UNIVERSITÀ POLITECNICA DELLE MARCHE

DEPARTMENT OF ENGINEERING

Master's Degree in Biomedical Engineering

Bioengineering of American Football: A Review

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ABSTRACT

American Football is one of the sports with the highest rates of injuries and head traumas. Additionally, there is epidemiological evidence that most American Football players are affected by hypertension. Linemen are subjects at risk of developing cardiovascular disease due to their weight and the isometric training. They appear to have hypertrophic cardiac adaptations and the consensus in the literature seems that this is not a positive adaptation as in the case of the common Athlete's heart. Given this premises, the main two lines of research are the biomechanics of football specific movement and in vivo cardiac monitoring. Studies regarding the biomechanics aims at understanding how to improve the movement of the player or how to prevent injuries during the specific movement. An important subset of these studies includes the biomechanics of concussion with the aim of prevention and recognition of concussive events. For what concerns in vivo cardiac monitoring, the number of studies on population of American football players is limited and the aim of these studies is to improve the training protocol of athletes, to monitor their recovery status or even to monitor their temperature during the training. This thesis expands the research of in vivo cardiac monitoring in American Football by recording with a Zephyr Bioharness 3.0 the electrocardiogram signal and the heart rate of fifteen players. The participants had an average age of 23.67 ± 3.42, an average weight of 94.67 kg ± 14.69 kg and an average height of 1,82 m ± 0.07m. The aim of the experimental study was to understand the effect of repeated short maximal sprints on the sympathetic and parasympathetic activity of each subject by analyzing the tachogram extracted from the recorded electrocardiogram. After each sprint, a PLATEAU phase was recognized, and a symbolic analysis was applied to the PLATEAU sequence during the training session and to pre-training and post-training sequences. Eventually, the median and interquartile range was computed for each one of the symbols. In 14 subjects the median value of the 2UV% in the PLATEAU phase was higher than the one found in pre-training sequences suggesting an early vagal reactivation after each sprint. Additionally, during the post-training sequences 11 subjects had lower values of 2UV% and 10 subjects had higher values of 0V% when compared to pre-training sequences, which respectively suggest a suppression of vagal tone and an increase in sympathetic tone, which could be explained by the intensity of the training session.

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Introduction

Bioengineering is a field of engineering that wishes to solve problems in medicine and biology. The issues that bioengineers face are issues of clinical relevance, such as the design of medical devices or diagnostic equipment, the investigation of biomaterials and pharmaceuticals, the study of the underlying physiology with mathematical systems, the analysis of pathology through the interpretation of bio-signals. Being a part of the engineering world, the tools, techniques, and methodologies employed are coming from mathematics, physics, and computer science.

Sport Bioengineering is focusing on sports-related issues of clinical relevance. An application is the monitoring of the health status of the athletes during training to discover undetected complications or to simply improve their performances. American Football is a subject of particular interest due to the vast number of athletes playing it from the youth to the professionals. It is the most popular sport in the United States with around a million high school athletes and 70.000 collegiate athletes [1] . The main two areas of research are concerning the biomechanics of sport-related movements and the cardiovascular screening of athletes.

In the first case, the high rates of injuries in American Football when compared to other sports was the main motivation of the research. Therefore, a better understanding of the kinematics and dynamics involved during the typical movement of the sport could give us clues regarding the reason injuries happen and could enable us to prevent or limit the occurrence of injuries. This concept is particularly important concerning concussion, an injury due to head trauma that could result in the worst cases in later cognitive complications, such as mood and behavioural changes of the athletes. For this reason, a great portion of the research regarding American Football is interested in understanding what is the dynamic that led to a concussion and how to prevent it, limit its occurrence or recognise it properly.

On the other hand, the research on cardiovascular monitoring, even if limited in the number of published papers, is motivated by recent epidemiological findings which support the idea cardiovascular risk factors and pathological phenotypes are diffused in the population of American Football athletes.

Chapter 1. The sport of American Football

The following chapter aims at introducing the sport of football to the interested reader based on the International Federation of American Football (IFAF) rulebook [2], which is also the one followed in Italy by the FIDAF (*Federazione Italiana Di American Football*). The National Collegiate American Association (NCAA) rules for American Football are the basic set of rules used to form the IFAF rules.

1.1 General rules

American Football is a discipline where twenty-two athletes, eleven for each one of the two teams, play on a rectangular field and with a ball having the shape of a prolate spheroid. An example of the field provided by the rule book is in figure 1. The dimensions of the playing field are one hundred yards in length and about 53.3 yards in width, where an IFAF yard is defined as 91.44cm. On the two opposite short sides of the playing field, we can observe two end zones, delimited by the two sidelines, an end line, and a goal line, which are ten yards apart. The objective of each one of the team is to cross the goal line to score six points with a touchdown. At the middle of the end-lines we can observe a goal post.

This is used to score either the additional point after a touchdown with a field goal kick between the two bars or to score three points whenever the offensive team is close enough to the goal post to kick the ball between the bars instead of aiming at the touchdown. Additionally, after scoring a touchdown, the offensive team could decide to make a 2-point conversion by playing to cross again the goal-line instead of the 1-point conversion achieved by a field goal kick.

The game starts after a coin toss which decides what team will have the ball first and what side of the field the defensive team is going to defend. The first action of the game is the free kick, where the defensive team (kick-off formation) kicks the ball from its 35 yards line to the offensive formation (kick-off return formation). A kick-off returner will catch the ball and start to run behind the blocks of his other teammates to bring the ball closer to the opposite goal line, whereas the other team will run to tackle the returner. From the point where the ball carrier was tackled, the ball will be placed, and the offensive team will start to play. Each time a ball carrier is tackled or goes out of bounds the point of maximum advancement will be considered for the ball placement. In addition, the referees will establish a line of scrimmage, defined as the line crossing the ball and perpendicular to the two side-lines.

A snap, consisting of a lineman (Center) throwing the ball between his legs to another player being the quarterback or running back, puts the ball in play. Each offensive team has up to four consecutive

attempts, called scrimmage downs, to gain ten yards, after which the count of attempts will be renewed. During the downs, the offensive team can advance using running plays, during which the ball is handled by a running back, that rushes through the defense, or passing plays, during which the quarterback can make only one forward pass thrown from a point behind the line of scrimmage to an eligible receiver. If the offensive team does not reach the desired gain of ten yards in four downs the ball possession changes unless it decides to kick the ball for a punt or, if close enough to the goal post, to try a field goal kick for a 3-point score. The punt is a kick where the kicker (or punter) drops the ball and then kicks it before it touches the ground. The punt is used during the fourth downs to give possession to the other team in a more favourable field position.

1.2 Player's positions

In each team of American Football, we can find three basic subunits:

- Offense, which is composed of eleven players that have the responsibility of gaining yards and scoring touchdowns
- Defense, who is composed of eleven players that aim at stopping the opposing offense and take possession of the ball
- Special Teams, who intervene in occasions such as kick-off, kick-off return, punt, punt return, and field goals.

Another subdivision of roles can be based on the specific skills, the usual body composition, strength, and velocity of the players. We can distinguish between two categories:

- Linemen, who usually weigh more, are stronger and focus on isometric training in the form of blocking and pushing the opponent. The linemen are set near the line of scrimmage forming a line of players. In the case of offensive linemen, such as the center, the guards, and the tackles, the objective is to protect the quarterback from rushers during passing plays or to push the defensive linemen backward to gain yards during running plays. On the other hand, the defensive linemen, composed of defensive ends and defensive tackles, try to stop the quarterback or the running back.
- non-linemen or skill players, who are leaner, faster, and have less absolute strength compared to linemen. Offensive skill players are receivers, running backs, and quarterbacks, whereas the defensive skill players are cornerbacks and safeties. Linebackers, who are defensive players positioned behind the defensive linemen, are usually considered in between these two classes having the characteristics of both [3].

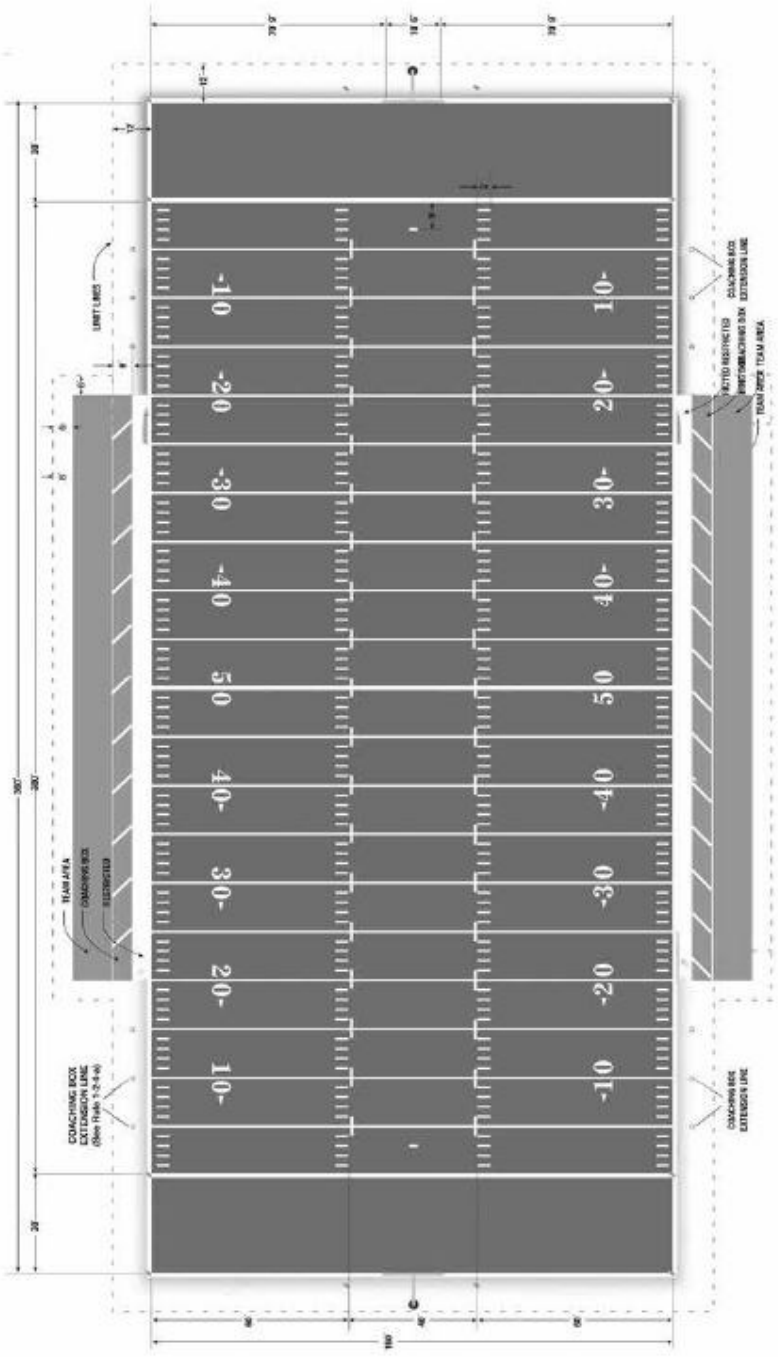

Figure 1. example of the American football field provided by [2]

Chapter 2. The cardiovascular system and American Football

2.1 Anatomy of the Heart

The heart is a specialized muscle [4], which is asymmetrically positioned slightly to the left side of the chest with an inclined orientation. The apex of the heart, the lowest point, goes to the left. The right part of the heart, formed by the right atrium and ventricle is more frontally oriented than the left.

The heart is formed by the right and left hearts, which are respectively responsible for the pumping mechanism of the blood in the pulmonary circulation, which brings blood to the lungs, and in the systemic circulation, which brings blood to the peripheral organs. The rhythmic contraction of the heart is a consequence of a precisely ordered sequence of contraction of the four different heart chambers, atria, and ventricles, communicating through valves [4] [5].

In particular, the right atrium gathers blood from the systemic circulation thanks to the vena cava superior and inferior. Then, blood flows to the right ventricle through the tricuspid valve and it is

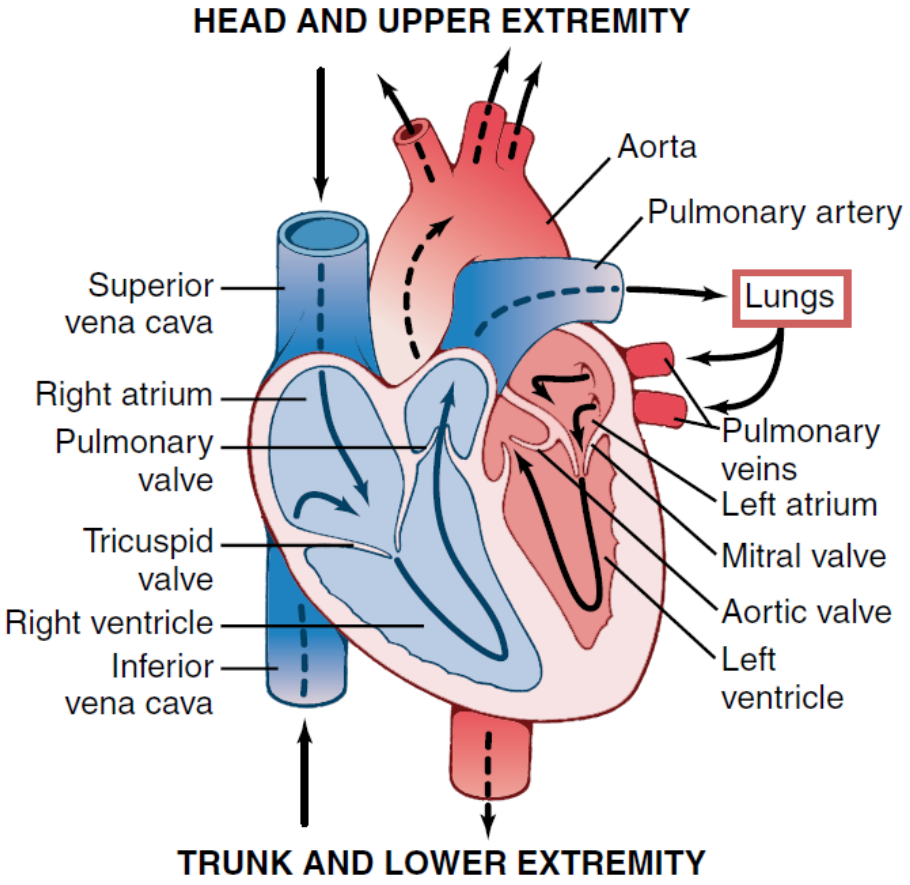

Figure 2. Structure of the heart [4]

expelled to the pulmonary arteries passing through the semilunar pulmonary valve. Thereafter, blood coming from the pulmonary circulation is collected in the left atrium thanks to pulmonary veins and it is passed to the left ventricle through the mitral valve. Finally, the left ventricle expels the blood into the systemic circulation via the aorta across the aortic valve (figure 2). Thus, the atrium works as a prime weaker pump that pushes the blood into the stronger pump being the ventricle [4].

The presence of chordae tendineae, attached to papillary muscles placed at the ventricular walls, constitute a mechanism of prevention of blood leak and blood backflow in the atrioventricular valves. These muscles contract with the ventricles increasing the tension on the cusps of the valves during the ventricular systole [5].

2.1.1 Cellular structure and function

Cardiac cells or myocytes are mononucleated and striated muscle cells. A major difference that distinguishes myocytes from skeletal muscle cells from a structural point of view is in their dimensions. Myocytes appear with a diameter of approximately twenty-five micrometers and a length of about one hundred micrometers whereas skeletal cells have similar diameters but longer lengths in the order of many centimeters [5].

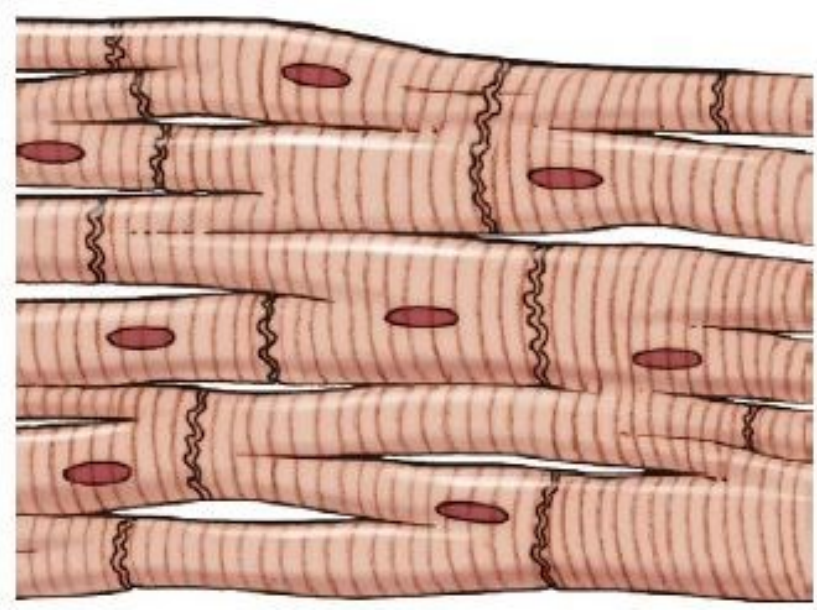

Figure 3. Structure of the cardiac myocytes, interconnected by intercalated disks [4]

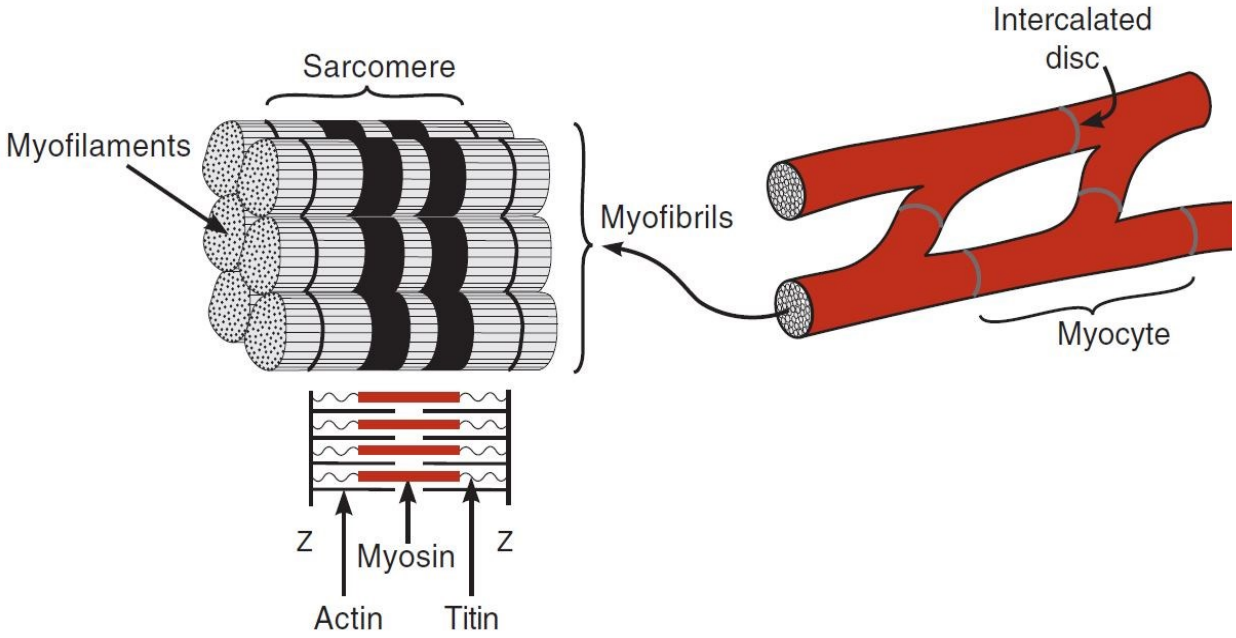

Figure 4. Microscopic structure of myocytes, formed of myofibrils [5]

One of the most important characteristics of myocytes is that they work as a functional syncytium because they are mechanically connected thanks to desmosomes and electrically connected thanks to gap junctions, forming in this way the intercalated disk as shown in figure 3 [4] [5]. Therefore, any spontaneous action potential generated in a certain site of the heart can propagate through cardiomyocytes. This aspect of the cells of the heart enables a proper contraction.

From a microscopical point of view, it is possible to observe that cardiac myocytes are formed by groups of myofibrils, composed of smaller myofilaments, and subdivided into elementary contractile units called sarcomeres, which are confined between two Z lines (figure 4) [5]. Each sarcomere consists of:

- thick filaments composed of about three hundred molecules of myosin, which is a large protein characterized by two heads that interact with the binding sites of actin during the process of contraction
- thin filaments positioned in a hexagonal arrangement around the thick filaments and formed by actin, tropomyosin, and troponin regulatory complex. Actin is a globular protein, and it interacts with the tropomyosin, a rod-shaped protein, which covers the binding site of actin. The troponin regulatory complex reacts to Ca^{++} binding to its subunits to regulate the position of the tropomyosin and to free up the binding sites of actin for the contraction mechanism described in section 2.3.

