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Assessment of sport-related psychophysical stress during athletics training and competition

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ABSTRACT

Heart rate variability (HRV) is the time interval variation between two consecutive R-wave peaks (RR) in the electrocardiogram (ECG). Historically, HRV was used to assess the cardiac performing time and frequency domain analysis over a RR series longer than 125s. In case of athletes, during workout or competition, ECGs tracks are usually shorter than 125s when acquired with portable sensors. On athletes, the HRV analysis is employed to estimate athletes' psychophysical conditions. In case of short ECG tracks, the symbolic analysis has been proposed to assess cardiac autonomic control in literature. Thus, the aim of the thesis is to assess sport-related psychophysical stress during athletics training and competition, applying symbolic analysis on RR series. Data are a collection of 200 ECGs acquired on 10 young subjects practicing athletics running, during two different workouts (strength and speed) and competition. In these three conditions, six common phases can be distinguished: pre-warm-up, post warm-up, post exercise 5 minutes, 10 minutes, and 15 minutes of rest after exercise. Data were pre-processed and analysed in Matlab® environment. RR series were extracted from each ECG and the symbolic analysis was applied on each tachogram. The results of symbolic analysis were compared through nonparametric rank sum test. The main results show no significant differences between speed training and competition, suggesting that similar sporting gestures do not provide differences in sympathetic or parasympathetic activity. Moreover, V0 is major in speed training and competition than in strength training indicating a greater sympathetic control during an aerobic activity than anaerobic one. The values of RRmean parameters confirms a high sympathetic activity in competition and speed workout than in strength training. The UV2 incidence is higher in strength training than in other condition. Finally, about 93% of the matches reporting statistical differences, are located in the phases following physical effort and report a level of sympathetic activity higher in competition than in training. A psychological component, linked to the great importance that subject gives to the race, creates sensations antagonist to the subsequent "relaxation", which normally occurs after physical activity leading to an abnormal activity of sympathetic system. The study suggests that symbolic analysis is an excellent tool for evaluating the cardiac autonomic control, while subjects are practicing sports. Symbolic analysis of the tachogram provides information about psychophysical athlete state and could be used as a complementary tool to stress assessment tests. Symbolic analysis has widespread application in many sectors, as in the economic and financial fields and, in the last 20 years, its use in the medical research field has increased a lot.

CONTENTS

I. INTRODUCTION

Heart rate variability (HRV) is defined as the variation of beat-to-beat time interval between two consecutive cardiac beats. HRV has been usually employed to detect cardiac autonomic control in physiological and pathological conditions [36]. The analysis of HRV indeed permits to estimate the balance between sympathetic and vagal controls on the heart. It is usually evaluated by HRV analysis in frequency domain by assessing the power of low frequency (LF) and high frequency (HF) oscillations [17]. In order to have a reliable estimation of the features extracted in frequency domain, the guidelines require that this type of analysis can be performed over a RR series or tachogram longer than 125s [36]. For this reason, the frequency domain analysis cannot be applied on short electrocardiograms (ECGs) typical of acquisitions performed on athletes during sport. Same considerations can be done for HRV time domain analysis, to have reliable measures in time domain, guidelines require ECGs longer than 125s [36]. The analysis of HRV on athletes is essential to estimate their psychophysical conditions, trough assessment of cardiac autonomic control, and better analyze physiological parameters that could be misunderstood or hide pathological conditions. It is known that the condition of the *'athlete's heart'* characterized mainly by a greater cardiac hypertrophy, modifies a series of anatomical and physiological parameters of the heart and in general of the entire cardiovascular system [7]. Moreover, monitoring the condition of physical and psychological stress of the athlete is essential to improve his performance and reduce the risk of injuries. Given the short duration of electrographic acquisition dictated by portable sensors during sport activity, the symbolic analysis has been proposed to assess cardiac autonomic control in this setting [35]. This approach is based on the classification four beats RR patterns, namely three consecutive RRs, and the estimation of specific pattern incidence. This strategy allows the application of symbolic analysis over very short RR series *e.g. 30 s*. Differences in symbolic patterns incidence allow the evaluation of cardiac autonomic control in term of sympathetic and parasympathetic activity, that are indexes of psychophysical state of athletes. [35]. The appropriateness of this methodology over very short time series makes it suitable in exercise physiology and sport medicine. Thus, the aim of the thesis is to assess sport-related psychophysical stress during athletics training and competition exploiting symbolic analysis.

II. CARDIOVASCULAR SYSTEM

1. Anatomy of cardiovascular system

1.1. Organization

The circulatory system is composed by a set of pipes, blood and lymphatic vessels travelling all the human organism in which blood and lymph flow. The circulatory system is divided in cardiovascular system, where blood flows, and in lymphatic system employed in lymph transport. The cardiovascular system consists of the heart, the main pumping organ ensuring the blood circulation, and a closed system of vessels called arteries, veins, and capillaries. The cardiovascular system performs several functions as the oxygen and nutrients transport, the removal of the cellular catabolism product from their source, some homeostasis processes, and the intervention in immune processes. All the cardiovascular system functions are made possible by the main work of the heart: pumping blood in the body through the vessels. The heart, or cardiac muscle is a striated involuntary muscle, and it has a system of valves employed to address the blood flow in the right direction. Rhythmic heart contractions push the blood in the arteries. These, moving away from the heart, branch in a higher number of capillaries with reduced diameter. While arteries main function is to transport blood, the capillaries are permeable and allow the exchange of water, nutrients and oxygen between blood and tissues [1]. The capillaries form networks from which, by confluence, the venules originate and, coming together with each other, constitute veins of small, medium, and large calibre that return to the heart. Veins represent only transport vessels. In humans a double circulation exists, thus the heart is made of two pumps in series: the first one pushes venous blood throughout the lungs to ensure the exchange of O_2 and CO_2 in a process called *pulmonary circulation*, while the second one pushes the arterial blood in all the other tissues of the body by the process called *systemic circulation*. Heart is composed by four cavities: two in rear and upper side of thorax called right and left atrium and two in the front and lower side called right and left ventricle. Each atrium is connected to the underlying ventricle through the orifice provided of valves. Veins converge in the atria and arteries rise from ventricles. The systemic circulation originates from left ventricles by means of aorta which branching carries blood in all the body. Then, from venous system the blood returns in right atrium by means of the two venae cave: inferior and superior. From here the pulmonary circulation starts. Blood enters in right ventricle; it is pushed in two pulmonary arteries and enters in the lungs through a series of capillaries allowing the

exchange of carbon dioxide (out) and oxygen (in) with alveoli [1]. From lungs, blood comes back in left atrium by means of four pulmonary veins: right superior, inferior, left superior and inferior. The heart and the schematic representation of all the process is shown in Figure 1. In the system circulation the oxygenated blood flows in arteries and the deoxygenate blood in the veins, vice versa in the pulmonary circulation. Thus, the right part of heart contains venous blood, the left part instead the arterial one [1].

