard in a hot environment or just don’t drink enough
water, your body becomes dehydrated. You lose water from the circulation to form sweat
in the former instance, and water consumption does not meet the rate of elimination in
the latter. In both cases you are losing proportionately more water from the blood than
losing sodium (the primary solute of interest). This increases osmolarity. In this situa-
tion, the kidneys will act to conserve water in order to stem further loss of water and the
resultant increase in sodium concentration (note that no new sodium is being put into
the system). It should be obvious that this is a survival mechanism; the body is naturally
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protecting itself. Further, the body is quite capable of functioning with transient low-water intake (or transient no-water intake); the correction to the situation is consumption of appropriate amounts of water when it becomes available.

HOW MUCH WATER IS APPROPRIATE?
If you are like most people, you were told in school, at home, in the doctor’s office or on TV to drink eight 8-oz. glasses of water per day (remember that the average U.S. drinking glasses for home use are 12, 16 and 20 oz.). Conveniently, this works out to the half-gallon (1.9 L) of urine the average person would excrete each day. While this seems like a great one-size-fits-all prescription, it really isn’t: This concept has never been tested. It derives from an unsupported recommendation in an archaic nutrition textbook. Does a 110-lb. (55-kg) exercising female need the same volume of water consumption as a 330-lb. (150-kg) exercising male football lineman? Of course not. The need for water is driven by metabolic rate and magnitude. The football lineman uses a much larger volume of water to support the biochemical processes that must occur to support his large mass. Even at rest, the basal metabolic rate of these two example individuals differs by two- or threefold.

So how do we approach this problem? Not enough water can have negative effects on performance. Ask any weight-class athlete about how they perform after cutting weight, and they will tell you performance is not up to its potential. But dehydrated athletes have set world weightlifting and powerlifting records, they have won gold medals, and they have completed marathons. Although dehydrated cells do not function optimally—as all metabolic processes take place in water—the body can continue to function near its current level of readiness.

Fortunately for most gym goers, dehydration is never a problem. There are always drinks available if needed, but the real reason it’s not a problem is simply because the average training session is not long enough to cause dehydration.

Let’s break this down into two questions:

1. How much water do we need to lose before performance suffers and bad things happen?
2. How long would a workout have to be in order for dehydration to occur and exert effects?

How much water do we have to lose before issues appear? It really depends on your body weight. The following continuum shows the percentage of body-weight loss needed to cause decreased performance, acute health problems or death. Note that a gallon of water weighs 3.8 kg or 8.3 lb. as you peruse the continuum, and put that figure in perspective with regard to your own body weight. A 200-lb. male, for example, would have to lose almost 4 gallons of water (about 32 lb., or 16 percent of body weight) to be at risk of death.
Figure 2.
Dehydration Continuum. The continuum assumes that you are completely hydrated—bladder emptied, and bowels voided—as a baseline, and that the water loss is a result of short-term water restriction and exertion, not as part of a pathological pre-existing condition. Note that this is a continuum, and that effects and symptoms gradually appear as dehydration worsens. Individual differences should be expected.

How long does a workout have to be to cause an athlete to lose 5 percent of body weight? Can you even lose 5 percent in a single workout? It depends. The biological safeguard of thirst kicks in by about a 2 percent loss. So if you can ignore the signals your body is sending you, and you can refrain from drinking even though your body is telling you to, and you can continue exercising for long enough, you can lose 5 percent of your body weight. Research tells us that in extreme instances a human performing severe exercise in severely hot and humid environments can lose 1.5 L (0.4 gal.) per hour. If we put this into the context of the continuum above, to reach 5 percent dehydration, a fully hydrated person would need to avoid drinking and perform near-maximal exercise in high heat for between 80 and 210 minutes (an hour and 20 minutes up to three-and-a-half hours), depending on body weight. Recall that 5 percent is where performance losses and symptoms begin to emerge. Reaching the more profound levels of dehydration would require double and triple these durations and would only be a factor in very extreme and long-duration events.

If your training session is less than 30 to 60 minutes in duration, in any environment, there is no advantage or benefit from drinking during the session, nor is there a danger from not drinking. Perhaps the best use of water in this instance would be to douse yourself to get your body temperature down (if you were outside in the heat).

**SOME CONSIDERATIONS**
Conventional wisdom and defensive medicine over the past few decades have led us to the point where the populace, fitness professionals and even clinicians believe fluid consumption should occur before training, during training and after training. Essentially,
overconsumption is promoted. The guidelines responsible for these beliefs were putatively developed to defend against some rather dire heat-injury problems induced by dehydration. In normal training sessions, even in the heat, hydration status does not have a strong correlation to heat injury. Adding water to the gut of a heat-injured person does not cure the problem; getting body temperature down by any means necessary does.

Conversely, too much water can carry with it a separate set of problems. The kidneys can easily deal with small-to-moderate excesses in water consumption. However, significantly large and excess fluid intake can lead to hyponatremia (low sodium content of the blood and tissues). This particular pathology was once a very infrequent finding in sport and exercise. There appears somewhat of a correlation between increased reports of hyper-hydration and hyponatremia and the evolution of hydration standards into the current recommendations from exercise-professional groups. Historically, these guidelines have heavily recommended that you drink before you are thirsty (disregard a physiological survival mechanism), drink consistently during exercise and continue to drink afterward.

Hyponatremia is most frequently found in running participants who run at a slow pace, who by virtue of lower exertion (slow pace) or environmental conditions sweat less, and who drink water frequently before, during and after running long (marathon-scale) distances. If we take the guidelines for pre-hydration and hydration during running as written without individualization and assessment of current hydration status (0.7 to 0.9 L in the four hours prior to running and 0.4 to 0.8 L per hour during exercise), an average marathon participant who runs the route in approximately five hours will have consumed 2.7 to 4.9 L of water by the end of the race. Potentially and conservatively, the runner might have sweated (in a hot environment) 3.75 L in that time (0.75 L per hour). So, about half the runners could be over-hydrated using the current guidelines.

Because average exercisers do not weigh themselves each morning and calculate potential water loss, nor do they check for urine osmolality (a laboratory check for hydration status), they do not have a means to monitor and adjust their consumption to alter hydration status. As such, most trainees simply follow the advice of the authoritative bodies and drink continually before, during and after exercise, placing themselves at risk of over-hydration. We as exercise professionals can help them avoid the risk of under- or over-hydration simply by getting them to follow the biological imperative that has kept us alive throughout human history: thirst. **Having drinks readily available to trainees when they are thirsty** is a safe and effective way to maintain and improve performance without the risks of under- and over-hydration.

For a more detailed analysis and explanation of the science and issues surrounding hydration read “Waterlogged” by Dr. Tim Noakes.
ANATOMY
“Gray’s Anatomy of the Human Body”
http://www.bartleby.com/107

PHYSIOLOGY
“Human Physiology”
http://en.m.wikibooks.org/wiki/Human_Physiology

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