- Titin, a large filamentous protein that enables the myosin molecules to be centered in the sarcomere

During contraction, the sarcomere shortens causing the distance between Z-lines to reduce and the thin filaments to slide over the thick ones.

The membrane surrounding the bundle of myofibrils, called the sarcolemma, has deep invaginations called transverse tubules or T-tubules, which enables better communication with the external environment and more efficient exchange between intracellular and extracellular ions. In proximity to the T tubules, it is possible to find the sarcoplasmic reticulum, a structure that is responsible for the storage and the regulation of calcium concentration inside the cell [5].

2.2 Electrical physiology of the Heart

2.2.1 The resting membrane potential

Cardiomyocytes have an electrical membrane potential, which is the potential measured between the outside of the cell considered at 0mV by convention, and the inside of the cell; at rest, this potential is equal to -90mV and it is negative due to the concentration of negative proteins and molecules inside the cytosol. The usual concentration of potassium, sodium, and calcium ions can be seen in figure 5; the mechanism of diffusion of these three ions is the one that determines changes in the membrane potential [5].

The resting membrane potential (E_m) is related to the concept of the equilibrium potential E, which is the needed membrane potential to oppose the diffusion movement of a particular ion due to its concentration gradient; it can be computed using the Nernst equation, which relates E to the concentration of the particular ion inside the cell (I_{in}) and outside the cell (I_{out}):

$$E = -61\ log\ (I_{in})/(I_{out}) \quad (1)$$

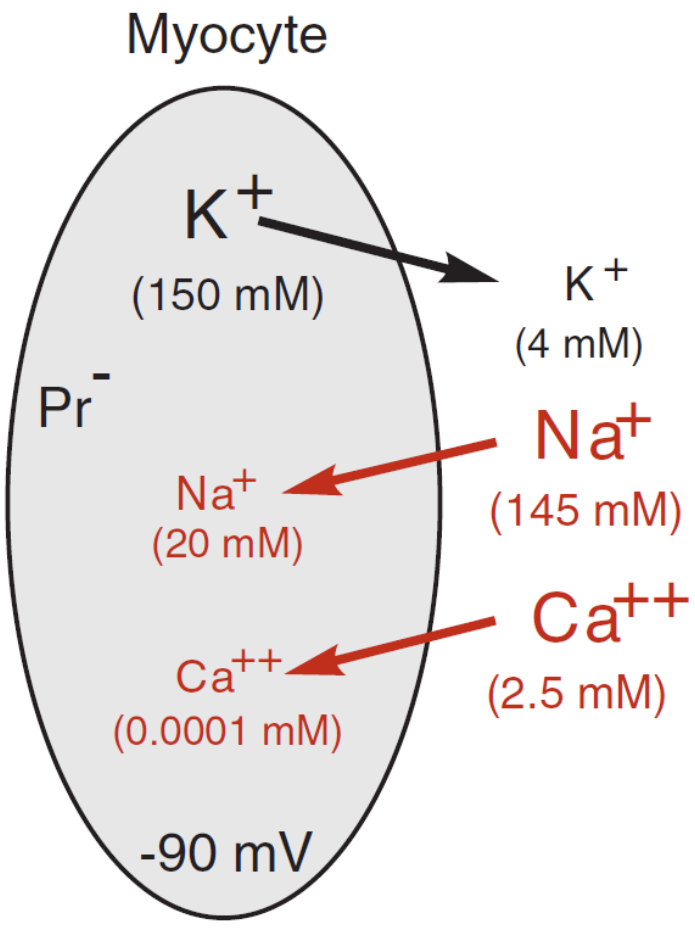

Figure 5. Typical concentrations of the major ions inside a myocyte [5]

Thus, knowing the usual concentration of each ion, we can quantify the equilibrium potential of potassium (E_K), sodium (E_{Na}), and calcium (E_{Ca}) which are equal respectively to -96mV, +52 Mv and +134 mV; The changes of the Em can be approximated as a weighted sum of the equilibrium potentials of each ion:

$$Em = g_k * E_k + g_{Na} * E_{Na} + g_{Ca} * E_{Ca} \quad (2)$$

The g_K, g_{Na}, and g_{Ca} are the ionic conductance, which are related to the membrane permeability to each ion and thereby to the opening or closure of the relative ion channels, which allow the diffusion of one particular ion. Even if the changes to E_m are determined by six variables, the most important are the ionic conductance because the changes in ionic concentrations are small even when Na^+ or Ca^{++} are pulled into the membrane. At rest, most of the potassium channels are open (g_K close to 1) whereas most of the sodium channels and calcium channels are in a closed state (g_{Na} and g_{Ca} close to zero).

2.2.2 Ion channels

Ion channels are responsible for the permeability of the ions to which they have an affinity. Changing their shapes, they enable or block the passage of ions; they could be of two types: receptor-gated channels which open or close based on the interaction with a receptor, and voltage-gated channels, which change their status based on variations of the membrane potential [5].

Important voltage-gated channels are fast sodium channels, which have two gates: the activation gate "m" and the inactivation gate "h." At rest, the activation gate "m" is closed whereas the inactivation gate is opened; when the cell is depolarized because of an adjacent cell the activation gate opens

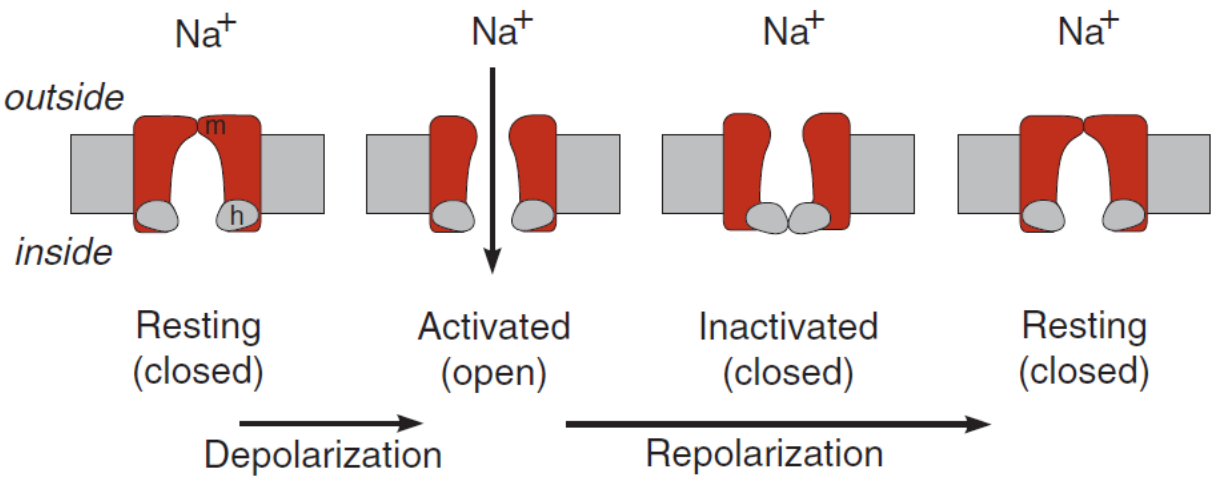

Figure 6. Cycle of Activation and inactivation of sodium gates channels

enabling the passage of sodium ions; after a few milliseconds, the inactivation gate closes, and the channels go to an inactivation/unavailable state. Only after the repolarization, the closed state is restored with the “m” closed and the “h” opened (figure 6).

2.2.3 Action potential

We can recognize two kinds of action potential inside the heart: pacemaker action potential, which occur spontaneously, or non-pacemaker action potential, which necessitates a depolarizing current from adjacent cardiomyocytes. The former can be described by the following points [5] (figure 7):

- Phase 0: a steep increase caused by an initial depolarization to -70mV due to an external stimulus activates fast voltage-gated sodium channels increasing the ionic conductance of the sodium and pulling many sodium ions inward. From figure 7 we can observe that g_{Na} increases, whereas g_K decreases due to the closure of potassium channels.
- Phase 1: initial repolarization of the membrane potential because of the opening of some transient potassium gates and inactivation of sodium voltage-gated channels

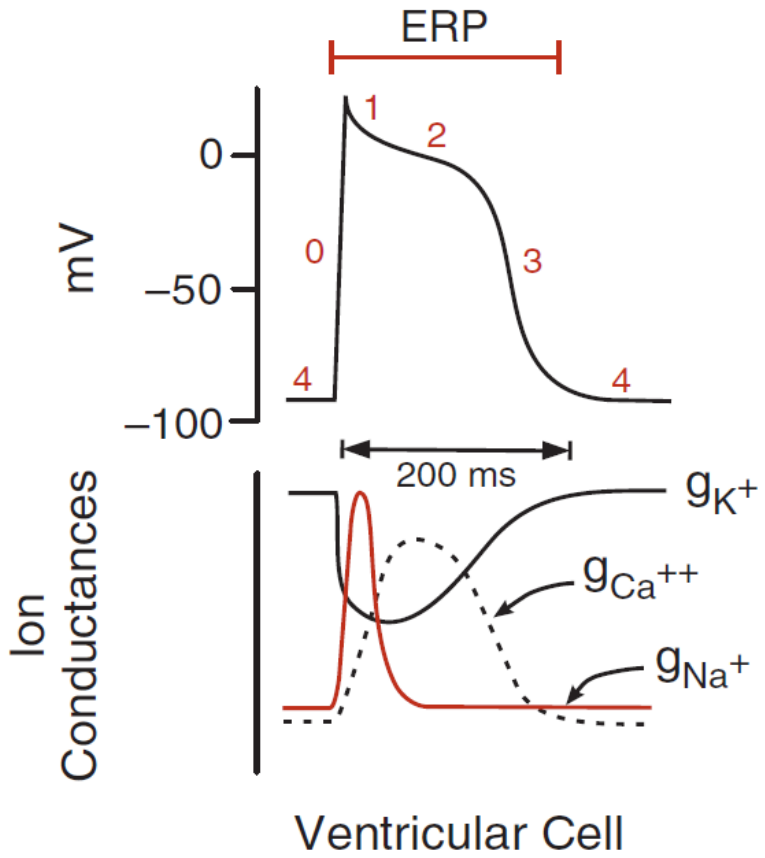

Figure 7. non-pacemaker action potential of a ventricular cell and the relative ionic conductance

- Phase 2: plateau of the membrane potential due to the opening of slow calcium voltage-gated channels, which cause an increase of the calcium ionic conductance (g_{Ca}).
- Phase 3: repolarization happens because of the opening of other potassium channels and the closure of the calcium channels
- Phase 4: resting membrane potential which is restored thanks to ATP-driven pumps, such as Na^+/K^+ ATPase and Ca^{2+} ATPase, and another mechanism in the form of the sodium-calcium exchanger.

During phases 0,1,2 and partly phase 3 there is an effective refractory period (ERP) where the cell cannot be ulteriorly excited because sodium gates are not activated. This functional limitation delays the next action potential giving the heart the time to collect and eject blood and preventing a period of sustained contraction (tetanized muscle). Then, the Relative Refractory Period (RRP) occurs where only a suprathreshold impulse can generate an action potential, however, due to the unavailability of most of the fast sodium channels, the action potential generated during this phase is going to increase slowly due to the influence of slow calcium channel [5].

For what concerns pacemaker action potentials, they are the ones generated by pacemaker cells, which can produce spontaneous electrical impulses. The main pacemaker site of the heart is the Sinoatrial Node.

We can distinguish three phases for a pacemaker action potential [5] as shown in **figure 8**:

- Phase 0: it corresponds to the upstroke of the action potential, and it is caused by the activation of L-type voltage-gated calcium channels which increase the conductance of Ca^{2+} (g_{Ca}) leading to a greater influx of calcium ions inside the cell when it reaches a threshold of -40mV. Moreover, a brief reduction of the outward flow of potassium ions helps the depolarization. When compared to phase 0 of a non-pacemaker action potential, it is possible to observe that the increase is less steep because it is not caused by fast sodium channels.
- Phase3: it corresponds to the repolarization of the cell membrane, which is caused by the mechanism of inactivation of the previously opened calcium channels and the activation of potassium channels leading to efflux of potassium ions. When the cell reaches a negative peak pf -65mV the phase ends.

- Phase 4: it is the phase responsible for the autonomous generation of the action potential. The interaction between three ionic currents is one proposed explanation behind the steady depolarization during this phase. Firstly, at the onset of this phase, the ionic conductance of the potassium is decreasing and therefore the outward potassium current is decreasing. Secondly, the presence of a pacemaker funny current, caused by a net inward influx of sodium ions, contributes to depolarization. Thirdly, a transient inward calcium current, caused by the opening of voltage-gated T-type calcium channels at -50mV, and the successive activation of some of the L-type calcium channels bring the potential to the necessary threshold for the next phase.

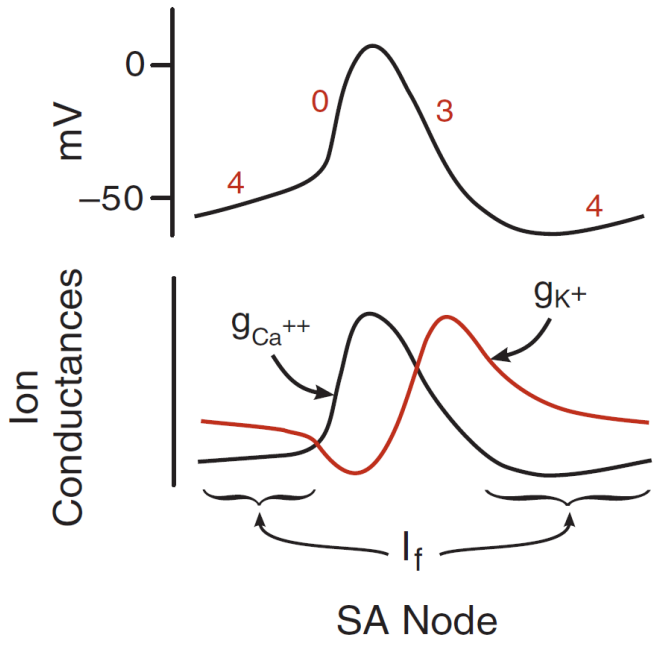

Figure 8. Pacemaker action potential and relative ionic conductance

2.2.4 Conduction system

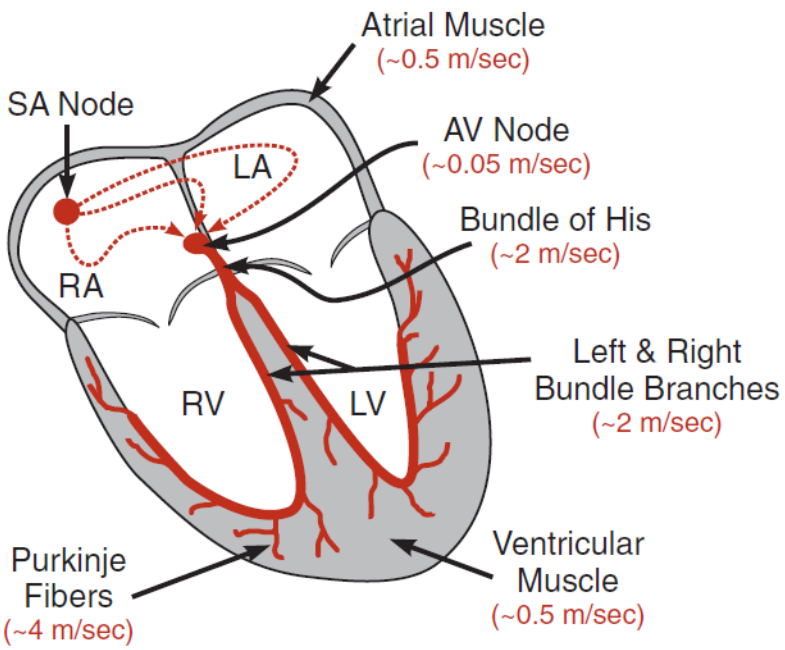

Figure 9. Conduction system in the heart with the typical conduction velocities

The cell-to-cell propagation of the electrical impulse is possible because of gap junctions, which enable the passage of ions between adjacent cells. Thus, when a cell depolarizes, the adjacent cells will start to passively depolarize due to an inflow of ionic current until it reaches a threshold of membrane potential, and a new action potential is generated in the adjoining cells.

Nevertheless, we found a structure of preferential conductive pathways that is the conduction system. The sinoatrial node (SA) generates the initial stimulus at the top of the right atrium, the pacemaking site. Then, the conduction through the atrium does not have a preferential pathway and in general, all the cardiac cells of the right atrium will propagate the electrical impulse until the atrioventricular node (AV node), which represents the only conducting path between atria and ventricles. The conduction in the ventricles starts from the bundle of His and then it splits into the left bundle branch and right bundle branch inside the interventricular septum. Eventually, the branches extend into terminal pathways called Purkinje fibers, which conduct the AP potential to the ventricular myocytes [5] (figure 9).

An important feature of the AV node is its conducting velocity equal to about 0.05 m/s. The consequence is a delay between atrial conduction and ventricular conduction and its physiological relevance can be summarized by the following two points [5]:

- it gives adequate time for the completion of the depolarization, contraction, and emptying of the atrium before ventricular depolarization and contraction
- it reduces the rate at which the AV node transmits impulses; this could be relevant for arrythmias such as atrial fibrillation.

2.2.5 Influence of the Autonomic Nervous system

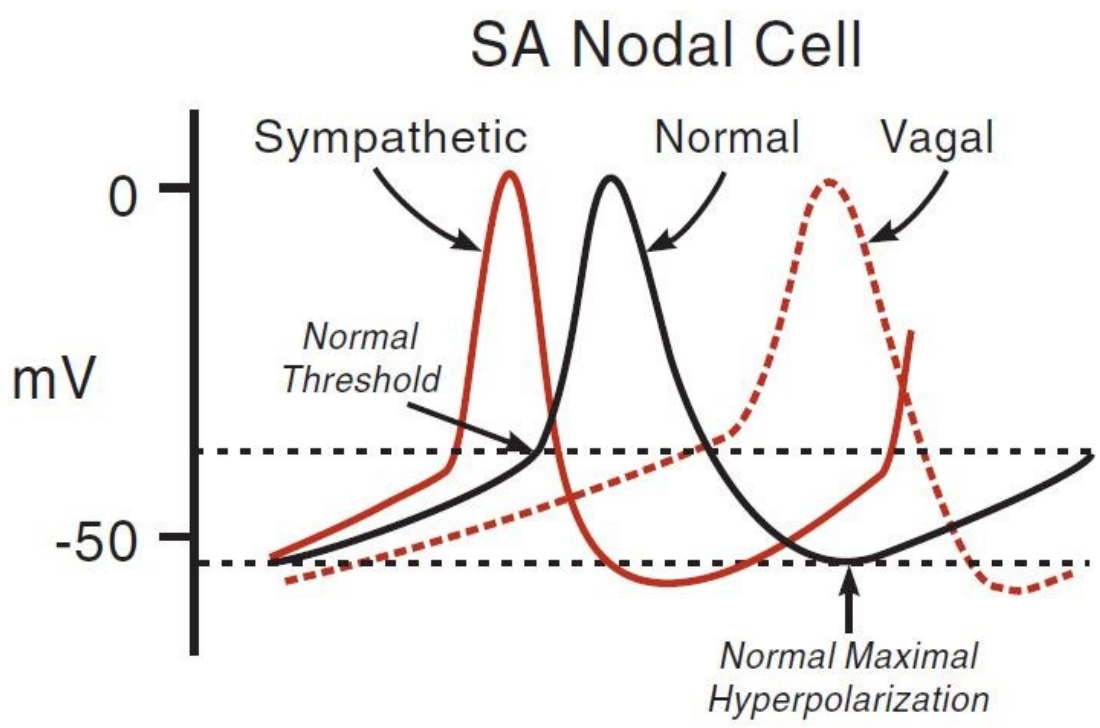

Figure 10. Influence of the autonomic nervous system on the pacemaker action potential

The rate at which the action potential pacemaker fires is influenced by the Autonomic Nervous System. The cardiovascular centers are located in the medulla which connects the brain and spinal cord. Medulla receives impulses from the higher centers and hypothalamus and afferent receptors of the body.

Efferent vagal and sympathetic nerves project on the heart and vasculature and influence the rate at which the pacemaker of the SA node fires. The vagal nerve slows down the firing rate, whereas the sympathetic nerve increases it [5].

The mechanisms through which the rate of the pacemaker firing can be changed are the following, summarized in figure 10:

- changes in the slope of phase 4
- alterations of the threshold potential for the onset of the action potential
- changing the level of repolarization in phase 3

We can observe how the sympathetic nerve can make the phase 4 increase steeper leading to a faster rate or it brings the threshold of activation of calcium channels lower anticipating the action potential.

On the other hand, the vagal nerve can influence the steepness of phase 4 depolarization in such a way that a slower depolarization would need more time to reach the threshold; it can also decrease the negative depolarization peak or increase the necessary threshold for the activation of voltage-gated channels.