1.2. Position and Size of the Heart

The heart lies in the chest cavity, the mediastinum, wrapped in a fibro-serous sac called pericardium. Thus, at the bottom the heart rests on the diaphragm separating it from the abdominal viscera, forward it is protected by the sternum and costal cartilages (from the third to the sixth), behind it corresponds to the thoracic vertebrae (T5-T8) and at the top it extends towards the upper opening of the chest. The anatomical position of the heart in the thoracic cavity is shown in Figure 2. The heart has a flattened cone shape, it is directed obliquely downwards to the left and forward forming an angle of 45 degrees with respect to the longitudinal axis of the body. Compared to the longitudinal axis, the heart is slightly rotated clockwise [2]. The lower border of the heart, that lies on the diaphragm and points toward the left, forms a blunt point known as the apex, instead, the upper border of the heart, its base, lies below the second rib.

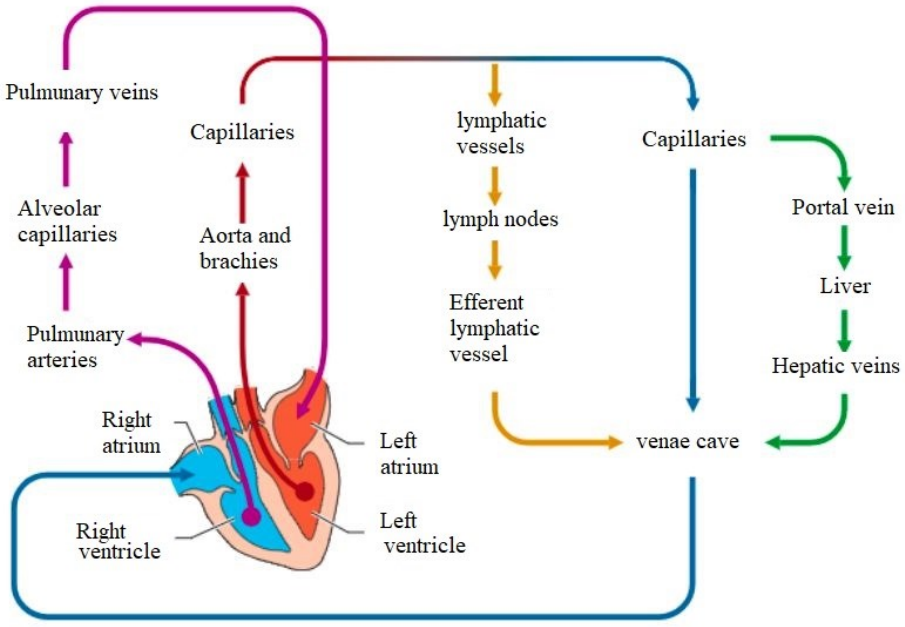

Figure 1: Schematic representation of both systemic and pulmonary circulation [1].

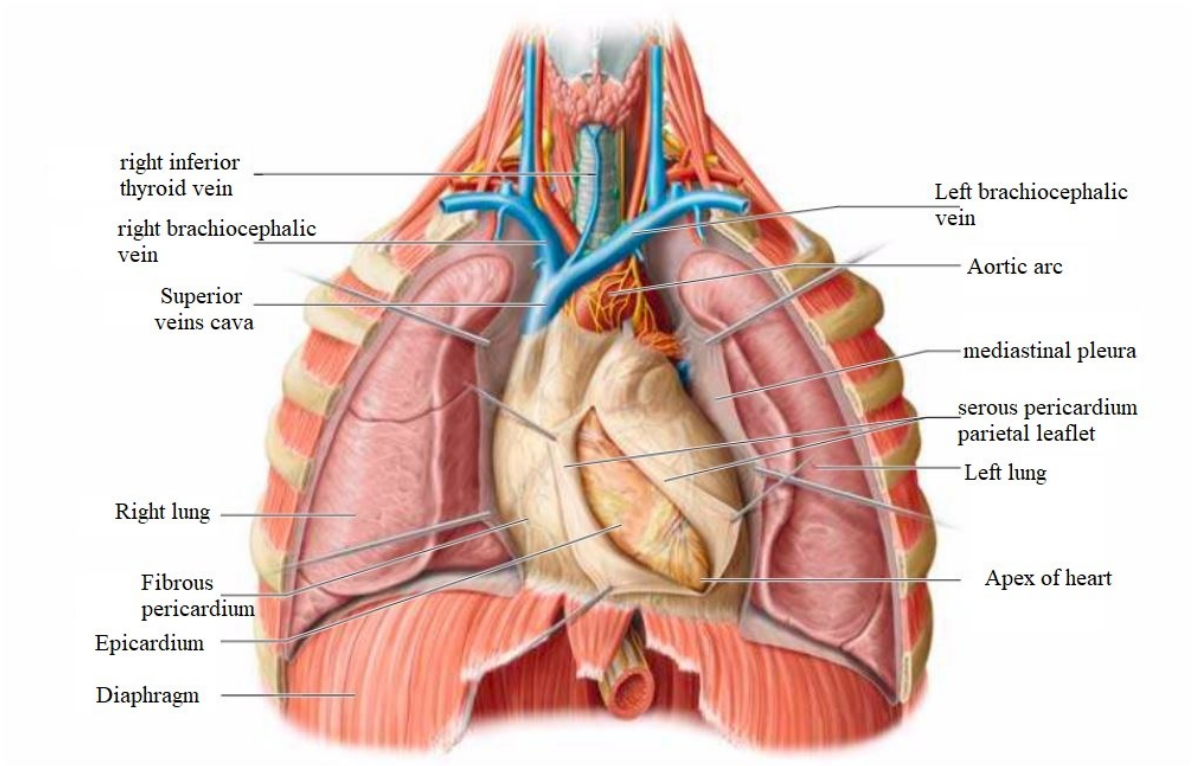

Figure 2: Anatomical heart position in the thoracic cavity [1].

border of the heart, its base, lies below the second rib. The boundaries indicate the heart size and have considerable clinical importance because a marked change in heart size can suggest certain types of heart disease. Approximately two thirds of the heart's mass is to the left of the midline of the body and one third to the right [3]. In the infant, heart is 1/130 of the total body weight, instead in adult about 1/300. Between puberty and 25 years, the heart consolidates its adult shape and weight: about 310 g is average for the male and 225 g for the female. In the adult, the shape of the heart tends to resemble that of the chest. In tall, thin individuals the heart is frequently described as elongated, whereas in short, stocky individuals it has greater width and is described as transverse. Heart approximate dimensions are length, 12 cm, width, 9 cm and depth, 6 cm [3]. Well-trained athletes can have much larger hearts due to the effects of exercise on the heart muscle, similar to the response of skeletal muscle.