2.3 Mechanical physiology of the Heart

2.3.1 Contraction Mechanism

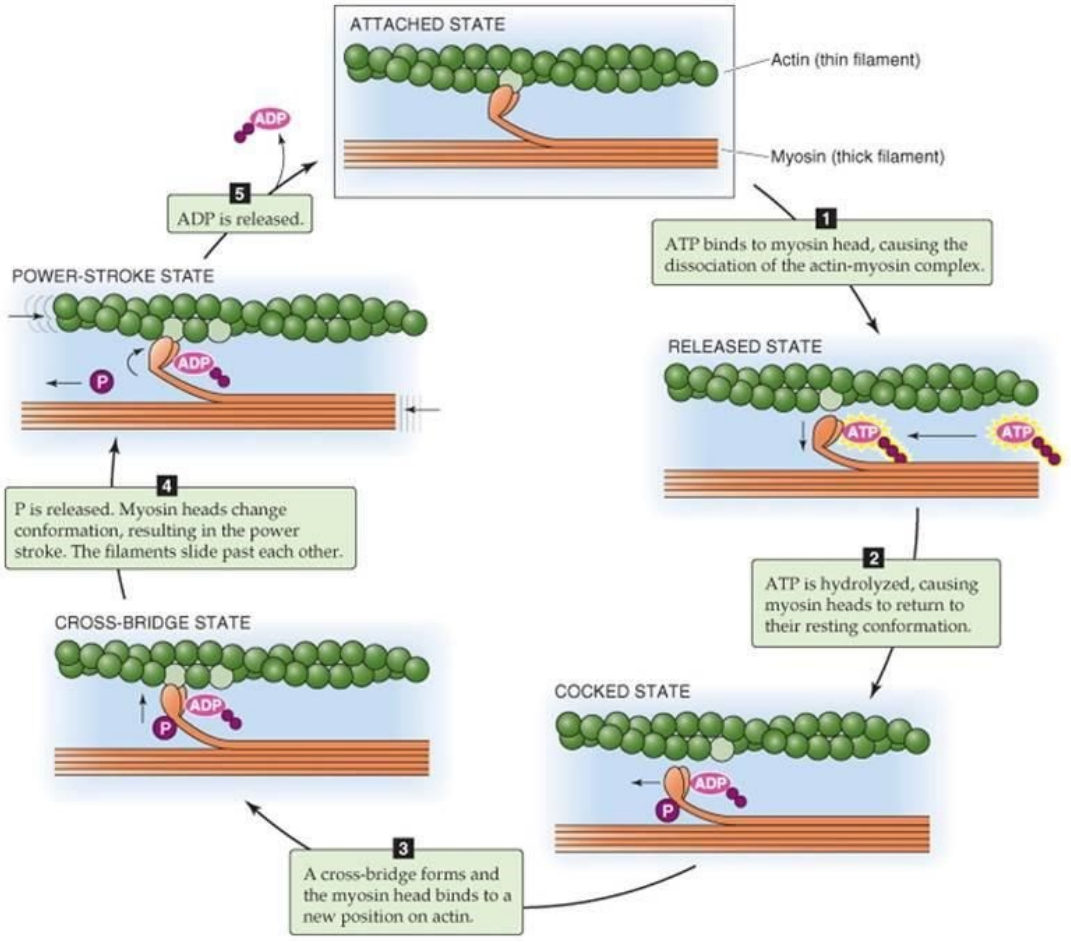

Figure 11. Cross bridge cycle in the cardiac muscle cell [6]

One of the determinants of the cardiac function as a pump is the contraction mechanism which is dependent on the relationship between calcium and the contractile protein of the myocytes in the form of sarcomeres.

The contraction-relaxation mechanism consists of the sliding action of thin actin filaments over thick myosin filaments thanks to the interaction between myosin heads and actin-binding sites. In particular, the thin filaments of actin, formed by a chain of spherical proteins, are coupled with tropomyosin filaments that fold around actin exposing or covering the binding sites, and a regulatory troponin complex, which can interact with calcium ions controlling the position of the tropomyosin filament in relation to the binding sites [6].

The following stages describe the cycle of contraction-relaxation mechanism [6] and are schematised in figure 11:

1. attached state: myosin head are attached to the binding site of actin after the contraction has happened
2. released state: ATP is the necessary molecule for the release of the myosin head and consequently the relaxation of the muscle. Thus, ATP binds to the head and it is hydrolysed by the ATPase activity of the myosin head dividing in ADP and releasing a phosphate group which binds with the myosin head and causes a conformational change of the myosin.
3. cocked state: the myosin heads are in a resting state, perpendicular to the actin filament, and they can be activated. They are in a state where the necessary energy for the successive sliding process is already stored.
4. cross-bridged state: thanks to the interaction of calcium ions with the troponin complex the binding sites of actin are exposed; thereafter, myosin heads bind to the binding sites of actin forming a cross-bridge; the mechanism of calcium regulation is explained in the next paragraphs.
5. power stroke state: the binding causes conformational changes of the myosin head, which pulls the actin filament and makes it slide over the myosin filament. Eventually, phosphate groups and ADP are released, exposing the ATP binding sites of the attached myosin.

2.3.2 Excitation-contraction coupling

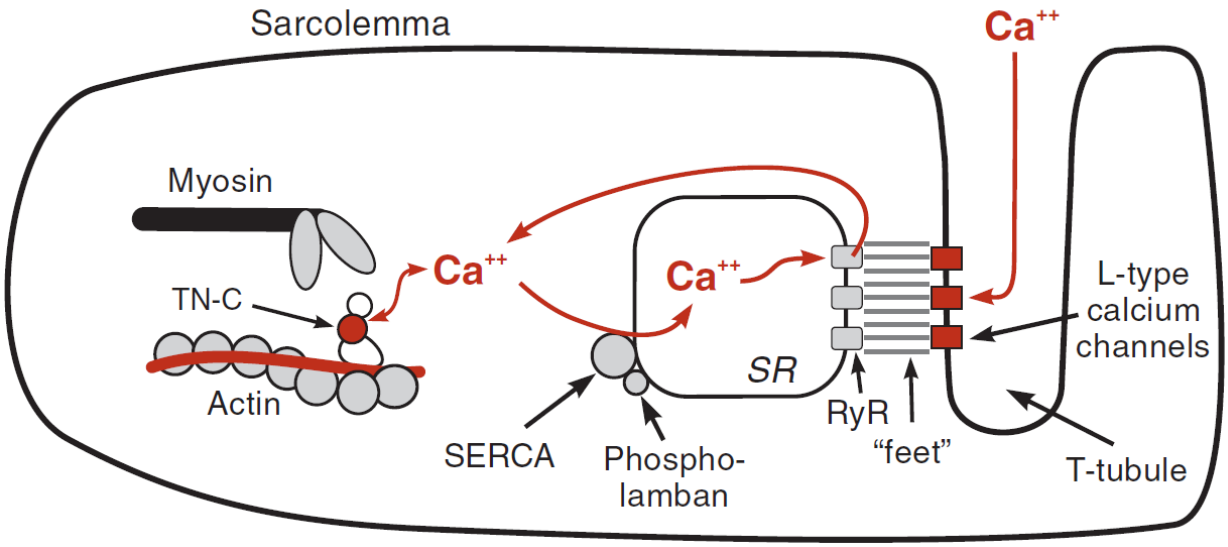

Figure 12. Schematic representation of the excitation-contraction coupling mechanism inside the myocytes

The mechanism relating the electrical activity of the myocytes with the contraction is the excitation-contraction coupling. The following points summarises the steps of this mechanism, schematised in figure 12:

1. The initiation of an Action Potential is also the onset of the excitation-contraction coupling. The action potential will start when the cell reaches a certain value of intracellular potential thanks to passive depolarization. Consequently, sodium channels activate causing a steep rise in the membrane potential
2. L-type long-lasting Ca^{2+} channels sense the depolarization and open, permitting the influx of the trigger calcium; this happens everywhere in the sarcolemma and inside the T tubules, which are in tight contact with the Sarcoplasmic Reticulum (SR) via Ryanodine (RyR) receptors.
3. RyR receptors sense the small calcium influx and then they communicate to the Sarcoplasmic reticulum for the release of the stored calcium ions. Intracellular calcium concentration is multiplied by a factor of one hundred. This is the reason the small amount of calcium entering through L-type calcium channels is called trigger calcium
4. In the presence of high calcium concentration and sufficient ATP, the previously described mechanism of contraction takes place; the troponin complex regulatory system will be subjected to a conformational change due to interaction with calcium ions and the tropomyosin will free up the binding sites for the myosin heads. Thus, the cross-bridge cycling will continue until the concentration of calcium ions inside the cells is large enough.
5. Then, the relaxation happens, corresponding to the repolarization of the action potential. The intracellular concentration of calcium ions decreases thanks to energy-dependent pumps, which bring calcium either outside of the cell or inside the SR. In the latter case, the pump is called Sarco-endoplasmic reticulum calcium ATPase (SERCA).
6. With lower calcium concentration the troponin complex dissociates from calcium, causing the binding sites of actin to be covered by troponin filaments
7. Eventually, the ATP causes the detachment of myosin heads from actin in the remaining cross-bridges

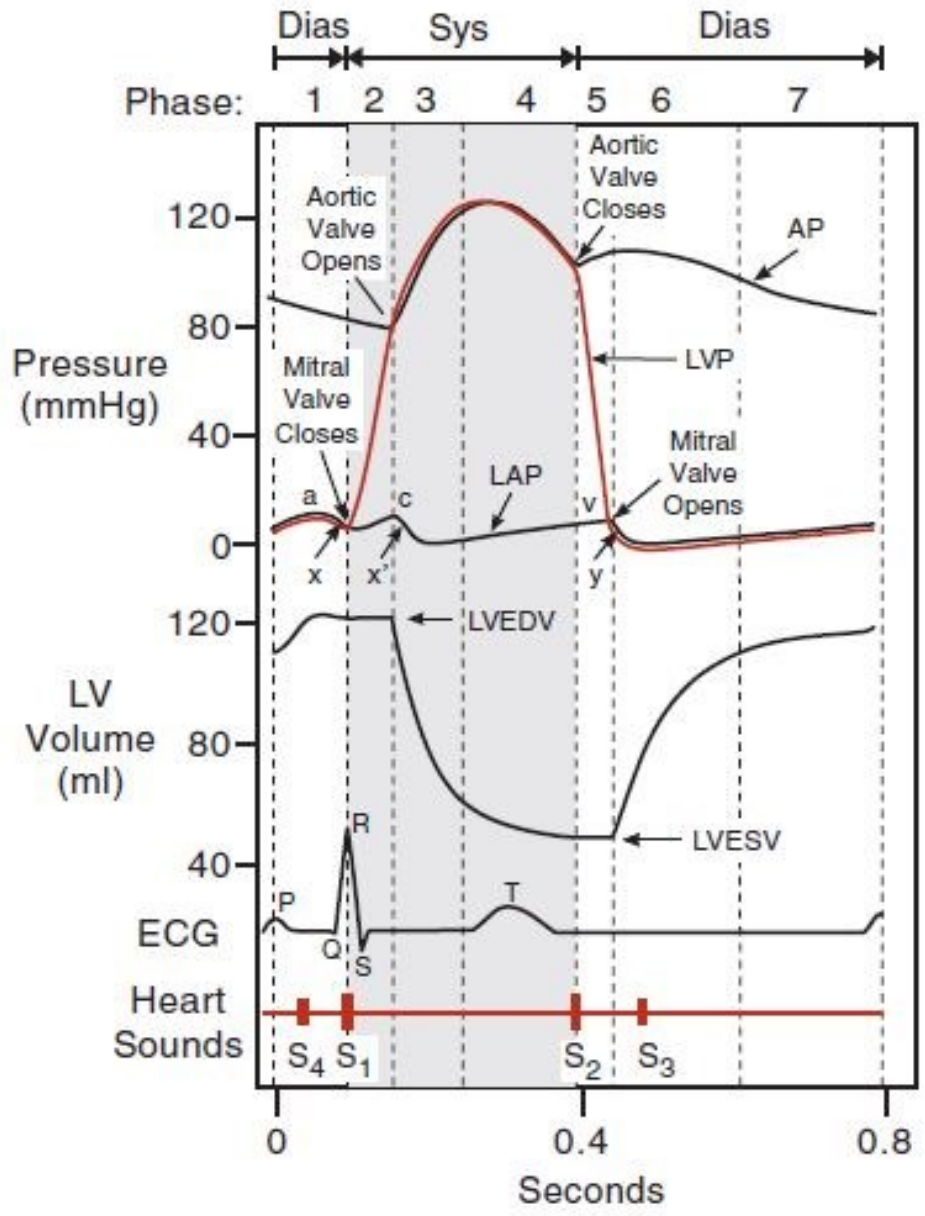

Figure 13. Wigger's diagram of the left ventricle cardiac cycle

2.3.3 Cardiac cycle

The cardiac cycle is the cyclic occurrence of mechanical events inside the heart between the onset of a heartbeat and the successive one. A cardiac cycle is composed of a relaxation phase called diastole and a contraction phase called systole. During diastole, the heart is filled with blood, whereas during systole the blood is expelled into the circulation. Usually, the events of the cardiac cycle are shown with Wiggers' diagram reporting the pressure curves of the aorta, left ventricle, left atrium, the left ventricular volume, and the corresponding electrocardiogram and phonocardiogram (figure 13). The

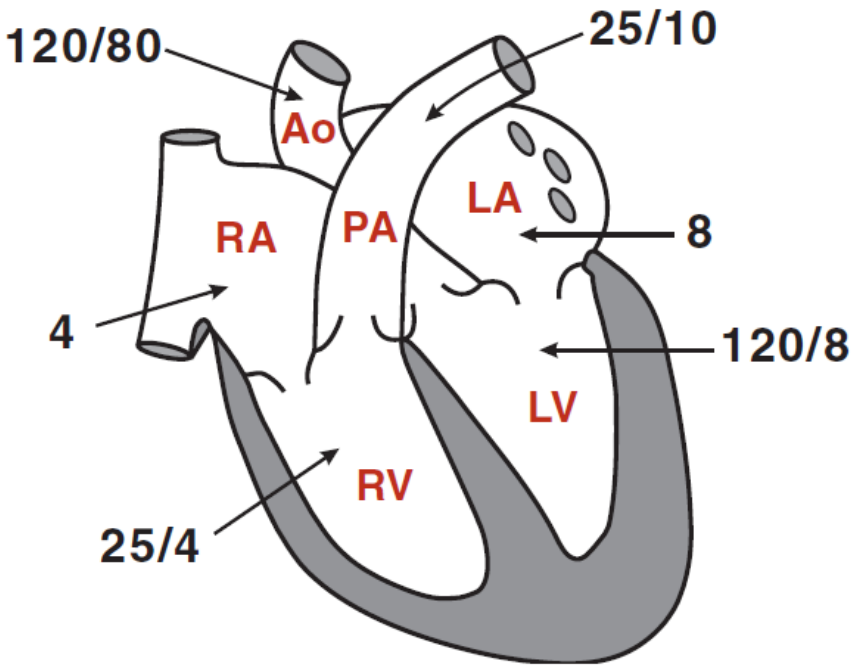

Figure 14. Typical values of pressure inside the heart chambers and for the outflow tracts. The values are expressed in mmHg and the higher ones correspond to maximal systolic pressures, whereas the lower ones to maximal diastolic pressure

patterns found in the right heart and the timing of the events are qualitatively similar. The greatest difference is in the pressure with the right side experiencing a lower level of pressure when compared to the left side. Typical values of pressure for the right ventricle range from a minimum of 0 to 4 mmHg during diastole to a maximum of 25 to 30 mmHg during systole. Other values of pressure can be seen in figure 14.

We can distinguish the following seven phases during the cardiac cycle [5]:

1. Atrial systole: during this phase, the Atrio-Ventricular valves are opened, whereas aortic and pulmonic valves are closed. The atria contracts enhancing ventricular filling in a mechanism that is sometimes referred to as atrial kick; it can be noticed in the curve of atrial pressure with the “a” peak followed by a decrease in pressure denoted “x”. However, ventricular filling during diastole is mainly a passive process and the atrial contribution usually accounts for about 10 to 20% of the total filling at rest and 40% at higher heart rates. The end of this phase corresponds to the end of diastole and the End Diastolic Volume (EDV) of the ventricles is about 120 mL in association with an end-diastolic pressure of 8 mmHg for the left ventricle and 4 mmHg for the right ventricle.

2. Isovolumetric contraction: The ventricular pressure steeply increases causing the AV valves to close and to produce the first heart sound. During this phase, all valves are closed, therefore no inflow or outflow of blood is possible, and the volume remains constant. For this reason, the contraction of the ventricles occurs in isovolumetric conditions. Atrial pressure starts to rise due to the venous inflow as can be observed by the “c” peak.
3. Rapid ejection: The intraventricular pressure surpasses the pressure within the aorta and the pulmonary artery. This pressure difference is the driving force that causes the opening of the aortic and pulmonic valves and the rapid ejection of the blood. During this phase, the maximal systolic aortic and pulmonary pressure can be recorded with respectively 120 mmHg and 25 mmHg. Simultaneously, the atria continue to fill due to venous inflow and their pressure slightly decreases because the atrial base is pulled downwards.
4. Reduced ejection: Ventricles start to depolarize and muscle relaxation occurs. Ventricular pressure is below the pressure in the outflow tracts; however, a reduced ejection continues due to inertia. Atrial pressure continues to rise. This is the end of the ventricular systole.
5. Isovolumetric relaxation: The ventricles continue to relax and the pressure decreases leading to a closure of aortic and pulmonic valves, which is associated with the Second Heart Sound. The pressure in the outflow tract of the ventricles slowly goes down and is characterized by a characteristic notch or incisura, whereas the ventricular pressure decrement is fast. The end-systolic volume is 50 mL
6. Rapid filling: Ventricular pressure goes below atrial pressure as the ventricles continue to relax and the atria continue to receive blood from the venous inflow. This pressure gradient causes the opening of the AV valves and the consequent passive filling of the ventricle. Before the opening of the valves, we can observe a typical peak in atrial pressure denoted “v”, followed by the “y descent”
7. Reduced filling or ventricular diastasis: Near to completion of ventricular filling, the ventricle pressure starts to rise causing a decrement in the rate of filling

It is common to display ventricular function with a volume-pressure graph like the ones shown in figure 15. The four phases of the loop are ventricular filling (a), isovolumetric contraction (b), ventricular ejection(c), and isovolumetric relaxation (d). The width of the graph, representing the

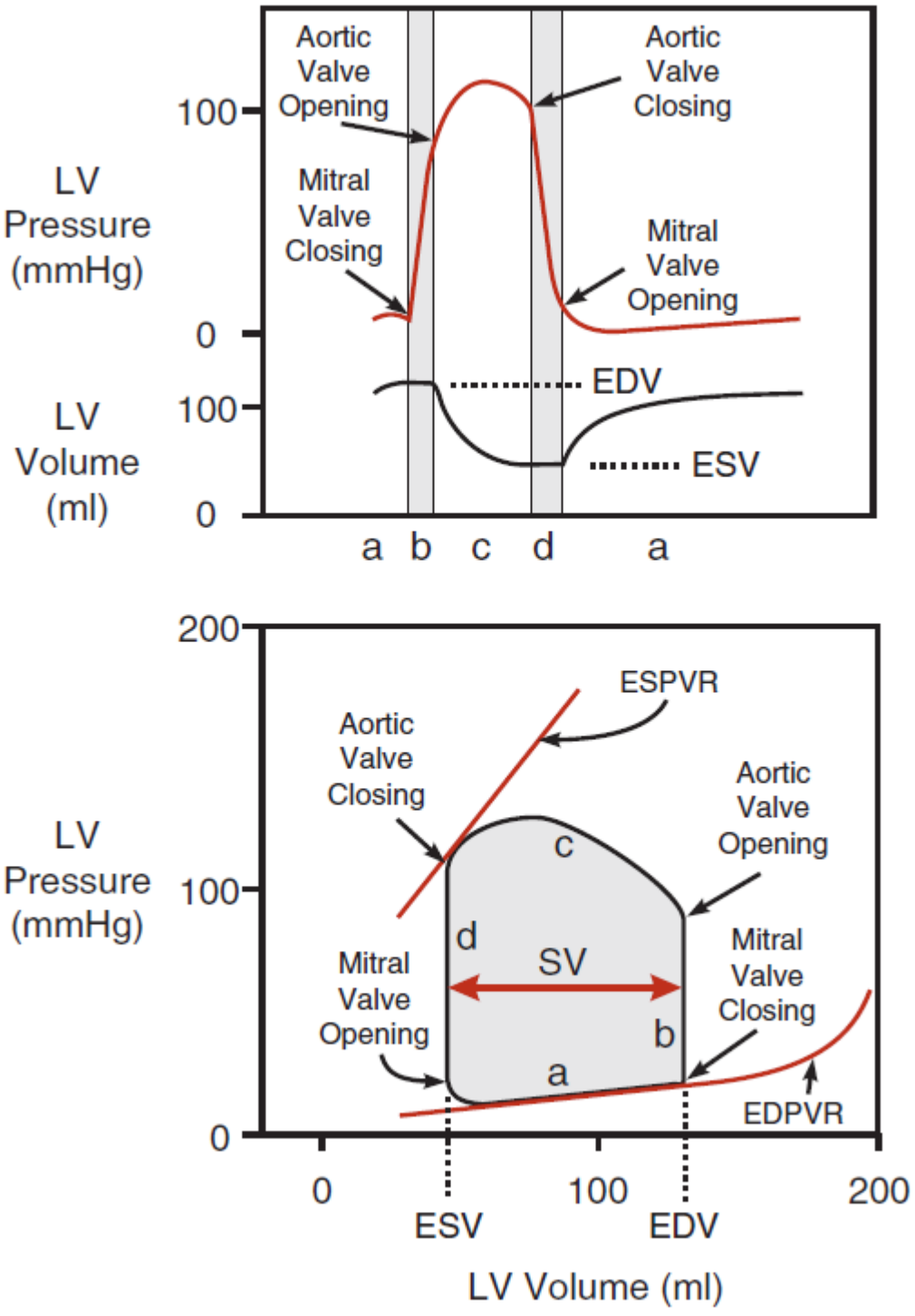

Figure 15. Pressure- Volume loop of a cardiac cycle and its relationship with the wigger's diagram. The ESPV and EDPV relationships are displayed.

difference between EDV and ESV is called Stroke Volume. The area inside the loop is called ventricular stroke work. A notable relationship is the one followed during the filling phase of the ventricle, called the end-diastolic pressure-volume relationship (EDPVR) because it is the reciprocal of ventricular compliance, defined as the ratio between pressure changes and volume changes. Another notable relationship is the end-systolic pressure-volume relationship (ESPVR) which describes the maximal pressure that the ventricle can produce at any given volume

2.4 The Athlete's Heart

Myocardial structure and function vary because of exposure to high-intensity physical activity. The modern view about this alteration, usually called Athlete's heart, is that it is a positive cardiac adaptation [7]. The heart changes with a usual enlargement of the cardiac chamber to produce higher stroke volume and higher cardiac output as a response to an increased oxygen demand given by the exercise training [7]. Moreover, there seems to be a difference in the cardiac adaptations between:

- isotonic exercise, typical of endurance athletes, who exhibit continuous elevated cardiac output with unchanged or decreased peripheral vascular resistance.
- isometric exercise, typical of strength training, is associated with higher peripheral vascular resistance and normal or moderately increased cardiac output. Because of the growth of peripheral vascular resistance, hypertension and marked Left Ventricular (LV) afterload could arise. This pattern is seen in American Football athletes

LV remodelling has been a topic of extensive research in the athletic population and the first study to suggest a difference between the previously described training regimens was the one by Marganroth et al [8] in 1975, where an echocardiogram was performed on 56 athletes and compared to a control group. Their results reported an increase in left ventricular end-diastolic volume and mass in athletes practicing sports with isotonic exercises, such as swimming and running with no appreciable changes in wall thickness; this adaptation is usually referred to as eccentric left ventricular hypertrophy. On the contrary, athletes practicing strength-based sports, focused on isometric exercises, were reported to have increased left ventricular wall thickness and mass with normal values of left ventricular end-diastolic volume; this adaptation is called concentric left ventricular hypertrophy.

One of the largest cohorts concerning LV dimension screening is the one by A. Pelliccia et al. [9] where 1309 elite Italian athletes, of which 957 men and 352 women, ranging from 13 to 59 years of age, across 38 different disciplines, were studied with echocardiography to assess the dimension of the left ventricular chamber. The authors reported a large variation in the LV end-diastolic cavity dimensions with a mean of 48 mm (min: 30mm, max 48 mm) in women and a mean of 55 mm (min: 43 mm, max: 70 mm). Fifty-five percent of the athletes had values in the acceptable range of less than 55 mm and 14% of the athletes were found to have cavities with values higher than 60 mm. Nevertheless, this marked increase in the cavity dimension was not coupled with LV dysfunction or wall abnormalities and it was related to body surface area and endurance sports.

For what concerns LV wall thickness, another study by Pelliccia et al. [10] can be considered, where a sample of 947 athletes was used. The study aimed to set an upper threshold for LV wall thickness in the athletic population and the conclusion was that values until a wall thickness of 16 mm, the highest value measured, were non-pathological. However, a major number of athletes had thickness inferior to 13 mm, and measures above 13mm were confined to just a subpopulation of fifteen rowers and one cyclist, corresponding to 1.7% of the total athletes. In these rare cases, also an enlargement of the left ventricular end-diastolic cavity dimension was reported with values ranging between 55 to 63 mm.

Furthermore, the left atrium has been deeply investigated with a large cohort study including 1777 athletes across thirty-eight sports by Pelliccia et al. [11]. Twenty percent of the sample had enlarged left atrial dimension, defined when the threshold of 40 mm was surpassed, however atrial arrhythmias were frequently diffused in athletes with both enlarged left atrium and normal left atrium.

Research on the right part of the heart seems to demonstrate a parallel development of the dimension of the right chambers. In the study by Henriksen et al. [12], 127 elite endurance athletes were analysed, and the authors reported higher cavity dimensions for both the right ventricle and right atrium when compared to previously found values in the general population. 2D echocardiographic measurements of the heart included:

- RV l, computed from the right apex to the middle point of the tricuspid annulus; mean values were 84.1 ±8.1 mm and 44.5 ± 4.5 mm/m2 when normalized to body surface area.
- RV b, computed in the middle of the right ventricle; mean values were 30.0 ±4.4 mm and 15.8 ± 2.0 mm/m2 when normalized to body surface area.
- RA l, defined as the maximal diameter from the tricuspid valve to the anterior wall of the atrium, was 55.4 ± 6.1 mm or 28.9 ± 3.2 mm/m2
- RA b, defined as maximal transverse diameter, was 49.4 ± 1.4 mm or 25.7±2.6 mm/m2

In strength athletes, the degree to which the right atrium grows remains not properly understood [7].

2.5 Cardiovascular issues in American Football Athletes

American Football participation seems to have negative consequences on cardiovascular health due to either cardiovascular risk factors or pathologic cardiovascular traits, particularly regarding the linemen position players [13] [14].

The reasons why participation in American Football can lead to the occurrence of cardiovascular issues are not properly understood, although as suggested by [13] it is the result of multiple concomitant factors, such as intense static exercise, scarcity of dynamic and aerobic conditioning, abuse of nonsteroidal anti-inflammatory drugs and diet.

Cardiovascular disease risk factors can emerge both during the career of athletes and after they retire from the field, therefore investigations should interest both retired professionals and players during the period of their activity [14].

The next sections briefly discuss the main cardiovascular risk factor for American football athletes, hypertension, and the main pathological cardiovascular phenotype, concentric left ventricular (LV) remodeling. Appropriate attention will be given to the differences between two categories of player positions: linemen and non-linemen.

2.5.1 Hypertension

Elevated blood pressure can be classified into four distinct categories based on current guidelines of the International Society of Hypertension [15]:

- normal Blood Pressure (BP) is defined as a Systolic Blood Pressure (SBP) less than 130 mmHg and a Diastolic Blood Pressure (DBP) less than 85 mmHg
- high normal BP when SBP is between 130 and 139 mmHg and/or DBP between 85 and 89 mmHg
- Grade 1 hypertension is defined with SBP between 140-159 mmHg or DBP between 90 and 99 mmHg
- Grade 2 hypertension is defined with SBP higher than 160 mmHg or DBP higher than 100 mmHg

Based on various guidelines, the classification and the terminology used could slightly differ. For example, the 2017 American Health Association guidelines [16] define stage 1 hypertension, an SBP between 130 and 139 mmHg or DBP between 80-89 mmHg, a stage 2 hypertension with every measurement above these thresholds, whereas normal BP is associated with SBP less than 120 mmHg and DBP less than 80mmHg; elevated BP is defined for SBP in the interval 120-129 mmHg and the DBP less than 80 mmHg.

Another classification, which is diffused in many cohort studies of American football athletes, is the one given by the Seventh Report of the Joint National Committee(JNC-7) [17] where normal BP is defined as SBP/DBP less than 120/80 mmHg and hypertension as SBP/DBP higher than 140/90 mmHg with a subdivision in stage 1 and stage 2 at the threshold of 159 mmHg for SBP and 99 for DBP. Moreover, the term prehypertension is introduced for the values in the interval 120-139 for SBP and 80-89 for DP.

Hypertension is a well-established risk factor related to later cardiovascular disease (CVD) mortality and high systolic blood pressure was the main risk factor accounting for 10.4 million deaths in 2017 [18]. Moreover, in a cohort study of 18.881 male university students [19], elevated blood pressure at university entry was correlated with an elevated risk of all-cause mortality, CVD mortality, and coronary heart disease (CHD) mortality in subsequent years even when adjusted for middle-aged hypertension. Thus, hypertension in early adulthood, the period of maximum exposure to a professional sports career for American football athletes, is an indicator of the potential development of cardiovascular disease.

In one of the largest cross-sectional studies of professional players, 504 National Football League (NFL) [20] players were analyzed to understand the occurrence of different CVD risk factors compared to an age and race-matched control group. It was found a significantly higher SBP and DBP ($p< 0.001$) for the American football athletes and even when stratified by positions the difference in SBP remained significant ($p< 0.001$). In addition, the incidence of hypertension and prehypertension, defined with the JNC-7 criteria, was significantly higher in the football players with 13.8% (95% CI: 11.0% - 16.7%) for hypertension and 64.5% (95% CI : 58.3% - 70.7%) for prehypertension, respectively against 5.5% (95% CI : 4.6% - 6.6%) and 24.2% (95% CI : 22.3% - 26.1 %) of the control group.

Other studies analyzed the consequences of the participation of a single season of American football on the blood pressure of the athletes. An example is given by paper [21], where six groups of the first-year college athletes in six different years spanning from 2006 to 2011 were analyzed prior to the season and after the season compared to a control group of rowers. For each year they observed a statistical increase in SBP and DBP for American athletes. Using the JNC-7 criteria during the postseason, 47% of football players had prehypertension against 39% in the preseason and 14% had

stage I hypertension, which did not have any occurrence in the preseason. Instead, in the control group, these changes were not present and the DBP statistically decreased.
A similar result was obtained by a paper [22], which reported that American Football participation was related to a significant ($p < 0.001$) growth in SBP from 119±10 in the preseason to 124 ± 12 in the postseason.

In a longer observational cohort study of three years, 126 US American Football players [23] were analyzed in the preseason of their first college year as a baseline measurement and then for the postseasons of the next three years. A positive progression of SPB was found with no statistical difference between linemen and non-linemen, even though the former group experienced a greater variation in SBP levels. Furthermore, SBP was found to be an independent predictor of the development of left ventricular concentric hypertrophy coupled with left ventricular dysfunction.

The reasons behind the development of elevated blood pressure in American football athletes could be because of vascular dysfunction and arterial stiffening, which in the general population anticipate the beginning of hypertension [24]. Jonathan H Kim. et al. [25] reported an increment in central aortic pressure, an index of central arterial pressure load, and pulse wave velocity, an index of arterial stiffening, in thirty-two athletes participating in one season of American football when compared to an age-matched population of non-athletic undergraduate students.

2.5.2 Player position difference in cardiovascular health

Some lines of evidence suggest the linemen as subjects at increased risk due to weight gain and higher prevalence of hypertension when compared to non-linemen.

Taking the previously cited analysis of a single season of football [21] [22], the linemen's position seemed to have a negative effect on blood pressure. Specifically, in the cohort study [22], even though the increase in systolic blood pressure appeared in both linemen and non-linemen, the extent of the variation was significantly larger among linemen with a rise of 10±8 mmHg against 3±7 mmHg in non-linemen. Additionally, using the JNC-7 criteria, linemen were more inclined to develop hypertension going from no cases in the preseason to 30% incidence in the postseason, whereas the number of prehypertensive cases among non-linemen remained unchanged and no cases of hypertensive athletes were reported.

In the cohort study [21] lasting six years with six different groups, analogous were the findings with linemen experiencing higher increment in SBP (12±9 versus 4±7 mmHg, $p < 0.001$) and DBP (4± 8

versus -1 ± 7 mmHg $p<0.001$) in one season when compared to non-linemen. Following the JNC-7 criteria, linemen in the postseason were the ones with most of the prehypertension cases (58%) or stage I hypertension cases (25%).

The other significant difference between positions of American football players is in the incidence of obesity or weight gain and considering that being overweight (BMI between 25 and 29.9) or obese (BMI higher or equal to 30) is strongly associated with CHD [26], this is a risk factor to be considered. American football athletes have a higher BMI when compared to the control population of the same age and race [13] and the weight of all the players has an increasing trend throughout different playing seasons [23]. Nevertheless, the linemen are reported to have significantly higher BMI levels than non-linemen and a significant increase in weight and BMI over a season [21] [22].

The degree to which BMI influences cardiovascular disease development in American Football Athletes is not clear [13], but weight gain seems to be an independent indicator of left ventricular concentric hypertrophy [23].

Another major difference between the two categories of field position is in the distinct cardiovascular adaptations, which are a consequence of different training stimuli with the linemen's training regimen focusing more on isometric exercise and non-linemen being exposed to both isometric and isotonic exercises [13].

In the cohort study [22] two were new perspectives introduced concerning both structural and functional remodeling of the heart among American football players:

In non-linemen, the left ventricular remodeling, which is eccentric in geometry, is coupled with global longitudinal strain (GLS) increase, an indicator of systolic function. This adaptive form of remodeling is like the one documented in endurance athletes.

In linemen, the LV concentric hypertrophy in conjunction with inefficient LV function demonstrated by a decrease of GLS suggests a maladaptive remodeling. The impaired GLS could be justified by hypertension or the concentric hypertrophy of the left ventricle. The pathological cardiac changes should be distinguished from the traditional form of adaptive heart remodeling named Athlete’s Heart.

The observations in the cohort study of Jonathan H. Kim et al. [23] support the concept of a maladaptive phenotype among football athletes with higher weights and SBP. The authors documented in all the athletes a progressive rise of both eccentric hypertrophy, which is a characteristic of the Athlete’s Heart, and concentric hypertrophy, which grew in its prevalence during the study lasting three years. Moreover, athletes with concentric LV hypertrophy had higher weights,

higher SBP levels, higher Pulse Wave Velocity (PWV), a measure of arterial function, and lower early diastolic left ventricular velocity (E'), a measure of cardiac function, compared to the remaining athletes.

Using a multivariate linear model weight gain and SBP were related to increasing PWV and concentric LV hypertrophy. Also, in this case, the authors suggest that this form of LV remodeling should not be labeled Athlete's heart because it does not seem an adaptive response to sport but the consequence of the previously cited risk factors.

Additional evidence of this form of maladaptive cardiac remodeling can be found in the cohort study of Rory B. Weiner et al. [21]. They reported that during preseason left ventricular hypertrophy was similarly prevalent in both the considered positions of linemen and non-linemen and the same can be said for the magnitude of increase throughout the season. Nevertheless, the geometry associated with the LV remodeling was different and the linemen were characterized by concentric LV hypertrophy. Additionally, in linemen, LV mass changes and absolute values of postseason LV mass were correlated respectively with changes in SBP and absolute values of SBP in the postseason.

Knowing the considerations made by Marganroth et al. [8] which distinguished between the remodeling of athletes participating in isotonic exercise and isometric exercise as eccentric and concentric variants respectively, a possible new explanation was introduced by Rory B. Weiner et al. [21]. Their findings expand the conventional idea that LV remodeling in sport is due to brief iterated hemodynamics change during the activity and support that the development of concentric LV hypertrophy is not just the result of blood pressure peaks induced by isometric activity, but it is a consequence of continuous hypertension during a period of athletic activity. With this perspective, LV concentric hypertrophy is seen as a form of hypertensive LV remodeling, which should be differentiated by the concept of the Athlete's heart, usually used with positive implications.

2.5.3 Cardiovascular mortality in retired American Football players

The first analysis of mortality in retired American Football athletes was the one conducted by the National Institute of Occupational Safety and Health in 1994 [27], analyzing the data coming from 6848 retired players. The study concluded that the overall mortality of the considered football players was 46% lower when compared to the general population. However, for those who played offensive and defensive linemen the cardiovascular mortality was 50% higher than the control group. Furthermore, stratifying by position, the linemen had a 3.7 times higher risk of CVD than the other players' positions. BMI larger than 32 was associated with a six times higher CVD risk than the lower classes of BMI.

In a successive study by Baron et al. [28], 3439 retired National Football League (NFL) players with at least five playing seasons between 1959 and 1988 were analyzed. The overall mortality was significantly lower with a standardized mortality ratio (SMR) of 0.53 (95% CI: 0.48 to 0.59) when compared to a control group. Only for defensive linemen, the overall CVD mortality was increased with a standardized mortality ratio of 1.42 (95% CI: 1.02 to 1.92) and even higher values for SMR of cardiomyopathy mortality with 5.34 (95% CI: 2.30 to 10.50). Moreover, they found a doubled risk of CVD mortality for the player with a BMI higher than thirty during their playing career.

A recent cohort study compared the mortality rates of retired NFL and Major League Baseball (MLB) players [29]. The authors concluded that American Football players had higher rates of mortality, which were a consequence of higher rates of CVD mortality and neurodegenerative disease mortality. The Hazard Ratio for all-cause mortality was 1.21 for the NFL players against MLP players with a confidence interval going from 1.06 to 1.39. Hazard ratios for CVD mortality and neurodegenerative disease mortality were respectively 2.40 (95% CI: 2.03-2.84) and 2.99 (95% CI: 1.64-5.45).

2.6 Cardiovascular screening in American Football Athletes

2.6.1 Pre-participation screening in American Football Athletes

Sudden cardiac death (SDC) is one of the major causes of death in athletes with incidence rates in the range of 1:40 000 to 1: 80 000 [30]. A study on the incidence and etiology of sudden cardiac arrest (SDA) and death in the USA over 4 years across different sport disciplines and different ages reported the occurrence of 331 cases, of which 158 survivors. A quarter of these cases (25.4%) occurred in American Football athletes [31]. Common causes of SDA comprehended hypertrophic cardiomyopathy (20.6%), idiopathic left ventricular hypertrophy (13.4%) and coronary artery anomalies (12.0%). Nevertheless, at present, the pathologies causing SCD in the American Football athlete population and the degree to which congenital/genetic diseases or developed cardiovascular adaptations are responsible for the occurrence of SDC are unknown [13].

Pre-participation cardiovascular screening employing a 12-lead electrocardiogram (ECG) should aim at recognizing pathological variants to decrease the level of incidence of SCA/D. From a study by Joseph K. Choo et al. [32] 1282 American professional football players were subjected to a 12-lead ECG analyzed independently by two cardiologists. Fifty-five percent of the players resulted to have an abnormal ECG with at least one significant abnormality. Early repolarization patterns and ST and T wave abnormalities were typical in Black players, whereas intraventricular conduction delays were typical in white players. This difference in the ECG abnormalities by race was also evident in the study by Anthony Magalski et al. [33] where it was highlighted a higher incidence of ECG abnormalities in the black race, which was found to be the only predictor of an ECG with out of normal characteristics. Stratifying by player positions, they noticed a higher prevalence of abnormal ECGs for players experiencing a transient burst of high-intensity activity in the form of sprinting, such as receivers, running backs, and defensive backs, whereas it was a less common finding for positions with a lower level of exertion, such as linemen and kickers.

2.6.2 Application of cardiac monitoring during training

Heart Rate to support coaching decision making

On-field Heart Rate (HR) monitoring has been proposed as a simple metric to measure the physiological stress experienced by different player positions. This kind of technology could provide teams an advantage to win matches giving coaches information regarding the effectiveness of the training regime. Consequently, it would be possible to optimize the training leading to practices that replicate game-like situations, minimize the occurrence of injuries, or improve the performance of the players [34].

In the study by Kate S. Early et al. [34], twenty-three NCAA Division I football players, grouped by defensive or offensive positions and by being linemen or skill players, were studied thanks to HR monitors (Bioharness 3.0 Zephyr Technologies, Annapolis, MD, USA). The authors considered both HR and activity level, computed as an average of the three directions of accelerations (figure 16).