1.3. External Heart Structure

The outer surface of the heart, covered by the visceral leaflet of the cardiac serous, has a sternocostal face, a diaphragmatic one, a base, an apex and two margins, right and left, called pulmonary faces. [2]. The outer structure is the *pericardium*. The pericardium, also called

pericardial sac, is a double-walled sac containing the heart and the roots of the great vessels. It has two layers: the *fibrous pericardium* that is the outer layer made of strong connective tissue, and an inner layer made of serous membrane called *serous pericardium*. Pericardium separates the heart from interference of other structures, protects it against infection and blunt trauma, and lubricates the heart's movements [4]. The outside layer is largely non-pliable even if it is capable of some change in shape. It acts to protect the heart against forces and pressure change from the outside. It is continuous with the outer layer of the neighbouring great blood vessels, fused with the diaphragm in its central area and attached to the posterior surface of the sternum by the steno-pericardial ligaments. The inside layer of the pericardium, serous pericardium is divided into two parts: the *parietal* serous pericardium, which lines the interior side of the superficial portion of the pericardial sac, it is fused to and inseparable from the fibrous pericardium; the *visceral* serous pericardium covers the myocardium of the heart and can be considered its serosa. It is largely made of a mesothelium overlying some elastin-rich loose connective tissue. The function of these layers is to lubricate the heart to prevent friction during heart activity [4]. For moving from the pericardium to the inner chambers, the wall of heart must be passed over. The wall of the heart is composed by three distinct layers of tissue: the epicardium, myocardium, and endocardium.

Epicardium lines the entire outer surface of the heart. It represents the visceral leaflet of the serous pericardium, and it is also reflected in the parietal leaflet. The two serous sheets delimit the pericardial cavity. On the surface, the epicardium is made of mesothelium, while deeply, the epicardium is connected to the myocardium by a connective layer called the 'subserosa canvas of the pericardium'.

The myocardium represents the thickest layer of the heart wall and is made of cardiac striated muscle tissue. It is divided into common myocardium and specific myocardium. The *common* myocardium represents the 90% of all the myocardium and constitutes its musculature ensuring its contractions. It is made up of mononuclear elements called *cardiomyocytes* connected to each other through intercalary discs. They have two components: *desmosomes*, which maintain adjacent cells together allowing the force created in one cell to be transferred to the adjacent cell, and *gap junctions*, which electrically connect cardiac muscle cells to one another. They allow waves of depolarization to spread rapidly from cell to cell, so that all the heart muscle cells contract almost simultaneously. Other structure of this tissue are *T-tubules* of myocardial cells, *myocardial sarcoplasmic reticulum* (MSR) and *mitochondria* occupy about one-third the

cell volume of a cardiac contractile fiber [2]. The *specific* myocardium constitutes the electrical conduction system as the elements that compose it are oriented towards a function of transmission of stimuli. So here there are fewer contractile cells but three cytotypes can be distinguished: *nodal, transition* and *Purkinje* cells. The firsts are also called pacemaker cells; they are concentrated in the sinoatrial (NSA) and atrioventricular (NAV) nodes and connected almost entirely by gap junctions that allow electrical signal transmission. The transition cells located especially on the periphery of the NSA and NAV, they have intermediate characteristics between specific and common cardiomyocytes. Finally, the Purkinje cells constitute the subendocardial networks in the walls of the ventricles; they are in contact with each other through gap junction thus being able to transmit electrical signal [2].

Thus, the myocardium has cells capable to autogenerate electrical signals and to stimulate contractile cells ensuring the functioning of the heart independently of central nervous system (CNS) control. However, CNS can play a role in the modulation of heart pace. [3].

The *endocardium* is a delicate layer of endothelial tissue lining of the interior of the myocardial wall. Endothelium is the type of membranous tissue that lines the heart and blood vessels. The endocardium covers beamlike projections of myocardial tissue called *trabeculae*: specialized folds or pockets formed by the endocardium which constitute the functional components of the major valves that regulate the flow of blood through the chambers of the heart [3]. Figure 3 shows both pericardium and the three layers of the heart wall.

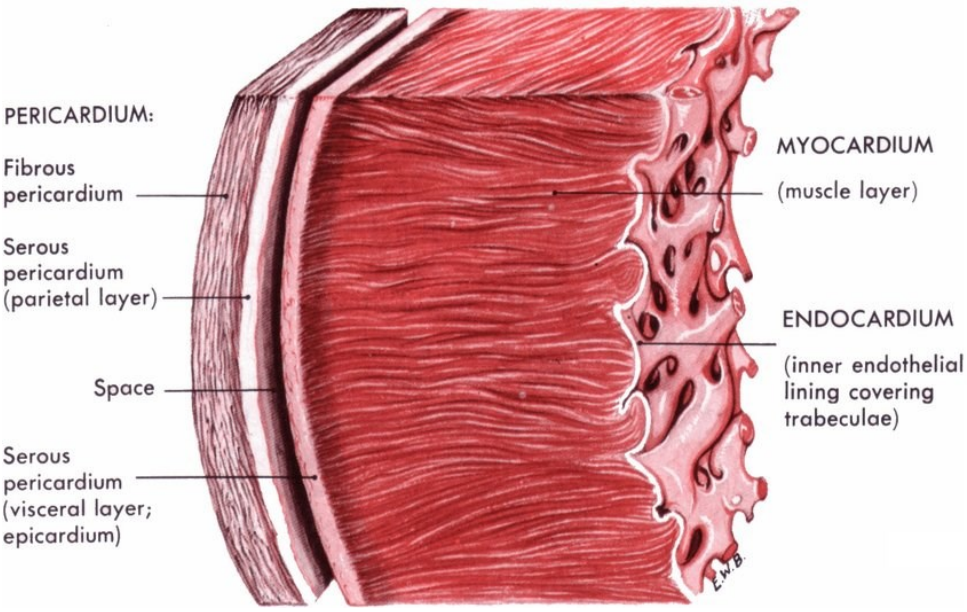

Figure 3: Pericardium and layers of heart wall from the outer to the inner side [1].

1.4. Internal Heart Structure

The interior side of the heart is divided into four cavities, or heart chambers. The two upper chambers are called *atria* and the two lower chambers are called *ventricles*. The left chambers are separated from the right chambers by an extension of the heart wall called the *septum*. In details, atria and ventricles are separated by interatrial and interventricular septa. The chambers have a similar structure in the inner surface although there are substantial differences between the right and left cavities [2].

The atria are often called the "receiving chambers" because they receive blood from veins. The internal surface appears smooth and regular centrally, while laterally it has a trabeculated appearance. Atria have a roughly globular shape, an average wall thickness of 1mm and, in the floor, they represent an orifice equipped with a valve which communicates with the underlying ventricle. The atria alternately relax and contract to receive blood, then push it into the lower chambers. Since the atria do not need to generate great pressure to move blood, because the distance with respect to ventricles is small, the myocardial wall of each atrium is not very thick [3]. In the lower part of the heart, ventricles are found. They have a conical shape, have an internal surface characterized by trabeculae. Among these, the first-order trabeculae are the *papillary muscles* that implant on the ventricular wall with one end while the other projects into the cavity. From their free end, very thin tendons originate, called the *tendon cords*, which insert into the atrioventricular valves. Papillary muscles and tendon cords constitute the *apparatus of tension* or valve anchoring preventing valve prolapse [2]. Ventricles receive blood from the atria and pump blood out of the heart into arteries, thus, the ventricles are considered the "pumping chambers" of the heart. The pumped-out blood must cover long distances: from heart to lungs (pulmonary circulation) or to all the body (systemic circulation) so more force is needed for the ejection with respect the atria one. Thus, the myocardium of each ventricle is thicker than the myocardium of atria. Moreover, the myocardium of the left ventricle is thicker than the right ventricle one, due to the different pathway covered by blood: the left ventricle pushes blood through most vessels of the body, whereas the right ventricle pushes blood only through the vessels that serve the gas exchange tissues of the lungs [1].

Figure 4 represents atria and ventricles. Both of them have orifices, arteriosus and atrioventricular provided with valves. The heart valves are mechanical devices that permit the flow of blood in one direction only. Four sets of valves are of importance for the functioning of the heart: the *atrioventricular* or *cuspid* valves and the *semilunar* valves.

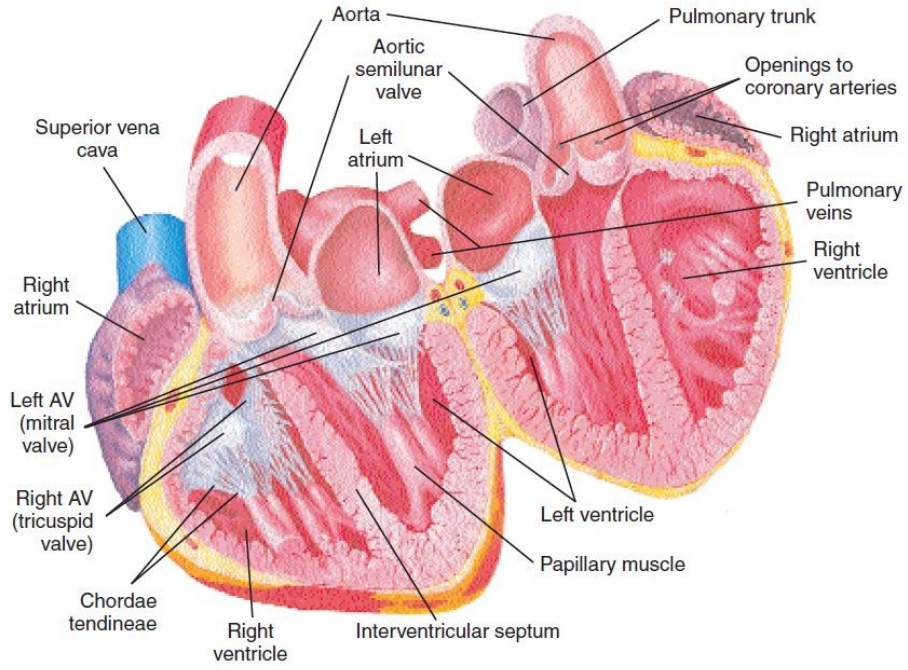

Figure 4: The main elements of the heart: atria, ventricles, valves, and major vessels [1].

The atrioventricular or cuspid valves guard the opening between atria and ventricles. The right atrioventricular valve is made by three flaps of endocardium. The free edge of each flap is linked to the papillary muscles of the right ventricle through some cordlike structures. Because the right atrioventricular valve has three flaps, it is also called tricuspid valve. The valve lying on the left atrioventricular orifice is similar to the right one, but it has only two flaps thus it is also called bicuspid or, mitral valve. The functioning of both atrioventricular valves allows blood to flow from the atria into the ventricles but prevents it from flowing back [2].

The semilunar or swallow's nest valves have a simpler organization than the previous ones. They consist of three membranous folds lined with endocardium with a shape that recalls that of a half-moon. The semilunar valve at the entrance of the pulmonary artery is called the *pulmonary* semilunar valve, instead that at the entrance of the aorta is called *aortic* semilunar valve. When these valves are closed, blood fills the spaces between the flaps and the vessel wall. Each flap is similar to a tiny, filled bucket. The inflowing blood smoothes the flaps against the vessel walls, collapsing the buckets and so opening the valves. The semilunar valves prevent blood from flowing backdown into the ventricles from the aorta and pulmonary artery [3]. Figure 5 shows both types of valves. The four valves of the heart lie on the same plane, oblique from left to right and from top to bottom called the valvular plane. On this plane is also

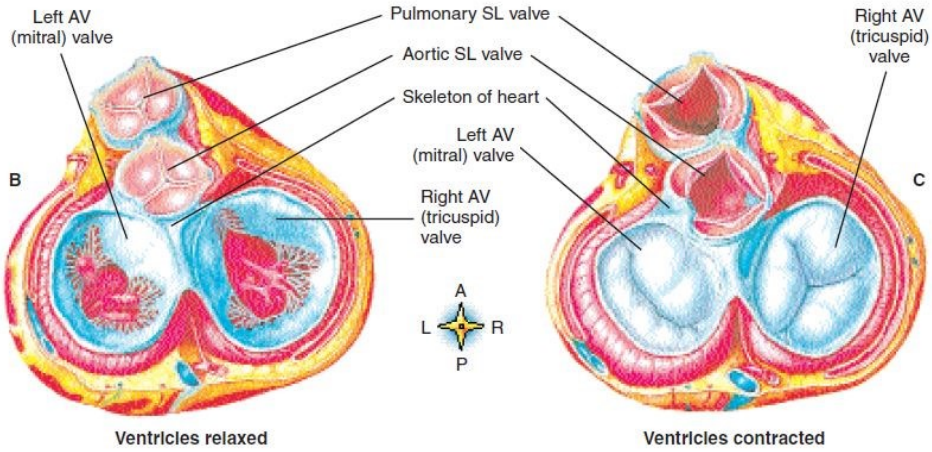

Figure 5: Cuspid and semilunar valves during ventricles relaxation (on the left) and ventricles contraction (on the right) [1].

contained the *skeleton of the heart*. It is a fibrous structure made by a set of connected rings that serve as a semirigid support for the heart valves (on the inside of the rings) and for the attachment of cardiac muscle of the myocardium (on the outside of the rings). The skeleton of the heart also acts as an electrical barrier between the myocardium of the atria and the myocardium of the ventricles [2].

1.5. Structure of Vessels

Blood vessels are defined as the blood ducts of the circulatory system used to transport blood through the body. The most important types are the arteries, veins, and capillaries that carry, respectively, blood from the heart to the rest of the body and vice versa. Blood vessels have the same basic structure changing with the variation of their calibre. They are divided into three layers called *tunics*:

- the *intima*, the innermost, is in contact with the basement membrane of the endothelium on which the blood flows.
- the *medium* in which smooth muscle fibro-cells arranged circularly are located.
- *adventitia*, composed of collagen and elastic fibers oriented according to the axis of the vessel.