Two reviewers analysed the burst in the Heart Rate and a MATLAB program was used to identify bursts of activity whenever a positive 10 bpm variation in 60 s happened. Mean HR, peak HR, time-

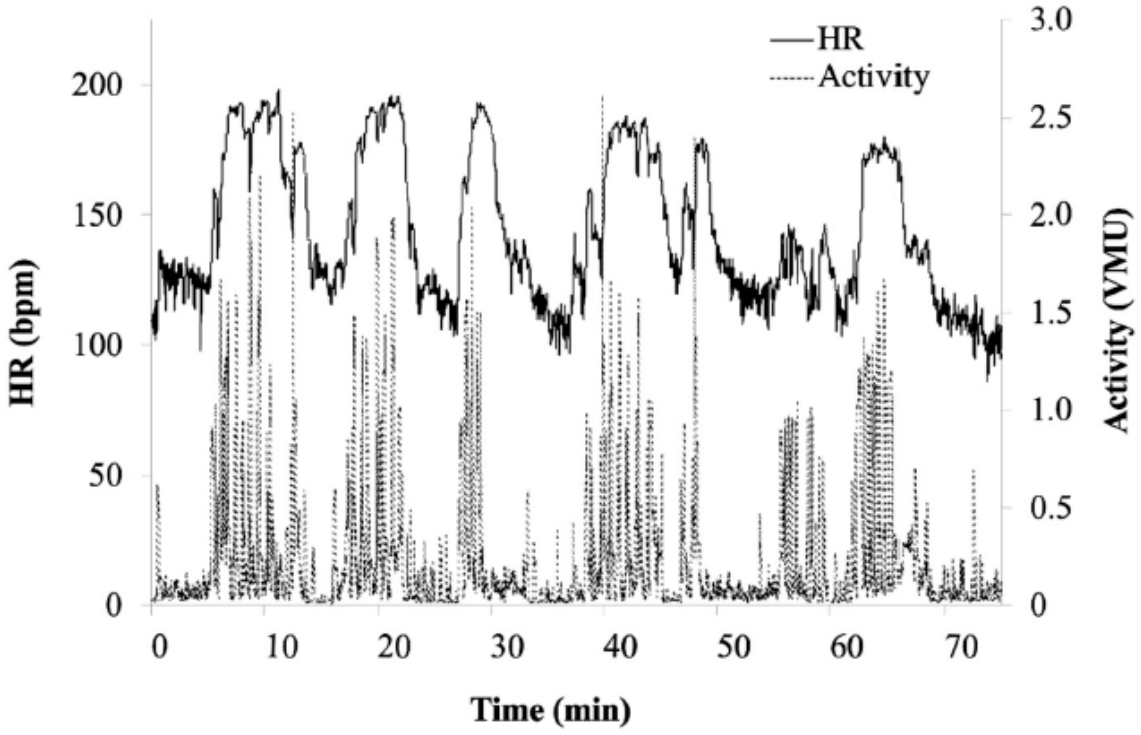

Figure 16. Example of a recording of heart rate and activity levels during a training session

to-peak HR, mean activity, and integrated activity were the parameters extracted from each burst. An

average number of three bursts per player was identified with a duration of 8.1 ± 3.9 min. The average HR peak was 182 ± 11 bpm, the mean HR was 157 ± 12 bpm, and the mean activity was 0.30 ± 0.05.

The burst analysis was shown to be reliable between the reviewers, meaning that for a given player it is possible to understand and quantify his activity, which could be beneficial to comprehend the player's positions physiological demands to better prepare them. Furthermore, a better understanding of the player workload could help athletes to enhance their performance and could aid coach decision-making during a game, such as giving more rest to players in a series of plays.

Moreover, an analysis of the HR distribution was conducted (figure 17). Players tended to have a moderate level of HR for most of the training with 16.7% ± 6.7% of the time at 60-65% of max HR and 15.1% ± 5.4% of the time at 65-70 % of max HR. Nevertheless, another typical interval of HR was between 85 and 90% of max HR, corresponding to high intensity, for 8.2±3.5 % of the time. This bimodal distribution is probably an expression of the intermittent nature of American Football, where high-intensity activity alternates with periods of rest.

Offensive players were more inclined to be at moderate intensity when compared to defensive players; the authors suggest that this is probably due to offensive players remaining at a higher

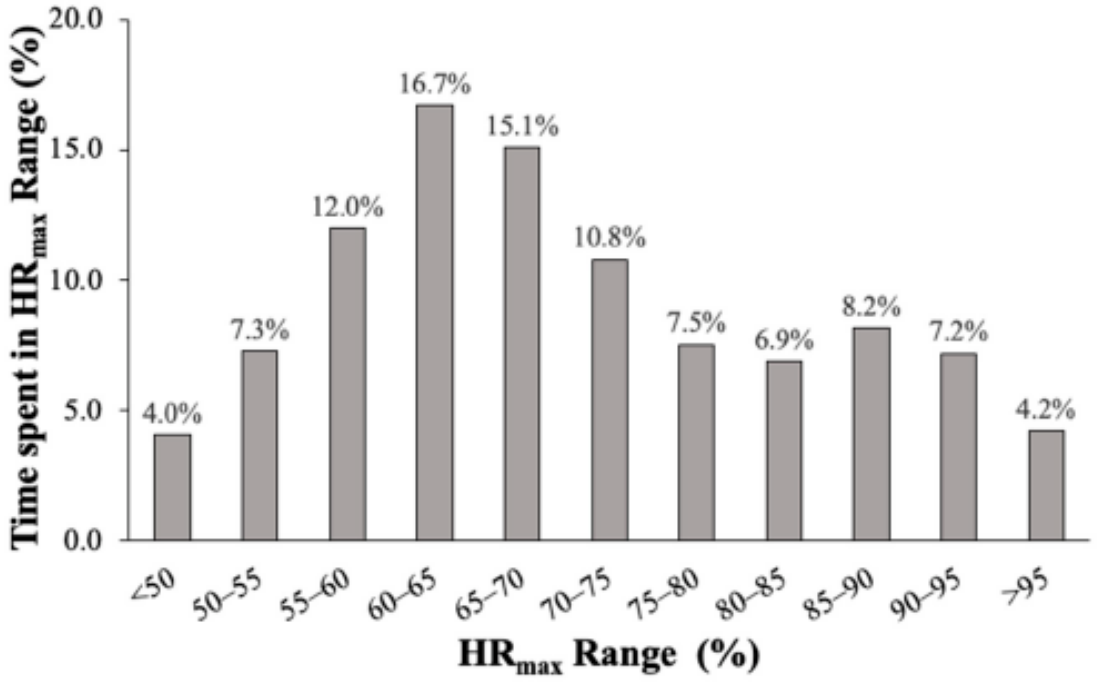

Figure 17. Hear Rate distribution in the sample of athletes

intensity pattern during the play whereas defensive players depended on short sessions of strenuous

activity. This pattern of activity reflects what is the role of a defensive player, who should be able to be reactive and respond immediately to the unknown strategies employed by the offense.

Additionally, defensive players had higher HR mean and HR peak suggesting higher cardiovascular effort than their offensive counterparts. This could be justified by a general better CV fitness of the offensive players, which are more accustomed to greater exercise intensity for prolonged distances during a training session.

Monitoring Recovery

Another application of cardiac monitoring is in the evaluation of Heart Rate Variability to understand if recovery of cardiac-autonomic activity to baseline occurs between training sessions [35]. A sample of twenty-five football players coming from a Division I college was analysed for each training day of a 4-week pre-season training session. The training load was measured from a wearable sensor recording the accelerations of the players in three directions and the extracted parameter was the square root of the sum of the squared values of the instantaneous rate of change of accelerations in each direction. Heart Rate Variability (HRV) was obtained 60-90 minutes before any practice from a finger sensor applied to a tablet for 1 min and the extracted parameter was the natural logarithm of the root mean square of successive RR intervals (lnRMSSD). The baseline logarithmic measure (lnRMSSD_BL) was obtained from an average of the intraindividual logarithmic measures of week two, where the athletes trained Monday, Wednesday, and Friday. On the other hand, whenever the Saturday training was added, the rest time from the previous Friday practice was about 20 hours, and the measurement was named lnRMSSDpost20.

The authors reported that for linemen the lnRMSSD_BL was significantly higher than lnRMSSDpost20, whereas skill players were almost fully recovered to baseline. This outcome indicates an inadequate cardiovascular recovery for linemen between sessions which could be justified by the lower cardiovascular and aerobic fitness of linemen. Moreover, a negative relationship was found between individual change in lnRMSSD and body mass, indicating that the decrement in lnRMSSD was higher among players with larger body mass.

An observational case report of a concussed American Football player taken from the previous study sample highlights post-concussion changes in HRV [36]. The authors found a meaningful reduction in mean lnRMSSD and a higher coefficient of variation after the concussive event with a trend that was similar to one of overtrained athletes. Additionally, they observed greater fluctuation of the

lnRMSSD after a concussion. They concluded that multiple HRV measurements after a concussion could be a simple alternative to tracking the recovery status of the athlete.

Prediction of core temperature

Heart Rate, measured by a Zephyr sensor, was employed to compute core body temperature thanks to the procedure proposed by Buller et al. [37] and it was validated against a gold standard being an ingested temperature pill [38]. Monitoring core body temperature could be important to prevent exertional heat illness.

A sample of 13 Division I football athletes wore the HR sensor, and 134 measurements were collected among all of them. The estimated body temperature was on average slightly lower than the gold standard with a difference in Fahrenheit of -0.195 (95% CI: -0.333; -0.05) (figure 18). The upper and lower limit displayed in the figure were respectively 1.42 and -1.803.

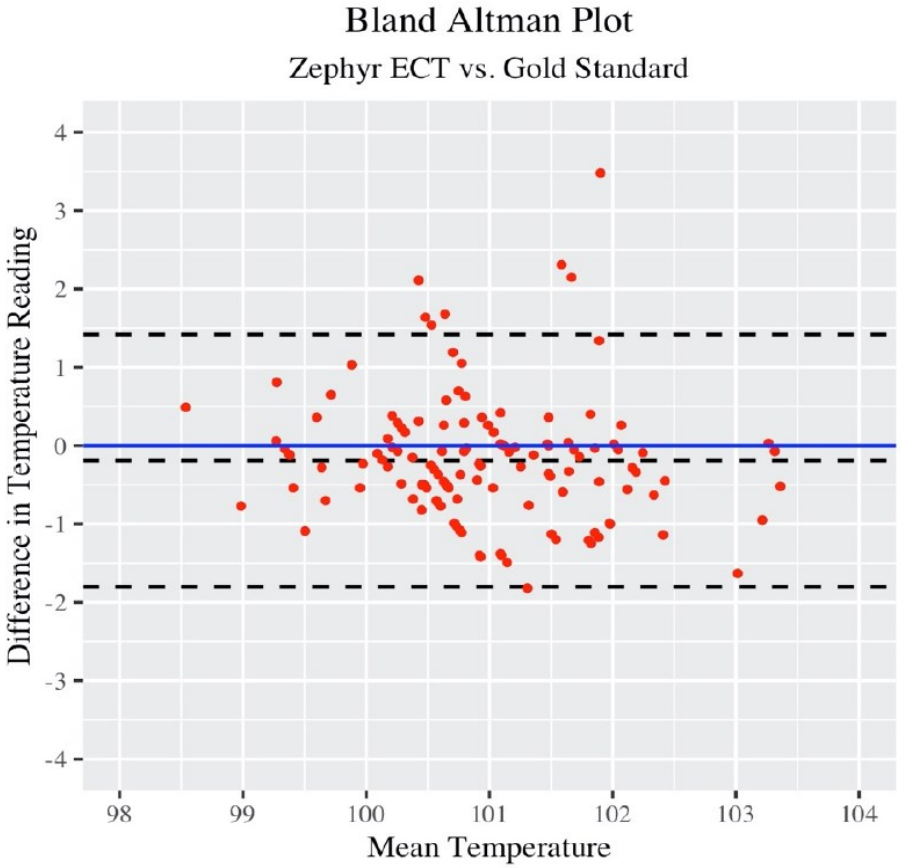

Figure 18. Bland Altman Plot showing the difference between the Estimated core temperature and the gold standard. The dashed line in the middle represents the bias and the other two dashed lines the upper and lower limit or the range where 95% of the differences are expected to fall

The authors stratified their analysis also based on body fat percentages considering the two classes of low body fat percentage and high body fat percentage based on the threshold of 6% of body fat percentage to understand if this variable was related to the accuracy of the estimation. They reported a mean bias that was slightly higher for the higher body fat percentage, but the range of variation, quantified as the difference between upper and lower limit, was lower in that case indicating a higher precision of the estimate for higher body fat percentage.

The concordance values reported for upper BF was 0.834 (CI: 0.534-0.731); instead for the lower BF it was 0.515 (CI: 0.336 - 0.659) (figure 19). The authors concluded that body core temperature assessment through HR was a feasible alternative for the monitoring of heat stress during training, however, they suggested that further research is needed to optimize the estimation in relation to body composition.

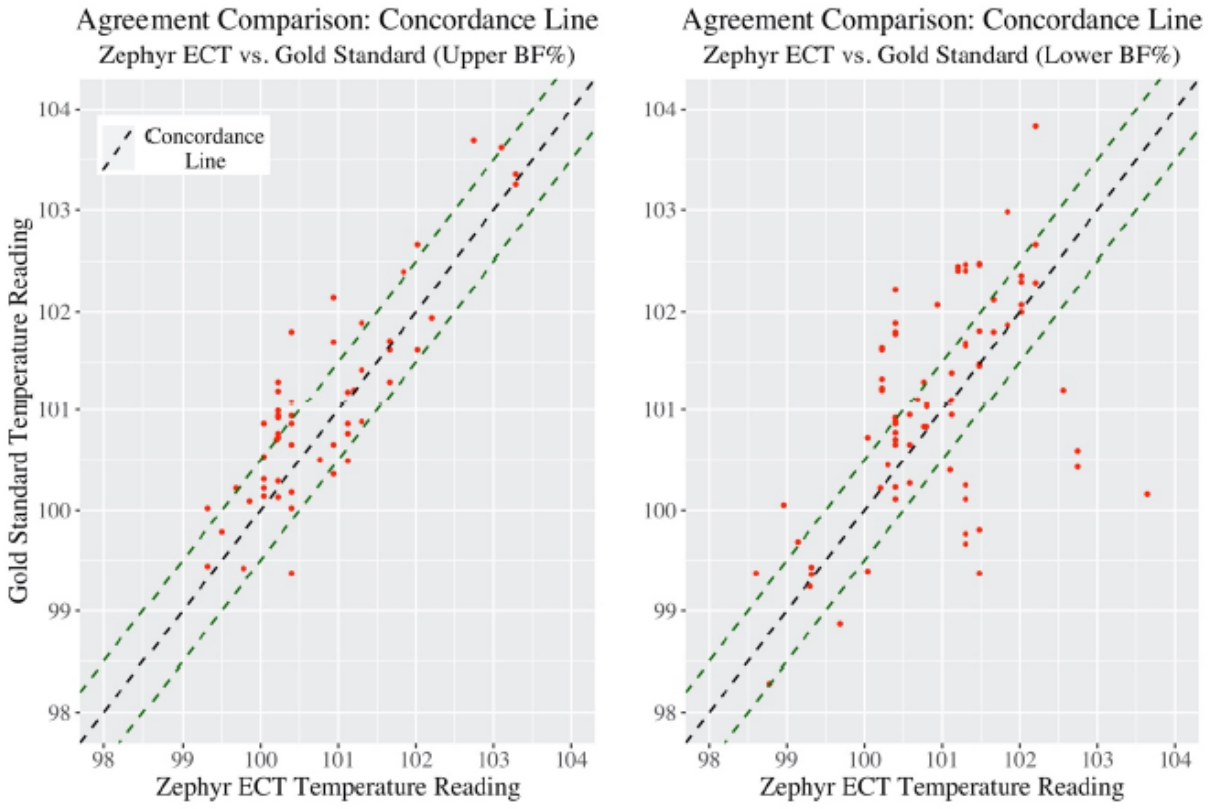

Figure 19. Concordance lines in the two considered subgroups

Chapter 3. Biomechanics and American Football

3.1 Biomechanics of American Football-specific movement

3.1.1 The football throw

The football throw biomechanics was firstly studied by Rash and Shapiro [39] who analyzed the throwing motion of 12 collegiate American football quarterbacks during the seasons 1990, 1991,1992. The data was collected directly on-field where the quarterbacks were instructed to drop back ten yards into a volume measurement where two 60-Hz cameras (figure 20) were used to record the subjects. A total of thirty frames were manually digitized with the ball release being the 20th frame for each athlete. The obtained data was then low pass filtered with a 10Hz cutoff because they observed that most of the power of the signal was below that frequency threshold during a pilot study in the laboratory.

The authors defined a local coordinate system for the Hand-Ball Segment, the Forearm segment, and the Upper arm segment.

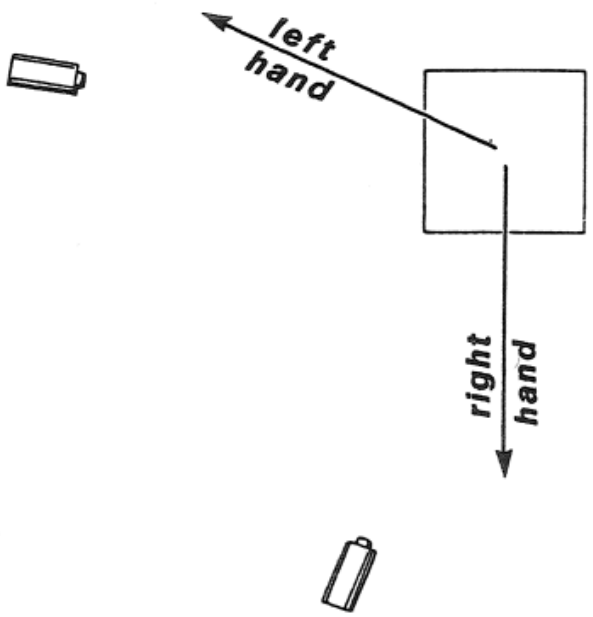

Figure 20. Diagram describing the placement of the video-cameras which aimed at the displayed volume of measurement

The reference values of the four considered joint angles are shown in figure 21 and the results for the angular displacement are shown in figure 22. They reported that from the Average Foot Contact (AFC) through the middle of follow-through (the phase after the release) the quarterbacks showed only minor changes in the kinematics and in particular the following characteristics:

- a peak of external rotation, which is shown in the figure as an average between the contributions of the different quarterbacks (AMER).
- a slight shoulder abduction from AFC to Average Maximum External Rotation (AMER) and evident adduction in the follow-through phase
- a steady horizontal adduction which increases its trend in the follow-through phase. In some cases, the movement switched to abduction around the AMER due to increased trunk rotation
- a slight elbow flexion around the AFC and an accentuated extension from AMER to Release and until 126% of the throw.

The angular velocities were consistent among the trails and the average is reported in figure 23. The main patterns were the following:

- Both the shoulder and horizontal abduction/adduction velocities were close to zero between AFC and release and in both cases a maximum adduction velocity was observed in the follow-through phase.
- There was a high shoulder external rotation until midway between AFC and AMER and then a steady increase of internal rotation until a peak reached after the release at 115±6% of the throw.
- A peak of elbow extension velocity was noticed at 103±4%

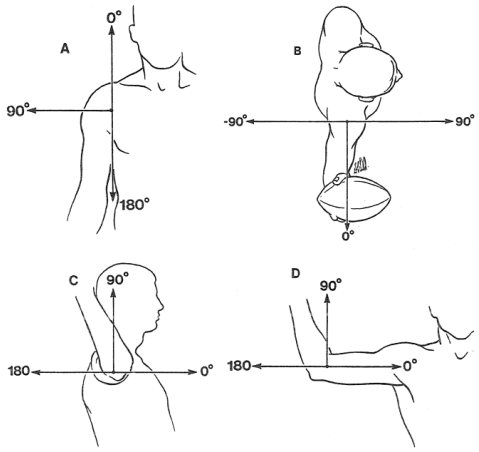

Figure 22. The reference values of the four considered joint angles; (a) shoulder abduction, (b) horizontal adduction, (c) external rotation, and (d) elbow extension

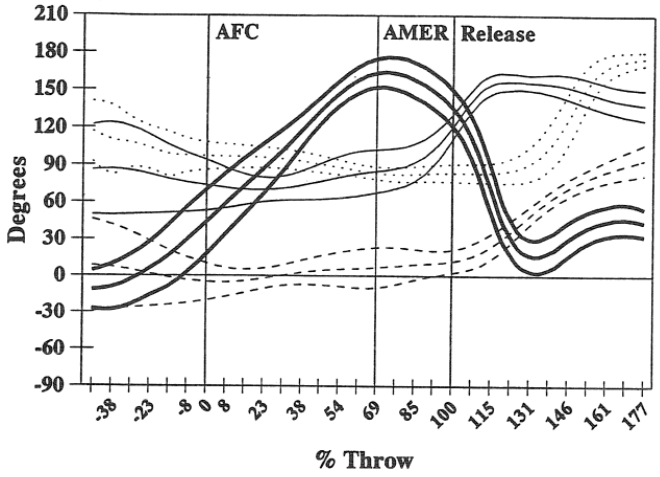

Figure 23. Angular displacement curves ± standard deviations: elbow ext-flex (thin solid), add-abd (dotted), horizontal add-abd (dashed), ext/int rotation (thick-solid).