Veins have a structure mostly similar to the arteries one, but the arteries have more robust and elastic walls than veins, because arteries have to withstand the strong blood pressure. Moreover, veins have valves called "swallow's nest", that prevent blood from flowing back due to the force of gravity. Such valves are not needed in the arteries, where reflux is hindered by the high

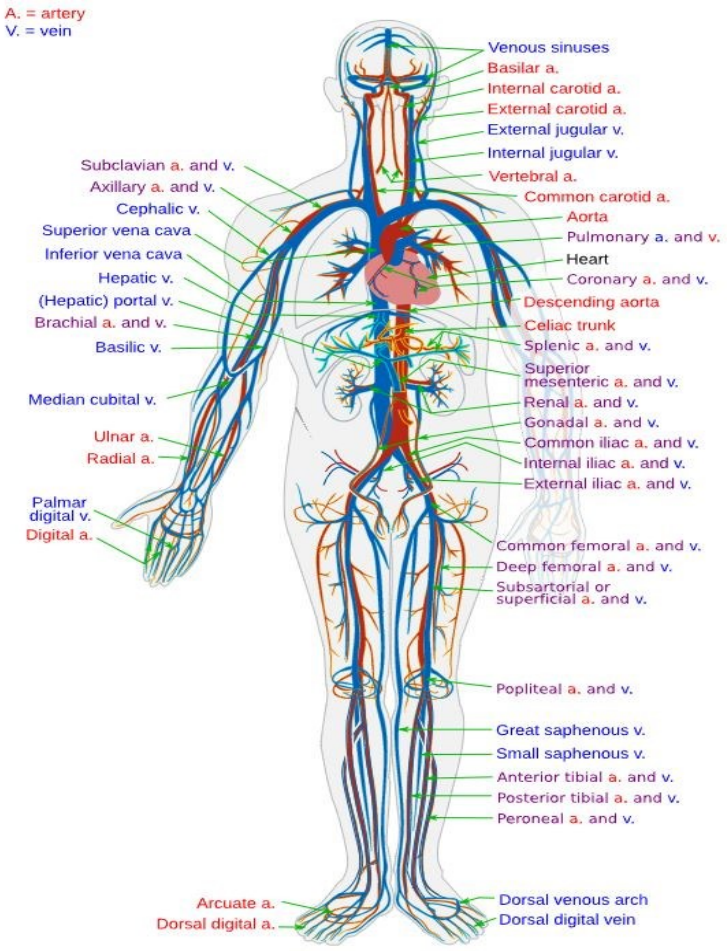

Figure 6: The main vessels of circulatory system: red for arteries and blue for veins [2].

pressure that the heart supplies to the blood. The main difference between vessel is that arteries move away from the heart while veins are vessels approaching the heart [2]. Capillaries consist of little layer of endothelium and sometimes connective tissue. When the blood vessels join to form a particularly vascularized region, there is an *anastomosis*. From one end to the other, blood vessels run through the human body for about 100,000 kilometres, 2.5 times the circumference of the Earth [5]. In Figure 6 the circulatory system is shown.

1.6. Structure of Coronary System

The heart is supplied by the right and left *coronary arteries* creating the coronary circulation to which about 5% of the cardiac output is destined. The coronary arteries and their main branches run on the external surface of the heart, covered by the epicardium, received in the interventricular sulci and in the coronary sulcus. The thinnest branches of the coronary arteries enter the thickness of the myocardium and reach the subendocardial layer. Coronary arteries originate from the ascending aorta at the aortic sinuses just above the semilunar valves of the

aortic valve. The caliber of the coronary arteries measures on average 3-4 mm and in 80% of cases the left coronary artery has a diameter slightly greater than that of the right. By convention, the *dominant artery* is defined based on the origin of the interventricular or descending posterior artery. In 90% of cases there is a right dominance as this artery derives from the right coronary one, while in 10% of cases the dominance is left [2]. Between the coronaries, as well as between the branches of the same coronary artery, there are often anastomosis represented by vessels that are too small to establish a valid collateral circulation. An anastomosis is a connection, or opening, between two structures (especially cavities or passages) that are normally divergent or branched, for example between blood vessels, nerves, or streams [6]. The right coronary artery originating from the right aortic sinus directs downward and to the right into the anterior portion of the coronary sulcus. After having surrounded the acute edge of the heart, it continues its course in the diaphragmatic face in a sulcus called *crux cordis*. Here it creates a 'U' loop called ‘descending interventricular artery’ which continues to the apex of the heart. In the case of left dominance, the right coronary exhausts before reaching the crux cordis. During its course, this artery supplies numerous collateral branches. Among the most important there are: two atrial branches that are distributed in the wall of the right atrium, one of which goes up through the sinoatrial node; ventricular branches are short and destined for the wall of the right ventricle; atrioventricular branches are small and distributed in a limited portion of the diaphragmatic face of the atrium and right ventricle; finally, branch for the atrioventricular node originates in the crux cordis in case of right dominance [2].

The left coronary artery originates from the left aortic sinus, goes obliquely down to the left. On its way it does not supply significant branches. It is 1cm long because as soon as it reaches the coronary sulcus, it divides into an ‘anterior interventricular artery’ and ‘circumflex branch’. The *anterior interventricular artery* continues the course of the left coronary artery to the right edge of the heart which it passes through to supply the diaphragmatic surface of the apex. This artery also supplies branches of the sternocostal face of both ventricles and the interventricular septum. The *circumflex branch* leaves the left coronary artery almost perpendicularly. It runs in the coronary sulcus, reaches the diaphragmatic face of the heart, and terminates before reaching the crux cordis. In the case of left dominance, it reaches the crux cordis and supplies the posterior interventricular artery. Various collateral branches are emitted from the circumflex

branch which are divided into atrial branches and ventricular branches. In 45% of cases the atrial branch emits the branch for the sinoatrial node. [2].

The blood inside the coronary circulation is collected from the coronary sinus, the anterior cardiac veins, and the minimal veins. The *coronary sinus* is a large and short venous vessel located in the diaphragmatic face of the heart, received in the left part of the coronary sulcus together with the circumflex branch. It is approximately 3cm long and opens into the right atrium. Its orifice has a valve called the coronary sinus valve. The veins that flow into the coronary sinus follow backwards the course of the main arterial branches and they are: magna cardiac vein, left marginal vein, oblique vein of the left atrium, posterior vein of the left ventricle, middle cardiac vein and parva cardiac vein. The *anterior cardiac veins* collect blood from the sternocostal side of the right ventricle and flow directly into the right atrium. *Minimal cardiac veins* are venules that collect blood from small parts of the myocardium and open without a precise order into the nearest cardiac cavity through small orifices [6]. All the coronary vessels described above are shown in Figure 7.

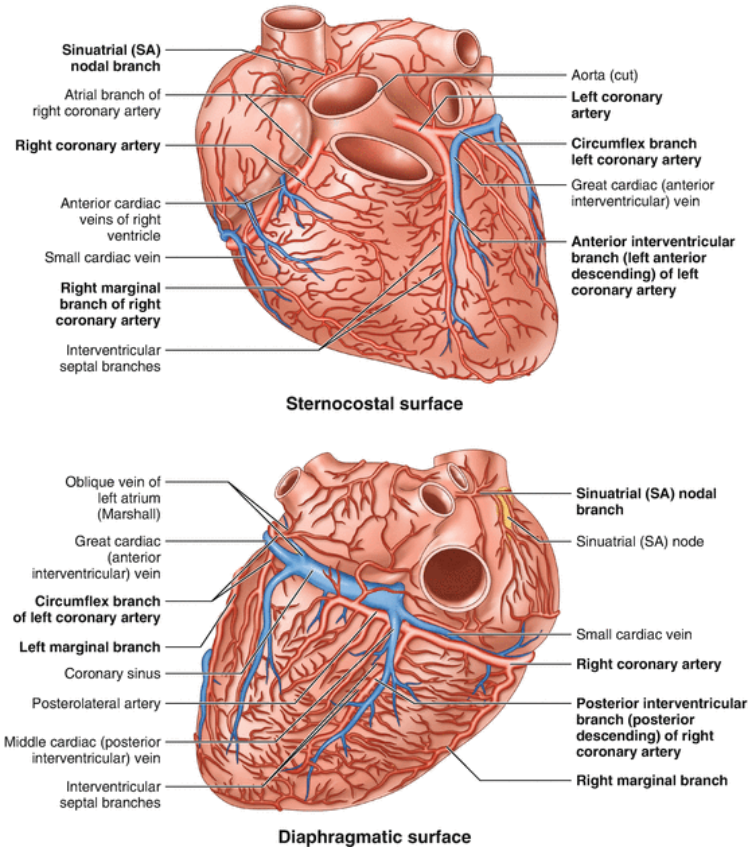

Figure 7: The main coronary vessels: sternocostal view (above), diaphragmatic surface (below) [1].

2. Physiology of Cardiovascular System

Over an average life of eighty years, the heart beats about three billion times. Each beat releases about 75ml of blood, thus in eighty years the heart moves about 225 million liters of blood. This activity is performed with great precision and regularity, adapting the rhythmic and pressure function to the metabolic needs that continuously vary in the organism, ensuring homeopathic control [1]. The mechanisms underlying the regulation of the heart are extremely sophisticated both from a mechanical and an electrical point of view. This chapter deals with the principles underlying cardiac function, which combines mechanical and electrical ones, and its regulation and modulation.

2.1. Excitation-Contraction Coupling in the Cardiac Muscle

The cardiac muscle is, like skeletal muscles, a striated muscle. However, the *excitation-contraction coupling mechanism* is different from skeletal muscles one. For this coupling, a *dyad* (specialized structure composed of T-tubules and cisterns of the sarcoplasmic reticulum) is involved. On the dyad T-tubules membrane there are DHPR receptors (*DiHydroPyridine receptors*) which act as calcium channels. When the action potential (AP) reaches the dyad, it induces the opening of these channels allowing calcium ions (Ca^{2+}) to enter the dyad. These Ca^{2+} bind to the RYR receptors (*Ryanodine receptors*), opening the holes for the release of huge quantities of Ca^{2+} from the sarcoplasmic reticulum to the cytoplasm. Using Ca^{2+}, the muscle fibers will then begin to contract. (*Mechanical Activity of the Heart*) [1].

2.2. Electrical Activity of the Heart

Given the strong dependence of cardiac contraction on the electrical activity of the heart, the latter has been studied and recorded since the early nineteenth century. The electrical activity varies as different cardiac regions vary and this is noted by the shape, duration, and onset time of AP during a cardiac cycle (Figure 8). The observation of Figure 8 suggests important indications: cardiac AP originates in the NSA, at the top of the right atrium, known as the heart's natural pacemaker; the potential generated by the NSA propagates in the two atria until reaching the NAV, a region on the border between atria and ventricles; The NAV is the only region that allows the passage of the AP from the atria to the ventricles. Indeed, after an evident delay, the AP propagates along the *bundle of His* which in turn creates two branches. They flank the walls

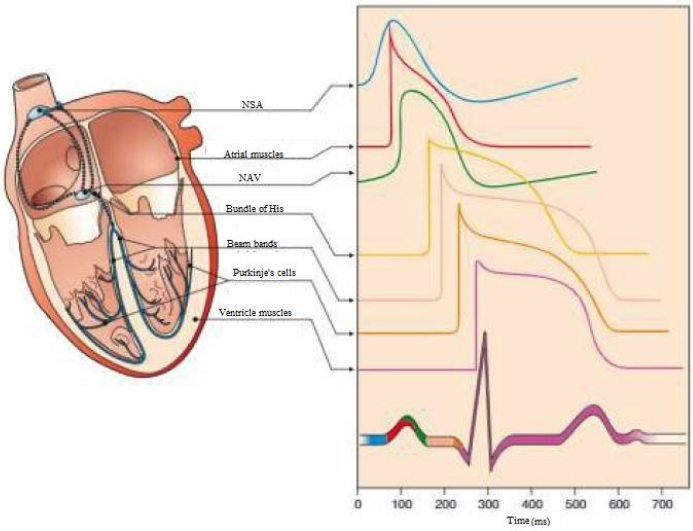

Figure 8: The electrical activity of different parts of the heart: shape and duration of action potential [2].

of the interventricular septum to the right and left, towards the apex of the heart. From here, the AP goes up along the ventricular walls through specific pathways such as the Purkinje fibers but also using the gap junction between the cells as the heart tissue is a functional syncytium [1]. As can be seen in Figure 8, the *working myocardium* (atria and ventricles) is characterized by the presence of a resting potential, that is, a membrane potential constant around -80 / -90mV. This means that the working myocardium is inert and needs a trigger to activate. This trigger is provided by the NSA and NAV cardiocytes which have no resting potential, but a phase called *diastolic depolarization*. The function of this phase is to bring the membrane potential from the most negative value (reached at the end of an AP) towards the threshold of a new AP. The NSA and NAV therefore do not need an external trigger, they function as cells with autonomous activity whose task is the generation of the stimulus to be propagated to the whole heart: pacemaker function. The AP is a rapid depolarization of the cell membrane that is established on a negative intracellular potential with respect to the outside of the cell. This change in membrane potential is associated with the presence of transmembrane ion currents and a capacitive current due to the redistribution of electric charges on the two faces of the membrane. For small and approximately spherical cells the distribution of electrical charges is uniform, while for cells with elongated portions (such as the axons of neurons) the phenomenon of distribution of charges is not uniform and is at the base of the AP propagation. At any moment, the AP will be in a precise position of the tissue and this part will be depolarized. Instead in the contiguous parts, the tissue is still normally polarized. The presence of regions with different distribution of electric charges generates an electric field and a positive charge immersed in an electric field is subject to a force

that can be described with a potential vector. The simplest electric field is that generated by a dipole: two opposite charges of the same intensity positioned in nearby points. A dipole vector, associated with the dipole field, describes the direction and intensity of the potential difference generated by the two charges. Through simplifications, the heart is represented as a space divided into two regions: one includes all the depolarized cells being the seat of the AP, the other contains all the cells polarized at the resting potential of the working myocardium. The boundary between the two regions can be considered as a wave front that advances over time during the propagation of the AP. Under these conditions the electrical state of the heart is described with a single *dipole vector* obtained as the vector sum of all the single dipoles associated with pairs of different charges [1].