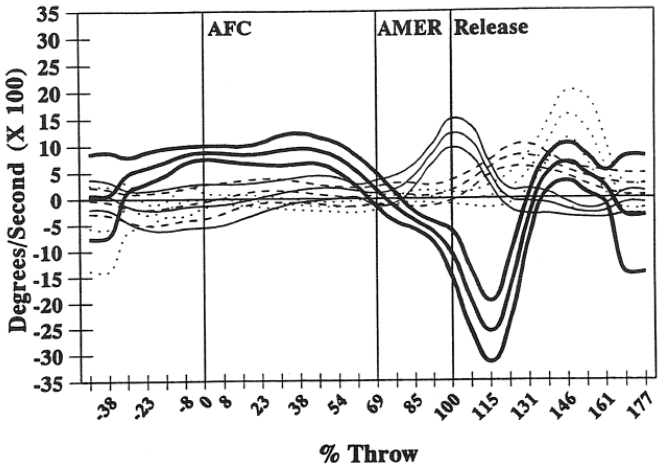

Figure 21. Mean curves of angular velocities -+ standard deviations: elbow ext-flex (thin solid), add-abd (dotted), horizontal add-abd (dashed), ext-int rotation (thick solid).

3.1.2 The long snap

The long snap consists of the ball being thrown backward between the legs of the long snapper, which is the specialized linemen for this specific movement. The football is in this way passed to the punter, who has the responsibility to kick it forward for the change of possession. A study By Michael G. Chizewski and Marion J.L. Alexander [40] analyzed the kinematics of this movement in ten players coming from high school and ten from university. The athletes performed different trials where they had to snap the ball to a target simulating a punter placed at 14 yards and only the fastest and most accurate release was taken into consideration for each player.

They aimed to understand the correlation between independent kinematic variables and the performance of the athletes in terms of accuracy and speed of the release. The necessary variables were computed with a video analysis coming from a 4-camera setup.

The authors measured a total of thirty-seven variables during the force production phase, where the long snapper throws the ball back until the instant of release. Three were the variables found to be significantly different between High school (HS) and university (UNI) athletes:

- Maximum angle of left elbow flexion, with a mean of 62.36° for the HS group against 80.23° for the UNI group
- Left elbow extension range of motion, with the high school athletes experiencing a mean 46.61° of range of motion, whereas the athletes from university experienced a 67.93° range of motion.
- Left elbow velocity, which was significantly higher for the UNI group with 751.99°/s against the 498.10°/s of the HS group

Then, during the release phase, nine measured variables were significantly different between the two groups as shown in table 1.

Table 1. List of the 8 variables found to be significantly different between the two populations. The ninth variable is the total distance given by the sum of right and left heel distance.

	High school athletes (N=10)		University athletes (N=10)		
Variable	**Mean**	**SD**	**Mean**	**SD**	**p-value**
Right heel distance covered (m)	0.15	0.086	0.30	0.081	0.0011
Left heel distance covered (m)	0.17	0.097	0.29	0.082	0.0055
Mean distance covered (average of both the feet) (m)	0.16	0.091	0.30	0.081	0.0024
Total flight time of the football (s)	1.04	0.13	0.87	0.052	0.0011
Total snap time of the athlete (s)	1.25	0.13	1.08	0.060	0.0025
Release height of football (m)	0.17	0.016	0.13	0.022	0.00052
Release height of football (% of snap height)	9.10	0.82	6.98	1.30	0.00025
Release velocity of ball (m/s)	13.21	1.41	15.15	0.79	0.00069

Eventually, they performed a regression analysis on all the population with the release velocity of the ball being the dependent variable and four chosen predictors, which were total snap time(*x1*), the height of the player(*x2*), left elbow extension velocity(*x3*) during the force production phase and right elbow flexion in backswing position(*x4*).

$$y = +17.438 - 8.201x1 + 3.180x2 + 0.022x3 - 0.021x4 \quad (3)$$

Given a coefficient of 3.180 for the subject's height, an increase of 10cm in the height of the player would predict a 0.30 m/s increase in velocity. The authors suggest that height could be important because taller athletes have usually longer arms, therefore having a greater swinging radius to provide greater velocity. On the other hand, backswing right elbow flexion and TST were negatively correlated with release velocity. In the first case, for every 5° increment of flexion, there will be a 0.1m/s drop in predicted velocity, which suggests that players should extend the right elbow when

they prepare for the long snap. The other variable indicates that a faster snap is beneficial, and it will result in faster balls.

Moreover, the authors suggested that an increase in release velocity is associated with a decrease in release angle and height.

3.1.3 Comparison of different starting stance techniques

A biomechanical analysis of three different starting positions, which are common in the sport of American football, was performed by Bruno Bonnecher et al [41] to understand the safest starting position concerning Sport-Related Concussions. Their analysis was conducted on a sample of twelve football players and thirteen athletes playing other sports. The subjects were asked to perform three sprints starting from three different stance positions shown in figure 24.

A stereophotogrammetric system was used to record joint motion and displacement (figure 25). Trunk inclination relative to the floor and the angle between head and trunk were computed and used to assess the verticality parameter as follows:

$$Verticality = (180 - Trunk\ inclination) + (180 - Trunk\ head) \quad (4)$$

Then, the authors defined the field of view (FOV) as a ratio between head height and verticality as follows:

$$FOV = Height\ of\ the\ head\ (\%) / Verticality\ (°) \quad (5)$$

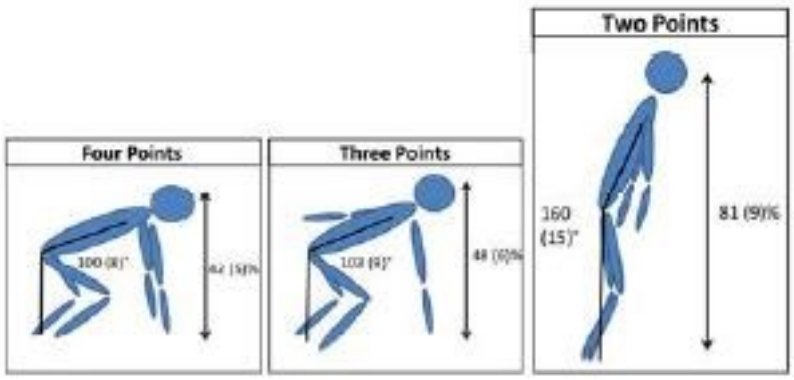

Figure 24. The three stance positions used in the study

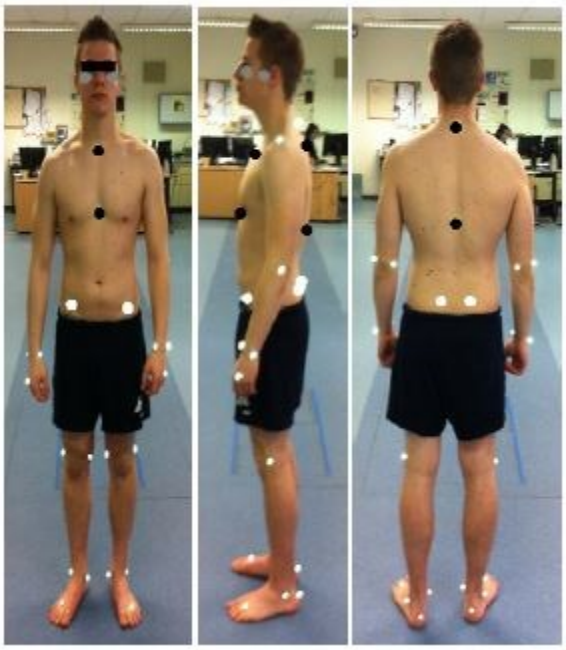

Figure 25. Marker positions for the motion analysis

The start and end time of the motion were evaluated thanks to the range of motion of the head with the start time corresponding to the instant of initial head acceleration and end time corresponding to head height in a standing position. The total excursion of time was called redress time and the head velocity and acceleration were computed based on head height and redress time. Body speed was assessed 1 s after the end time of the starting sprint. Eventually, the authors quantified the kinetic energy that would produce an impact between two players having different starting techniques.

Their results showed that a 4-point stance seems to be a more dangerous starting stance technique because of the reduced FOV and the higher head and body speed. A reduced FOV would imply less awareness of the positions of the surrounding players on the field. The 4-point stance had the significantly lowest FOV with a value of 0.35 ± .05 compared to 0.42 ± .07 for the 3-point and 1.56 ± 0.43 for the 2-point stance. The redress time between the three starting techniques were not significantly different, because head speed was significantly greater for the 4-point techniques with an average of 0.87 ± 0.11 m/s against 0.78±0.13 and 0.27±0.14 for 3 and 2-points, respectively. The body speed was not significantly different between 4- and 3-point stances with respective values of 2.67±0.55 and 2.61±0.56, where the body speed for the 2-point technique was significantly lower with a value of 2.33 ± 0.52.

The higher head speed and body speed translates to higher kinetic energy applied to the head during impacts and more dangerous collisions. Thus, the 4-point technique was the least safe and the authors suggest that there is a certain association between player start position, player FOV, and the collision energy experienced after the starting sprint.

3.2 Biomechanics of the foot in American Football Athletes

Patrick O. Riley et al. [42] analyzed the foot kinematics of American football athletes during three NFL combine tasks: 3-cone drill, shuttle run, and standing high jump. They recruited nine athletes distributed in various positions and they asked them to perform the football-specific tasks with a particular focus on eleven events during which the players were instructed to hit a force platform.

The events of interest were:

- Initiation and termination of the task with forward start and plant, lateral start, and plant, jump and jump landing
- Change of direction: 90 degrees right cut, 90 degrees left cut, reversal of the movement
- Running and Walking

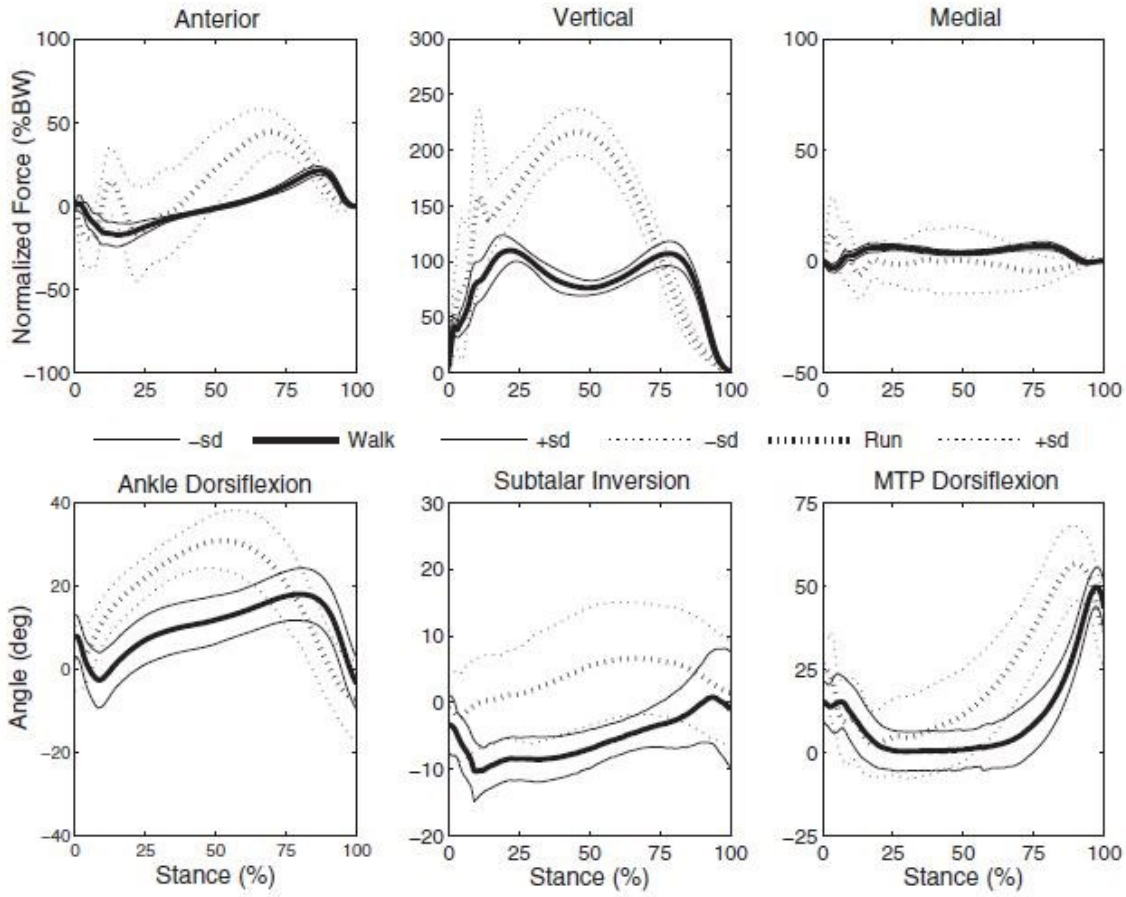

Figure 26. The three components of GRF and ankle and foot joint angles mean curves with the relative standard deviation; walking corresponds to solid lines and running to dashed lines. The force is positive anteriorly, upwards, and medially.

The authors used a foot model consisting of three rigid bodies: the talus, the midfoot, and the toes. A set of eight markers was used on the foot with redundancy for the midfoot segment. The rotations around the talocrural joint (TC), the subtalar joint (ST), and the metatarsal phalanx joint (MLP) were computed with reference to the orthostatic standing positions where the angles were considered equal to zero.

In figure 26 the graphs display the ground reaction components and the joint angles of TC, ST, and MTP for walking and running. The GRF components for running were like the ones found in the literature.

In figure 27 graphs showing the GRF and joint angles for the change of direction mechanism are shown. The vertical component of the GRF for the cuts appeared with a single peak analogous to the one found during running, whereas the reversal of motion exhibited two peaks similar to the walking pattern. In both cases, there was a peak of posterior braking force and for the reversal of motion, there was a second propulsive peak in the posterior direction. For the cuts, we can observe a large medial

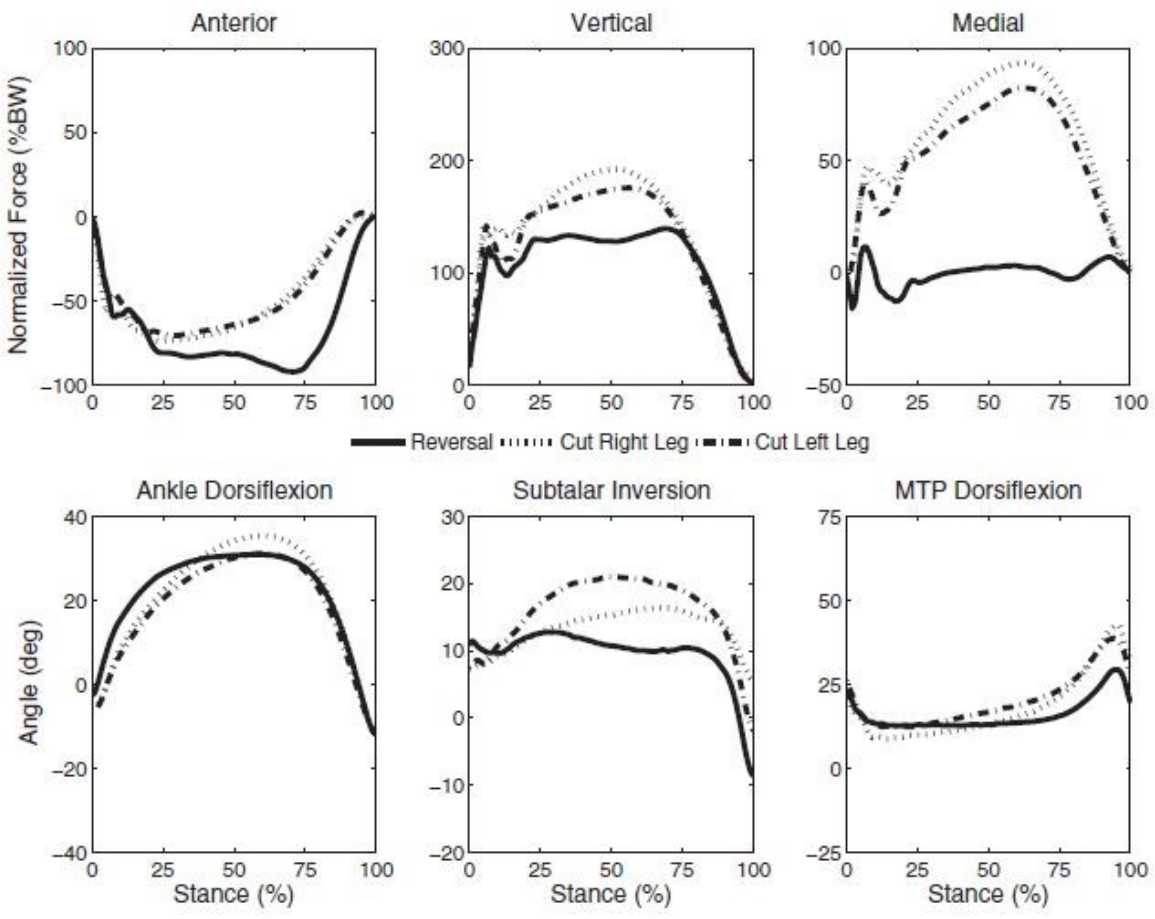

Figure 27. Mean curves of GRF components and joint angles for change of direction events. Reversal corresponds to solid line, cut to the left on the right leg corresponds to dotted line, cut to the right on the left leg corresponds to dot dashed line.

force applied during the late stance. For what concerns the joint angles, the ankle dorsiflexion, subtalar inversion, and MTP dorsiflexion were similar to running. However, the cuts had larger ranges for the subtalar inversion and the reversal of motion had a reduced range for the MTP dorsiflexion.

For the initiation and termination events, the GRF and joint angles are shown in figure 28. We can observe that the kinematic of termination and initiation are specular. For the forward start, a peak of an anterior force larger than 50% of body weight (BW) was reported, whereas for the lateral start the peak was found in the medial direction and with a higher value of around 100%BW. All the initiation mechanisms exhibited a similar peak in the vertical direction of 150%BW. We can notice that in all the movements the initiation is characterized by a rapid ankle plantar flexion and subtalar eversion. Moreover, the forward start was characterized by an evident MLP dorsiflexion.

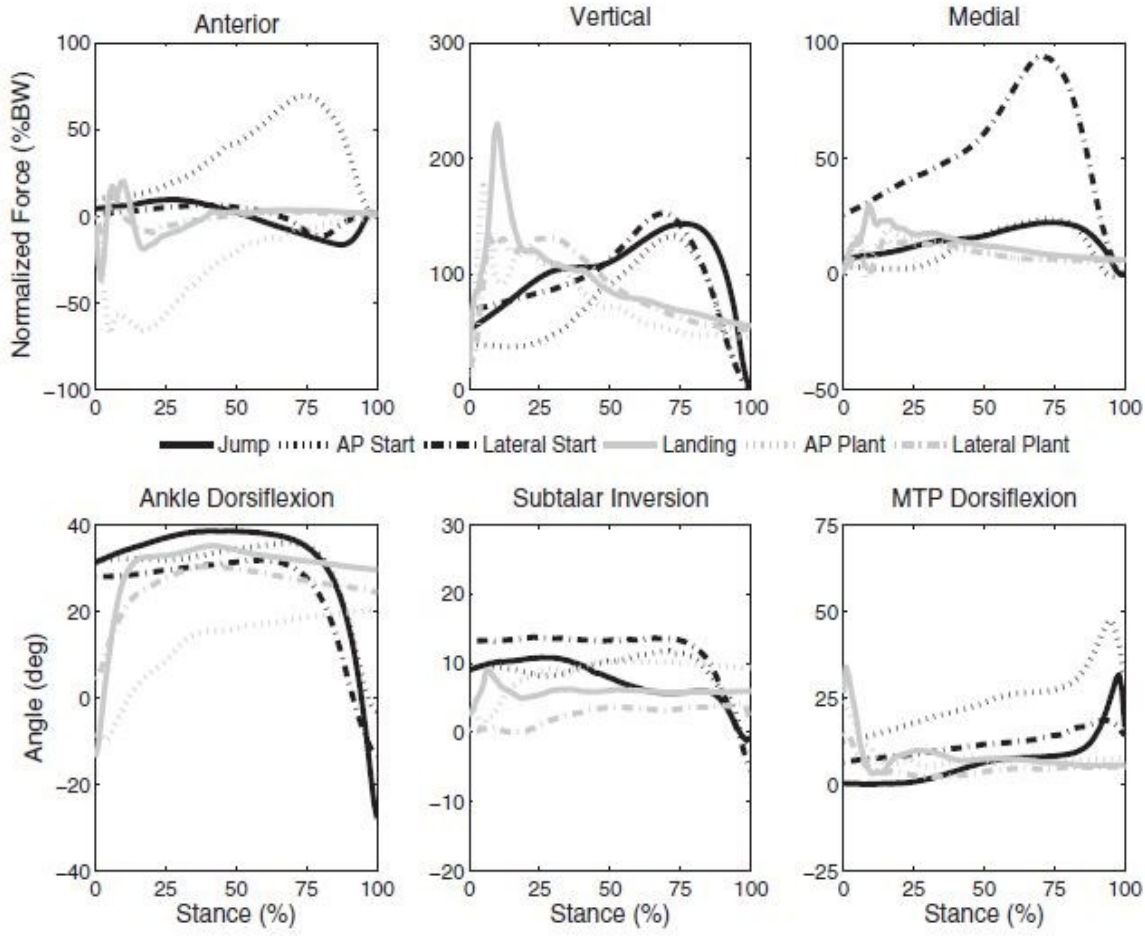

Figure 28. Mean curves for movement initiation and movement termination events. Jump, jump landing corresponds to solid line, AP start, AP plant corresponds to dotted line, lateral start, lateral plant corresponds to dot dash line. Black is associated with movement initiation, grey is associated with movement termination

3.3 Biomechanics of concussion

The measure of on-field concussion biomechanics has been an objective of research in contact sports such as American Football for almost 60 years [43] driven by the desire to develop better protection devices and adjust the rules to protect the players.