2.3. Mechanical Activity of the heart

The heart is a muscular pump whose function must adapt to continuous variations in blood output and haemodynamic load imposed by physiological needs. Cardiac activity consists of relaxation and contraction phases that alternate continuously throughout life. Between the electrical excitation of the sarcolemma (AP) and the development of force by the sarcomere, there is a chain of events which, although similar, differs from that of the skeletal muscle. In the myocardium, a single action potential generates a single contraction (systole) of sufficient amplitude and duration to expel the volume corresponding to the systolic stroke [1]. Figure 9 shows the relationship between the heart's action potential and its major mechanical events constituting the cardiac cycle. It is the succession of mechanical events that characterize the heart's activity: each cycle includes a systole (contraction) and a diastole (relaxation). Mechanical events of the cardiac cycle occur simultaneously in the left and right heart; for simplicity, the left one is analyzed.

Atrial contraction: Cycle starts with the firing of NSA stimulating atria to depolarize them. Atria contraction causes the increase of pressure within the atria, forcing blood into ventricles through the opening of mitral valve. As atria contraction completes, atrial pressure begins to fall, reversing the pressure gradient to cross the mitral valve, causing it to close. Closing of mitral valve causes the first heart sound and marks the beginning of systole.

In ventricular depolarization, ventricles start to contract increasing their pressure. For a moment the aortic valve remains closed and the ventricles contract within a closed space: *isovolumetric contraction or systole*.

Rapid ejection: Ventricular ejection starts when ventricular pressure exceeds the pressure within the aorta: aortic valves open and blood is ejected out of the ventricles.

Reduced ejection: As ventricular repolarization begins, ventricular pressure starts to fall, and the force of ejection is reduced. When ventricular pressure drops below aortic pressure, the aortic valve close, marking the end of systole, the beginning of diastole and causing the second heart sound.

In the *isovolumetric relaxation* as ventricles relax with all valves closed, ventricular pressure drops rapidly, and volumes remain unchanged. Meanwhile, the atria are being filled with blood and atrial pressure rise slowly.

Ventricular filling starts when ventricular pressure drops below atrial pressure, causing mitral valve open, allowing blood to flow down the ventricles passively. The atria contract to finish the filling phase and the cycle repeats [2].

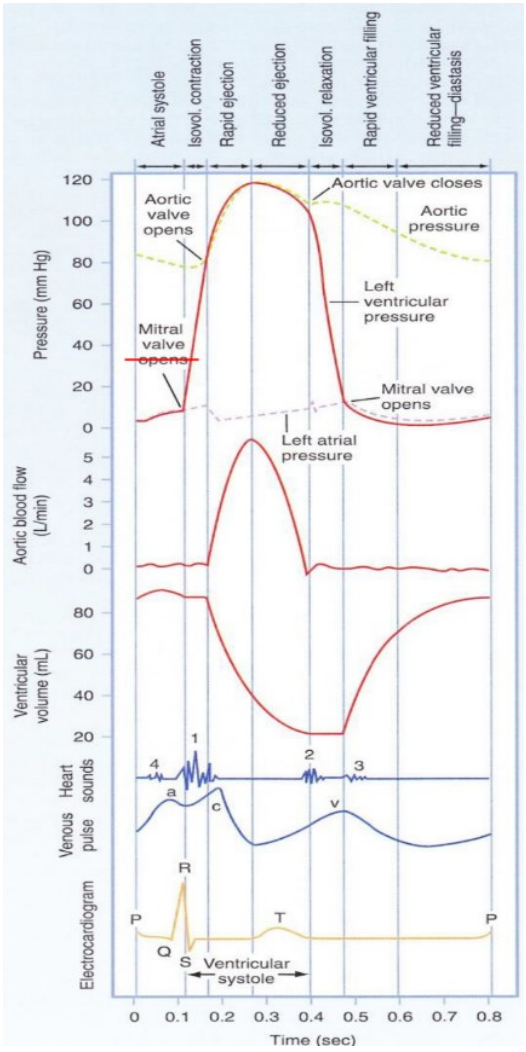

Figure 9: The relation between the electrical cardiac events and the mechanical ones constituting the cardiac cycle [2].

Figure 10 shows the cardiac cycle. The aortic pressure oscillates between a minimum value, diastolic pressure, of 80 mmHg and a maximum value, systolic pressure, of 120 mmHg. Duration of the cycle is about 0.8-0.9 seconds at rest (72bpm). The duration of the two phases is not equal: systolic phase lasts about 1/3 of the entire cycle (0.3s) and the diastole lasts about 0.6s (2/3 of cycle). Figure 11 shows another way to describe the cardiac cycle: the *PV-loop* (pressure-volume graph) which represents the relationship of pressure and volume in the left ventricle. The graph in Figure 11 allows to read several parameters of relevance. First, the *stroke volume* (SV), which is the amount of blood pumped by one ventricle during a contraction, measured in milliliters per beat, can be calculated as follow:

$$\mathrm{SV} = \mathrm{EDV} - \mathrm{ESV} \tag{1}$$

Where EDV is the end diastolic volume and ESV is the end systolic volume. At rest, SV is about 70 ml. Another important parameter is the *cardiac output* (CO), which measures the effectiveness of the heart pumping. It is calculated as:

$$\mathrm{CO} = \mathrm{SV} \cdot \mathrm{HR} \tag{2}$$

Where HR is the heart rate. CO has the dimension of a flow (ml/min) and informs how much blood is pumped to the systemic circulation (by LV to aorta) in a minute. For an average resting heart rate of 72 bpm and a stroke volume of 70 mL per beat, the cardiac output is 5040 ml/min. The *ejection fraction* (EF) is the ratio between the SV and EDV:

$$EF = \frac{SV}{EDV}(\%) \tag{3}$$

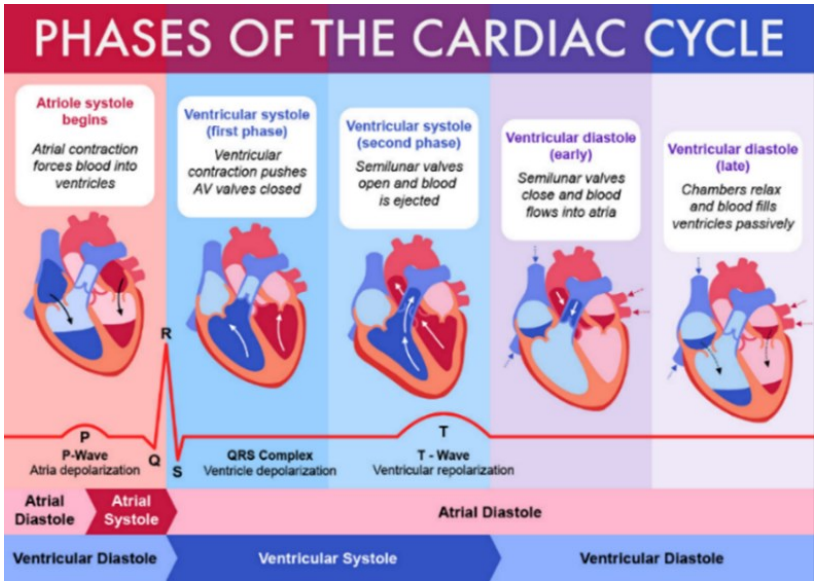

Figure 10: Phase of cardiac cycle [2].