The first experimental procedures on the biomechanics of concussion in professional American football athletes were based on video recordings of concussive or non-concussive impacts that were then recreated in a laboratory (figure 29) using Hybrid III anthropometric test devices (ATDs) [44] [45].

An example is a paper [45] where thirty-one recorded head impacts were properly reconstructed in a laboratory reproducing the head kinematics. The measured translational velocity of the head of concussed players was 9.3 ± 1.9 m/s. The concussed player was characterized by higher variation in head velocity with 7.2 ±1.8 m/s against 5.0 ± 1.1 m/s ($p<0.005$) of the injured players and higher maximum acceleration with 98 ±28 g against 60+/- 24g for the average rotational acceleration was reported to be 6432±1813 rad/s^2 against 4029±1438 rad/s^2 for sub-concussive cases. The study concluded that a concussive event was mainly related to translation acceleration and hits given to the front or the side of the face mask of the helmets.

However, the limitations of these reconstructions are several and resumed into the following points [43]:

- mistakes in the determination of impact velocity or location from the video
- the degree to which ATDs are similar to the reality
- datasets that largely include concussive events
- low frame rates of the video recording
- video methods enable to understand just the velocity of the helmet

Figure 29. Example of laboratory reconstruction [44]

3.3.1 Head Impact Telemetric System

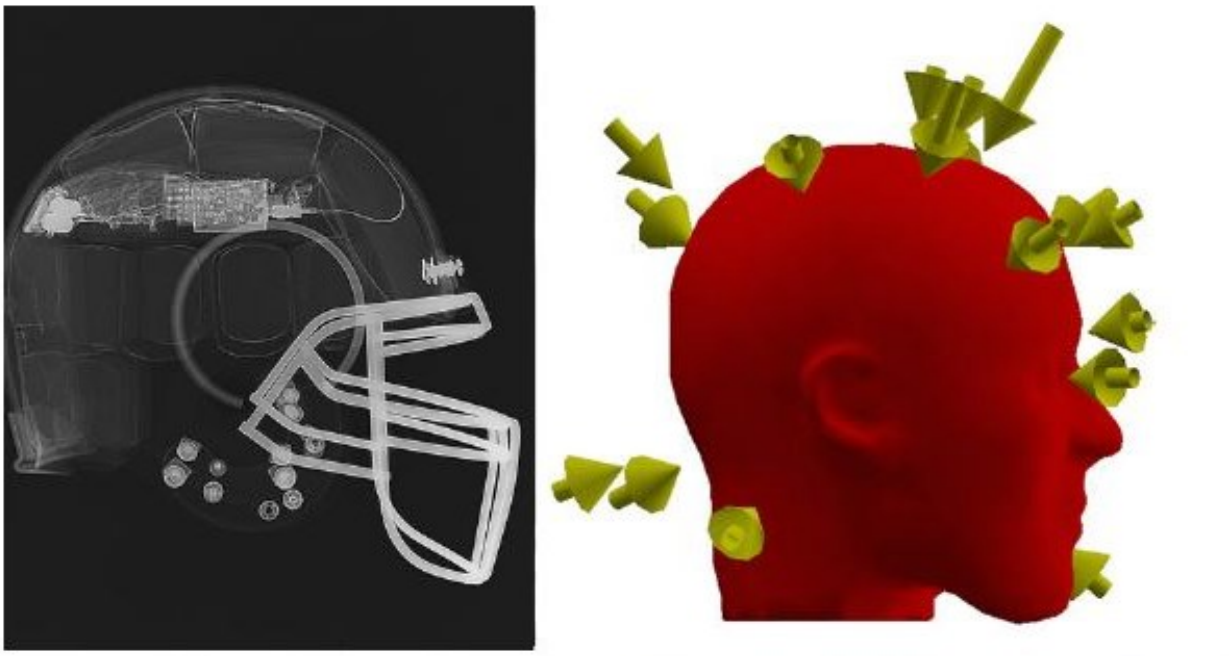

Figure 30. X-ray view of the HIT system inside a helmet on the left and the measured impacts on the right [48]

The Head Impact Telemetry (HIT) System is a technology formed by six spring-mounted accelerometers, placed in direct contact with the head, and hardware parts to acquire data and transmit it remotely, which are inserted in commercially available helmets (figure 30). This technology enables to make the football field a living laboratory [46] allowing for the collection of large amounts of data concerning the frequency, position, or magnitude of collisions [43], during game or practice situations.

The first application of this system was in collegiate football players [47], where it was validated against impact tests using Hybrid III dummies equipped with a configuration of a 3-2-2-2 head accelerometer. The HIT system had a 4% error in the estimation of linear acceleration, rotational acceleration, and Head Impact Criterion (HIC) values. For the impact location instead, the average error was ±1.20 cm. Further validation of the HIT system against the gold standard being laboratory reconstruction is given by [46], where the HIT system outputs were reported to be correlated with the output obtained by ATD in conditions similar to the ones happening in the NFL. The measurements of the two approaches were applied to simulated impacts and the peak linear acceleration for all the considered impact locations was strongly correlated using a linear regression model with an average overestimation of the HIT system of 0.9% when compared to the laboratory measurement. The instrumented helmet measurements were also correlated with rotational acceleration for three of the four considered locations of impacts with an average underestimation of 6.1%, mainly due to the

frontal impact which was not correlated. The authors also reported a correlation between the approaches in the estimation of Severity Index (SI) and Head Impact Criterion (HIC), even if there was no correlation for frontal impact. Thus, the HIT system demonstrated to be a reliable system for in-vivo measurements.

From the analysis reported by Duma et al. [47], thirty-eight college players were analysed for a total of thirty-five practices, ten games, and 3312 head impacts. The average peak linear acceleration was reported to be 32g ± 25g with 89% of collisions being under 60 g. The average SI and HIC were respectively 36 ± 91 and 26 ± 64. The mean rotational acceleration around a medial-lateral axis of the head was 2020 ± 2042 rad/s2, whereas the mean rotational acceleration around an anterior-posterior axis was 905 ± 1075 rad/s2. They recorded just one concussive event with a linear acceleration of 81 g, an SI of 267, a HIC of 200, and a rotational acceleration of around 5600 rad/s2 in both the considered axis.

At the high school level, Broglio et al [48] analysed the head impacts with the HIT system in thirty-five high school players during a season consisting of fifty-five practices and thirteen games. They recorded a total of 19.224 impacts with an average of 15.87 ± 17.87 impacts experienced by players in a single session. The mean number of impacts during games was significantly higher ($p < 0.01$) than during practices with 24.54 ± 22.41 and 9.16 ± 8.64 respectively; the linemen were the ones to experience the greatest number of impacts. Stratifying helmet impacts for location, the collisions to the front of the helmet were the most frequent. Moreover, game impacts had a statistically higher linear acceleration (24.76±15.72 g) and rotational acceleration (1669.79 ± 1249.41 rad/s2) with respect to practice (23.26 ± 14.48 g and 1468 ± 1055.00 rad/s2). A difference was even found for player positions with defensive linemen experiencing significantly higher linear acceleration than defensive skill and offensive linemen, whereas a significantly higher rotational acceleration was found in linemen when compared to all the other positions.

Always in high school, similar monitoring was performed by Broglio et al. for a larger sample of athletes [49] during four competitive seasons from 2005 to 2008. A total of 54247 impacts were recorded across 128 practices and thirty-eight games. The mean linear acceleration was 25.1 ± 15.4 g and the mean rotational acceleration was 1627.1 ± 1182.9 rad/s2.

The use of this technology in college and high school athletes gave the possibility to create large datasets such as the one used by [50] where data from more than 60.000 impacts were employed to

validate their analysis. From this dataset, the average accelerations of concussive impacts were 104 ± 30 g and 4276±1931 rad/s2, respectively for linear and rotational dynamics. In sub-concussive impacts, the average linear acceleration was 26±19 g and the rotational one was 1072±850 rad/s2.

Generally, the mean linear acceleration in both college and high school athletes seems to be approximately around a value of 25 g, however, there is a difference in the distribution of magnitude for the impacts with the college athletes experiencing higher impact acceleration than high school athletes [48].

3.3.2 Concussion risk with biomechanical data

Concussion biomechanics can be applied to both injury prediction and prevention [48] thanks to the recognition and validation of biomechanical parameters or injury metrics useful for the identification of injured players or the development of new and better protective gear, such as helmets. Injury metrics are at the basis of safety standards and performance quantification for sports equipment. The National Operating Committee on Standards for Athletic Equipment (NOCSAE) has based its impact test validations on the severity index (SI) [51], defined as the integral in time of the linear acceleration to a power of 2.5 and being an expression of the head impact severity on the anterior-posterior axis [52].

$$\mathrm{SI} = \int a^{2.5} dt \tag{6}$$

In this way, the occurrence of clinically relevant head injury was cut down [43], however as shown in laboratory reconstructions [44] the upper limit threshold for SI, considered by the NOCSAE at 1200, could be too high in particular for lateral head impacts. For this reason, the development of new and appropriate metrics is fundamental for the protection of football players and to reduce the risk of concussive events.

One of the first injury metrics based on both linear and rotational acceleration was HIP, head impact power, proposed by Newman [44] and evaluated on twenty-four head impacts reconstructed in the laboratory and against different predictive measures of mild traumatic brain injuries. The HIP definition is the following, where a stands for linear accelerations and α stands for rotational acceleration:

$$HIP = A\,a_x \int a_x\,dt + B\,a_y \int a_y\,dt + C\,a_Z \int a_Z\,dt + D\,\alpha_x \int \alpha_x\,dt + E\,\alpha_y \int \alpha_y\,dt + F\,\alpha_Z \int \alpha_Z\,dt \tag{7}$$

It is a measure given by the sum of each acceleration component times the integral of the same component in time times a weight given by the mass or the moment of inertia along the direction of the component. HIP demonstrated higher significance than the other measures to discriminate concussions from sub-concussions.

An analysis of a criterion based on thresholds for rotational acceleration and linear acceleration was conducted by Broglio et al. [49]for a sample of impacts from high school players recorded with the HIT system. Using a classification tree, they found that resultant linear acceleration, rotational acceleration, and impact location were good predictors of concussion. They defined any impact to the front, side, or top of the helmet over the threshold of 5582.3 rad/s2 for rotational acceleration and over the threshold of 96.1 g for linear acceleration as at substantial risk of concussion.

The study by Rowson s. et al [50]. proposes a new injury metric, the combined probability of concussion CP, based on both the peak linear and rotational accelerations to assess concussion risk.

$$CP = \frac{1}{1+ e^{-(b0+b1\ a+b2\ \alpha+b3\ a\ \alpha)}} \tag{8}$$

The authors used multivariate logistic regression analysis on a dataset of 63.011 head impacts monitored with Head Impact Telemetry systems. To account for the know unreported cases of concussion, they also determined a concussion incidence rate of 38.8 concussions per 10.000 impacts from the literature, and 207 sub-concussive impacts were reassigned to concussive events to reach 244 concussions in compliance with the incidence rate. The novel metric was also validated against a dataset of reconstructed impacts [45].

The Receiving Operating Characteristics for the CP were the highest when compared to linear acceleration and rotational acceleration metrics, however, the AUC of the CP was significantly higher than only the rotational acceleration AUC. The CP metrics was also the one that produced the lowest number of false-positive rates. (Figure 31)

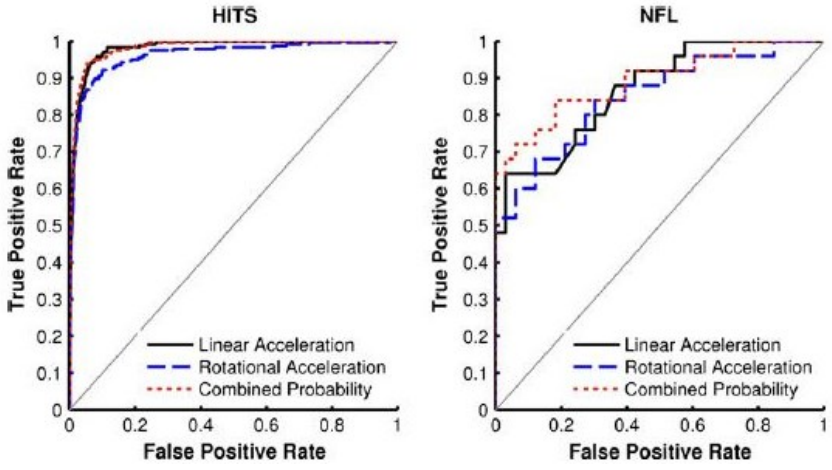

Figure 31. AUC graphs for the two considered datasets

3.3.3 Mouthguard sensor

One of the problems of the HIT system is that the accuracy of the measurements is worsened by the relative motion between helmet and head [48]. Thus, researchers have proposed alternative methods for the monitoring of athletes, such as mouthguard sensors, which were reported to have fewer displacements from the skull when compared to skull and skin sensors [53]. Another advantage of mouthguard sensors is the fact that they can be applied also to sports where helmets are not used.

The use of mouthguard sensors has not been extended to on-field monitoring, but it is in a phase of validation. An example is given by a study [54] where a mouthguard sensor was validated with benchtop tests, in vitro tests, and in vivo tests. In the first case of benchtop tests, the mouthguard sensor was compared to an accelerometer in the evaluation of linear acceleration after a drop test and it was compared to an angular rate sensor in dynamic rotation tests. For the linear acceleration, the prediction of the mouthguard sensor had an R2 of 0.99 when compared to the accelerometer. For the rotational acceleration, the authors introduced a correction algorithm to adjust the estimation of the internal gyroscope of the sensor and the outcome of the mouthguard sensor had also an r squared of 0.99.

The in vitro tests were performed on ATDs against a gold standard configuration of accelerometers 3-2-2-2 with a setup reproducing an American Football player and another setup reproducing a boxer athlete. They also reported a linear model, which fits data coming from the reference gold standard and the mouthguard sensor with values of R-squared of 0.98 or 0.99 (figure 32 and figure 33). The

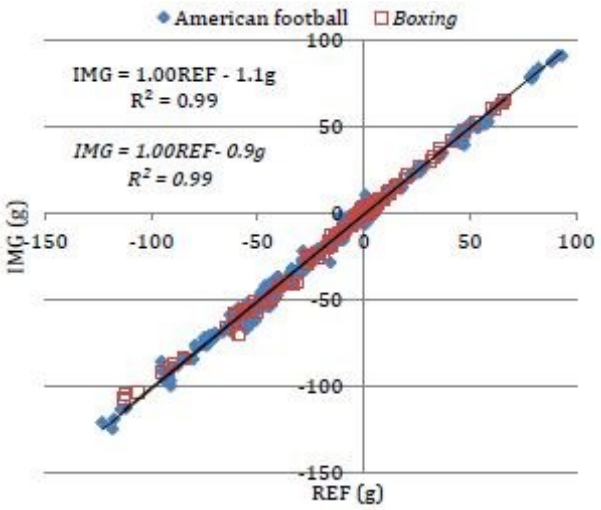

Figure 32. Linear fit between the mouthguard measure of linear acceleration and the gold standard configuration of 3-2-2-2 accelerometers. In both the simulated cases of American football and boxing

results suggested that the mouthguard sensor could be effectively use for the measurements during training with good accuracy.

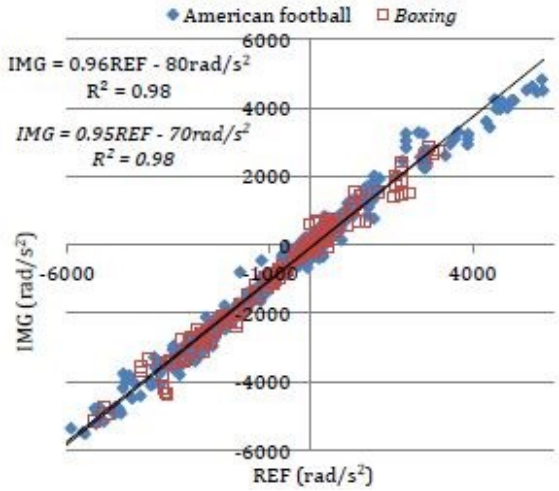

Figure 33. Linear fit between the mouthguard measure of rotational acceleration and the gold standard configuration of 3-2-2-2 accelerometers. In both the simulated cases of American football and boxing

3.4 Common Injuries in American Football

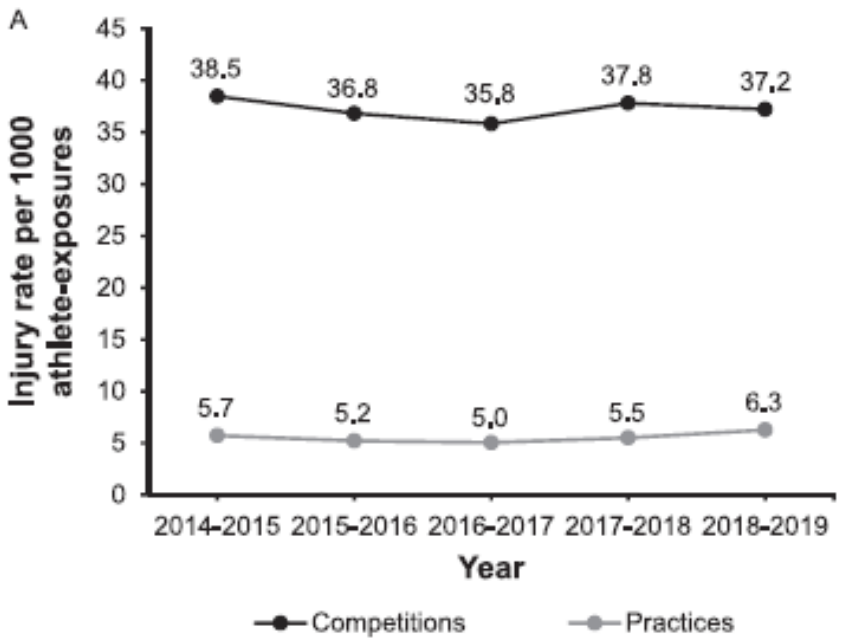

Figure 34. Diagram showing the injury rate per year in the NCAA football during practices and games

American Football has one of the highest injury rates among different sports with the average game injury rate of 35.9 injuries per one thousand Athlete-Exposure(A-Es) against 13.8 taking the average of fifteen sports included in the study by Jennifer Hootman et al. [55]. On the other hand, the mean injury rate for practices seems to be similar to other sports at around 4 per 1000 A-Es [56] [55].

In a recent epidemiologic study conducted in the period from 2014 to 2019 by Avinash et al. [57], the injury rates for practice were always between 5 and 6 per 1000 A-Es, whereas the injury rates for games had a maximum of 38.5 in the first years and a minimum of 35.8 in the 2016-2017 season (figure 34).In both cases, the occurrence of injuries remained constant and the incidence in practice was significantly lower than the incidence in-game.

The most injured body parts were the knee (15.5%), shoulder (13.5 %), ankle (12.5%), and head/face (9.2%). The most frequent injuries were sprains (28.5%), strains (18.7%), contusions (13.7%) and concussions (7.5%). The major injury mechanism was player contact accounting for 50.9% of the reported injuries with a higher prevalence in games than in practices. Most injuries happened during general play (17.5%), blocking (15.8%), tackling (14.0%), or running (11.8%). The player position that was most exposed to the occurrence of injury were defensive backs, offensive lineman, and defensive linemen.

In the population of football players injuries of the hip/thigh/upper leg, knee and ankle were reported to be the most prevalent injuries from 1988 to 2019 and the major cause of their occurrence is usually player contact [56] [57] [58]. For the knee, there were three main kinds of injuries: Anterior Cruciate Ligament (ACL), Posterior Cruciate Ligament (PCL), and menisci [56]. During the period between 1988 and 2004 a deep analysis of the major injury mechanisms concerning the knee showed that the three main determinants of knee injuries were being tackled (16.8%), being blocked (15.0%), and a rotation about a planted foot (28.6%) [56].

The non-contact mechanism seems to be a crucial factor in the occurrence of knee injuries, in particular ACL. In a study where sixty-nine video recording ACL injuries in NFL players were analyzed, 72.5% of the injuries occurred due to pivoting or cutting when the hip was abducted or flexed, and the knee was in early flexion or abduction and the foot was in abduction or external rotation. [59]

3.4.1 Concussion

A concussion is a form of mild Traumatic Brain Injury (mTBI) defined as a head trauma that induces a form of impairment of brain function with a limited duration and severity. The possible loss of consciousness should not exceed 30 min, the initial Glasgow Coma Scale should give a score of 13-15 and the post-traumatic amnesia should not be greater than 24 hours [60].

It could be caused by the head being struck or hitting an object or by a rapid succession of acceleration and deceleration motion of the brain. Subjects with an mTBI could experience a wide variety of symptoms collectively addressed as Post-Concussion Syndrome (PSC), including physical symptoms, such as nausea, vomiting, and dizziness, cognitive limitations, such as in the level of concentration, perception, memory, language, or behavioral changes, such as irritability and depression [60] [61].

For example, in a review of concussion rates in high school and college sports in the 2005-2006 season, the most common reported symptoms were headache (40.1%), dizziness (15.3%), confusion (8.6%), loss of consciousness (3.9%) and amnesia (6.4%) [62]. Additionally, half of the concussed athletes had symptoms for 3 days or less and more than half of the athletes returned to play in 9 days or less.

American Football athletes are a population where concussions are a common injury [57] [58] being the 7.40% and 8.09 % of the reported injuries respectively in practices and games during the period lasting from 2014 to 2019 in the NCAA [57]. Similar were the percentages of concussions reported for the surveillance of the previous 10 years with 8.4% and 9.4% for practices and games, with the injury rate per 1000 A-Es being 0.40 and 3.01, respectively.
When compared to other sports in High School and College, American Football was the one with the highest rates of concussions in the 2005-2006 season with 40.5% of the total occurrence [62].

In a recent review of the long-term consequences of brain impacts by Ira R. Casson and David C. Viano [63], it was reported that the neuropathological condition of American Football players exposed to different brain traumas during their career is mainly behavioral, cognitive, or based on alterations of the mood.

Chapter 4. Experimental Study

4.1 Acquisition protocol

Fifteen American Football players regularly training and participating to the Italian Football League championship were enrolled in the study. The participants had an average age of 23.67 ± 3.42 (min: 18 years-old, max: 33 years-old), an average weight of 94.67 kg ± 14.69 kg (min: 70 kg, max: 130 kg) and an average height of 1,82 m ± 0.07 m (min: 1,70 m, max: 1,97 m). Each athlete signed a consensus for the treatment of their data in compliance with the GDPR. All the acquisition started between the hours 19.20 and 20.20 and ended between 22.00 and 22.30 depending on the length of the training session, which usually lasted 2 hours.

The players wore a Zephyr Bioharness sensor during a normal training session. The acquisition protocol consisted of the following phases:

- Pre-training phase lasting at least 5 minutes(min); during this phase a Kardia Alivecor 6L sensor was used two times to measure a 6-lead ECG for 30 seconds(s). The acquisition times were standardized to be after 2 min from the start of the Zephyr acquisition and after 3 min from the first Kardia acquisition.
- Training phase, which consisted in the typical phases of a training session that are elucidated in figure 35
- Post-training phase lasting 5 min; during this phase two acquisitions with Kardia Alivecor 6L were made just after the training session was concluded and 5 minutes after.

Following the instruction for the placement of the Zephyr sensor, the grey sensor pads of the strap were first moistened under water to increase the performance of the sensor itself. Then, the device was attached to the strap and eventually the strap was put on the subject with the device centered under the left armpit. At that point the subject was asked to sit down and relax as much as possible and the acquisition started by pressing the button on the device as shown in the figure 36. A stopwatch was used to record the main events. For the Kardia Alivecor 6Lacquisition, each subject was instructed to place their thumbs on the sensor pads of the Kardia 6L and to place the third sensor pads on their left ankle.

Figure 35. Scheme representing the phase of the acquisition protocol. The training protocol consisted of phases that repeated in a standardised way between the training session. It was always possible to identify mobility, linear speed, kicking game, individuals, 7v7 and pass pro rush, game simulations, field goal and coach's speech. Mobility was the time dedicated to warm up; Linear speed was a protocol on intermittent sprints with increasing distances being 10yards, 20yards, 30 yards and 40yards; Kicking game was dedicated to Special teams such as punt, punt return, kick-off and kick-off return; Individuals was the time dedicated to the development of role-specific skills; Pass Pro Rush was the time were defensive and offensive linemen simulated protection and tackling of the quarterback; 7v7 was the time dedicated to offensive skill players against defensive skill players. The field goal was a simulation of another kicking game that aims at kicking the ball between the bars of the goal post to score points

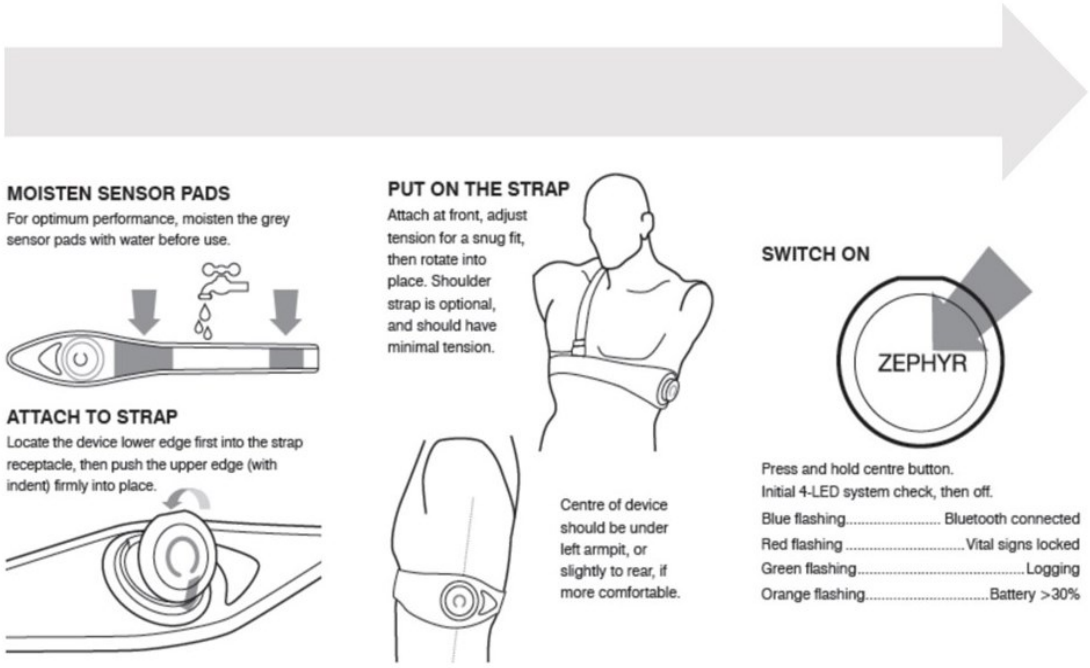

Figure 36. Process of application of the Zephyr sensor. Taken from the UserGuide of the Zephyr Bioharness 3.0

4.2 Data analysis

The process of analysis of the raw data coming from the acquisition consisted in three main phases being data pre-processing, feature extraction and statistical analysis and they were all performed on MATLAB 2021b ®. The aim of the analysis was to automatically compute the PLATEAU phase in the tachogram signal after each maximal sprint and then to apply a symbolic analysis to these phases to comprehend the dynamic of the sympatho-vagal balance during the training session of each subject.

The raw electrocardiogram coming from the Zephyr was first used to generate the tachogram exploiting the Pan-Tompkins's algorithm for the recognition of R peaks provided by the BioSig toolbox of MATLAB. The tachogram was cleaned of possible ectopic beats by applying a maximum and minimum threshold on the amplitude of RR intervals based on the values that were associated to a frequency of occurrence less than 0.001 in the histogram distribution of RR values.

4.2.1 Feature extraction and statistical analysis

The feature extraction procedure was applied to all the three phases of the acquisition protocol, and it was slightly different for the training phase and the phases outside the training session.

For the training phase it was necessary to recognize the maximal sprints and the consequent PLATEAU and RECOVERY phases. The algorithm uses as input signals the cleaned tachogram of the training phase, the Heart Rate signal (HR), and the Activity signal, computed as the magnitude of the acceleration vector. The outputs of the algorithm are the PLATEAU phase recognised after each sprint and the relative RECOVERY corresponding to the phase between the offset of the PLATEAU and the onset of the next sprint.

The algorithm consists of the following steps:

- A threshold of 1.0 was applied to the activity signal to recognise the phases of maximal activity during the training. The User Guide of the Zephyr Bioharness suggest that a 0.2 corresponds to walking and 0.8 to running, therefore the threshold was chosen arbitrarily in a range higher than 0.8 to ensure the recognition of maximal effort activity and reduce the number of possible sprints. The sprints lasting just 1s were excluded.
- The HR signal was first smoothed with a moving mean having a window of 15 samples and the local maxima happening after each sprint were stored to be used in the next step
- From the offset of each sprints the successive 100 beats were checked 10 beats at a time to find a PLATEAU phase based on two conditions. The first condition is that two adjacent RR values should have a variation of 15% of the local maximal HR, evaluated in the phase before, and each RR value should have a 15% variation of the local maximal HR when compared to

the first value of the PLATEAU. This first condition has the problem that identifies also phases having increased or decreased trends as PLATEAU, therefore a second condition to ensure the constant behaviour was imposed. The second condition is a threshold on the angular coefficient of the linear polynomial fitting the values of the analysed cardiac beats. Thus, to ensure a general constant behaviour the angular coefficient was set to be lower than 1. If both the conditions were true the selected beats were labelled as PLATEAU and the procedure was repeated on the next sequence of 10 beats by using the first RR interval value of the previous sequence as the reference for the first condition; on the other hand, if one condition was false, the procedure was repeated starting 5 beats ahead with another sequence of 10 beats.

- From the offset of the PLATEAU until the onset of the next sprint the RECOVERY after the sprints was evaluated if at least 100 beats were available. In this case, a RECOVERY phase was recognised by just applying the previous first condition and limiting the maximum length to 40 beats, because the symbolic analysis work best for short sequences. This last condition was applied to the pre-training phase (PRE) and post-training phases (POST) to find short sequences of the tachogram to analyse.

Eventually, for each PLATEAU phase a symbolic analysis was performed. It consisted in the following steps:

- The tachogram sequence was linearly detrended
- The range of values of the RR were divided by 6 intervals from the minimum to the maximum labelled from 0 to 5. Each RR value was identified as belonging to a class or symbol going from 0 to 5 if it was inside the specific interval
- The sequence of symbols was used to generate a set of adjacent symbols of length three
- These short clusters of three symbols was classified in four possible behaviours being constant or no variation(0V), when all the symbols were equal (ex. 1,1,1 or 2,2,2), one-like variation (1V), when two consequent symbols were equal and the third one was different (ex. 1,1,2 or 1,3,3), two-like variation (2LV) when all the symbols were different but with either an increasing trend or decreasing trend (ex. 1,2,3 or 5,2,1) and two-unlike variation (2UV) for clusters with peaks or valleys (ex. 1,2,1 or 3,1,5)
- The frequency of occurrence was reported for each symbol in percentage

For each subject the median and interquartile range of all the PRE, PLATEAU, RECOVERY and POST phases were performed.

4.3 Results of the experimental study

The results of the statistical analysis are reported in Tables 2,3,4,5.

Table 2. Median and interquartile ranges for the symbols in the PRE phase

subjectID	PRE_0V	PRE_1V	PRE_2LV	PRE_2UVL
1	0.11(0.07)	0.37(0.21)	0.16(0.12)	0.29(0.22)
2	0.13(0.18)	0.37(0.07)	0.21(0.08)	0.24(0.28)
3	0.09(0.13)	0.43(0.08)	0.11(0.07)	0.33(0.15)
4	0.16(0.08)	0.44(0.18)	0.13(0.05)	0.28(0.17)
5	0.1(0.09)	0.49(0.24)	0.15(0.08)	0.3(0.22)
6	0.13(0.11)	0.26(0.36)	0.13(0.11)	0.39(0.29)
7	0.11(0.04)	0.42(0.16)	0.13(0.07)	0.33(0.09)
8	0.11(0.1)	0.45(0.1)	0.17(0.07)	0.27(0.12)
9	0.14(0.08)	0.51(0.13)	0.12(0.03)	0.24(0.27)
10	0.17(0.22)	0.42(0.16)	0.17(0.1)	0.14(0.1)
11	0.15(0.13)	0.38(0.12)	0.17(0.04)	0.23(0.13)
12	0.04(0.1)	0.43(0.17)	0.22(0.09)	0.25(0.13)
13	0.29(0.04)	0.46(0.17)	0.03(0.07)	0.21(0.14)
14	0.14(0.06)	0.49(0.08)	0.19(0.18)	0.18(0.12)
15	0.04(0.15)	0.34(0.26)	0.12(0.1)	0.41(0.11)

Table 3. Median and interquartile ranges for the symbols in the PLATEAU sequences

subjectID	PLATEAU_0V	PLATEAU_1V	PLATEAU_2LV	PLATEAU_2UVL
1	0.05(0.1)	0.39(0.13)	0.14(0.15)	0.4(0.19)
2	0.07(0.14)	0.4(0.18)	0.1(0.08)	0.43(0.2)
3	0.1(0.13)	0.4(0.17)	0.1(0.11)	0.4(0.16)
4	0.1(0.15)	0.43(0.13)	0.08(0.08)	0.35(0.13)
5	0.08(0.15)	0.4(0.17)	0.11(0.18)	0.4(0.2)
6	0.08(0.17)	0.4(0.2)	0.1(0.11)	0.4(0.23)
7	0.1(0.13)	0.4(0.14)	0.1(0.08)	0.35(0.16)
8	0.1(0.2)	0.4(0.2)	0.1(0.1)	0.4(0.17)
9	0.1(0.16)	0.35(0.19)	0.1(0.1)	0.41(0.2)
10	0.13(0.1)	0.4(0.14)	0.08(0.05)	0.36(0.13)
11	0.1(0.12)	0.4(0.12)	0.1(0.09)	0.4(0.15)
12	0.14(0.15)	0.4(0.21)	0.1(0.1)	0.3(0.3)
13	0.08(0.21)	0.4(0.19)	0.05(0.13)	0.38(0.3)
14	0(0.1)	0.4(0.25)	0.1(0.1)	0.42(0.2)
15	0.17(0.15)	0.44(0.13)	0.03(0.06)	0.34(0.14)

Table 4. Median and interquartile range of the symbols of the RECOVERY phase lasting from the offset of a PLATEAU to the onset of the consequent sprint

subjectID	RECOVERY_0V	RECOVERY_1V	RECOVERY_2LV	RECEOVERY_2UVL
1	0.21(0.28)	0.42(0.18)	0.08(0.11)	0.2(0.28)
2	0.18(0.18)	0.42(0.13)	0.08(0.08)	0.26(0.19)
3	0.13(0.23)	0.39(0.16)	0.08(0.1)	0.32(0.24)
4	0.18(0.18)	0.42(0.16)	0.08(0.08)	0.26(0.24)
5	0.21(0.16)	0.45(0.14)	0.11(0.1)	0.18(0.17)
6	0.18(0.2)	0.45(0.15)	0.11(0.11)	0.24(0.18)
7	0.21(0.26)	0.42(0.17)	0.08(0.1)	0.21(0.23)
8	0.11(0.18)	0.39(0.16)	0.08(0.08)	0.34(0.17)
9	0.17(0.18)	0.42(0.16)	0.08(0.08)	0.29(0.18)
10	0.13(0.16)	0.4(0.12)	0.08(0.11)	0.34(0.16)
11	0.16(0.16)	0.39(0.15)	0.07(0.08)	0.32(0.21)
12	0.26(0.27)	0.45(0.18)	0.08(0.14)	0.15(0.15)
13	0.4(0.32)	0.36(0.2)	0.04(0.08)	0.14(0.17)
14	0.18(0.23)	0.45(0.15)	0.11(0.12)	0.21(0.16)
15	0.14(0.14)	0.45(0.12)	0.08(0.08)	0.3(0.19)

Table 5. Median and interquartile range of the symbols of the POST phase

subjectID	POST_0V	POST_1V	POST_2LV	POST_2UVL
1	0.23(0.15)	0.43(0.07)	0.16(0.12)	0.18(0.09)
2	0.05(0.19)	0.45(0.19)	0.16(0.15)	0.32(0.28)
3	0.22(0.16)	0.44(0.23)	0.11(0.08)	0.24(0.16)
4	0.26(0.15)	0.47(0.14)	0.11(0.09)	0.13(0.12)
5	0.11(0.11)	0.49(0.09)	0.17(0.08)	0.17(0.09)
6	0.13(0.18)	0.47(0.13)	0.13(0.1)	0.18(0.15)
7	0.21(0.14)	0.5(0.16)	0.2(0.13)	0.09(0.06)
8	0.26(0.15)	0.5(0.16)	0.07(0.07)	0.15(0.2)
9	0.24(0.21)	0.49(0.08)	0.08(0.04)	0.19(0.14)
10	0.3(0.18)	0.43(0.08)	0.08(0.05)	0.17(0.08)
11	0.14(0.11)	0.43(0.08)	0.12(0.1)	0.29(0.13)
12	0.18(0.07)	0.42(0.15)	0.21(0.14)	0.16(0.08)
13	NaN(NaN)	NaN(NaN)	NaN(NaN)	NaN(NaN)
14	0.13(0.18)	0.42(0.2)	0.25(0.09)	0.11(0.15)
15	0.19(0.27)	0.43(0.15)	0.16(0.15)	0.12(0.1)

Discussion and Conclusion

American Football is the sport with the greatest participation in the USA with athletes spanning from childhood to early adulthood. The considerable number of people surrounding the sport led to a greater interest in its issues.

In particular, the findings of the high concussion rates in football and the relation of concussive events with the impairment of cognitive skills have motivated a great part of the research for the understanding of the dynamics of head impact traumas. The studies in the biomechanics of concussion regard:

- laboratory reconstructions of video recorded head impacts to better understand the typical patterns of linear accelerations and rotational accelerations found during the collisions between two players [44] [45],
- the development of in vivo monitoring systems to track the frequency, locations, and dimensions of the impacts [46] [47] [48] [54],
- the validation of new metrics for the recognition of concussive events and to develop better protective gears for the players [44] [50]

The knowledge gap in the field of concussion monitoring regards primarily the understanding of concussion tolerance, defined as the amount of severity of head impacts in terms of head kinematics that an individual or groups of individuals can experience without showing symptoms related to concussion [43].

Another research line in biomechanics consists of the study of football-specific movements to improve the performance of players or to understand the dynamic of injuries during those specific actions. The analysis of the motion of the football pass [39], the long snap [40], the starting stances [41] and the foot in sport-specific tasks [42] are examples of other biomechanical applications.

The other great concern in the American Football population is the high prevalence of cardiovascular health problems. The most common cardiovascular risk factor is hypertension [20] [21] [22] [23], and the most typical pathological phenotype is concentric LV remodelling [21] [22] [23], which does not seem to be a form of the Athlete's heart. Linemen seem to be more subjected to cardiovascular issues due to their training focusing more on isometric exercises and their large weights. For this reason, preparticipation screening seems to be a viable option to recognise abnormal conditions and prevent complications such as SCD.

In vivo cardiac monitoring research focuses mainly on improving the performance of players and the coach decision making [34], tracking the recovery status of players between training sessions [35] or even after a concussion [36] or as an option to estimate heat stress to prevent heat illness [37].

This thesis expands the research field of cardiac monitoring in sport introducing a preliminary analysis of the dynamics sympatho-vagal balance during the training by applying a symbolic analysis to short sequences of the tachogram signal selected using an automatic procedure. The frequency of appearance of symbols computed by symbolic analysis was showed to be linearly correlated to sympathetic activity with the 0V class, and parasympathetic activity with the 2UV class [64]. Moreover, two recent studies demonstrated that the PLATEAU phase in the tachogram after a maximal sprint are intervals that are correlated to the sympatho-vagal balance in PRE and POST phases [65], and they are the result of the interaction between sympathetic and parasympathetic activity [66]. In the last study by Storniolo et al. [66] it was reported that the symbols were more sensitive to the various phases of the exercise protocol and that the PLATEAU phase corresponded to a phase of early vagal reactivation with the 2UV% higher in PLATEAU phase than in PRE phases.

It is possible to notice that in Table 3 the percentage of 2UV classes in all the subject from 1 to 14 have a median value that is higher with respect to the PRE sequences, which could be interpreted as an early vagal re-activation during this short PLATEAU sequences after a maximal sprint.

Additionally, it can be noticed in Table 5 that in POST sequences generally the 0V% values are higher than the ones in the PRE sequences of Table 2 (10 subjects of 15) and the 2UV% have generally lower values than the ones in the PRE sequences (11 subjects of 15); this result seems in contradiction with the one from the study of Storniolo et al. [66] , where in the POST phases the percentage of 2UV were statistically higher than the ones in the PRE phases. However, this could be explained by the vagal suppression that happens after an intense training in the hours following the training as showed in the study by Jamie Stanley, Jonathan M. Peake and Martin Buchheit [67].

The main issue of the proposed analysis is in the lack of validation for the automatic recognition algorithm which could provide false PLATEAU phases and therefore impair the symbolic analysis and its interpretation.

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