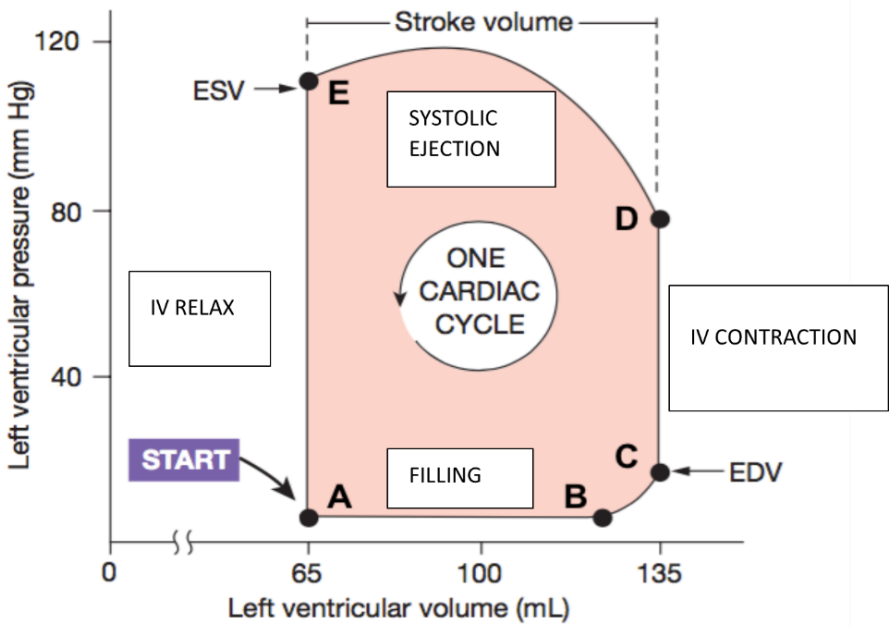

Figure 11: The PV-loop: relation between pressure and volume in the left ventricle [2].

2.4. Coronary Circulation

Most of the venous blood flowing from the coronary circulation (up to 90%) is entered into the right atrium via the coronary sinus. Of the remaining 10%, a part reaches the right atrium through the small coronary vein of Galileo and a part is drained by the cardiac veins of Tebesio which open both into the atrial and ventricular cavities. At rest, coronary blood flow is 60-80mg / min / 100g of tissue; the value increases during physical activity up to 200-300mg / min / 100g. Figure 12 shows the trend of coronary blood flow with respect to the aortic pressure: this flow increases during the diastole period. In the beginning of isovolumetric contraction, the coronary flow has a high value, while during this phase, the flow decreases due to the pressure exerted by the myocardium on the coronary vessels. During rapid ejection, the increase in aortic pressure leads to an increase in coronary flow while in the reduced ejection phase it decreases. During the isovolumetric relaxation phase, the coronary flow increases again reaching its peak due to the lower pressure exerted by the myocardium during diastole [1]. The compression effect exerted by the myocardial tissue on the coronary vessels does not have a uniform value at the different depths of the ventricular wall: it increases from the superficial or subepicardial layers to the deep or subendocardial ones. The coronary vessels are richly innervated by the sympathetic and parasympathetic systems and have different types of regulation system.

Reflex nerve regulation: sympathetic endings containing adrenaline have a constricting effect while parasympathetic endings containing acetylcholine have a dilator effect. Moreover, there is also a peptidergic innervation which plays a fundamental role in the modulation of coronary resistance.

Metabolic regulation: Among the various regulations, this is the weightiest. An increase in oxygen consumption leads to an increase in coronary blood flow to meet the metabolic needs of the tissue. Low oxygen concentrations induce the local release of vasodilating substances such as *adenosine*.

Hormonal regulation: there are several hormones capable of regulating coronary blood flow by dilating or narrowing blood vessels. Among these, *catecholamines* and *renin* are coronary-constrictors while *insulin* has a double effect: acting on the CNS it induces a sympathetic discharge and therefore vasoconstrictor, on the other hand, locally it has a vasodilating action. In addition, progesterone and testosterone are dilator hormones.

Autoregulation: an increase in perfusion pressure causes an increase in the degree of contraction of the smooth muscle of the coronary vessels, an increase in vascular resistance, while a reduction has the opposite effect. This mechanism tends to keep blood flow constant in the presence of changes in perfusion pressure. These mechanisms act simultaneously and can control coronary vascular resistance.

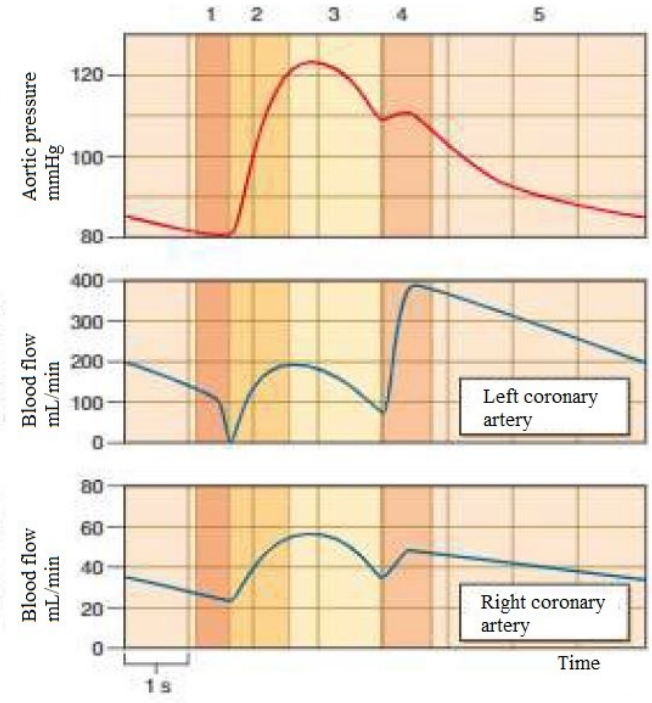

Figure 12: trend of coronary blood flow with respect to the aortic pressure in time [2].

2.5. Modulation of Heart Activity

As seen in the previous chapters, the heart is capable of self-generating electrical impulses to guarantee the physiological number of beats in a sort of steady state condition (70bpm). When the subject's conditions change, for example during physical activity or other, the heart must dynamically react by changing its normal activity to meet the physiological needs of the whole body at that moment. In this context, the modulation of the electrical activity of the heart is fundamental and is performed by various factors: the autonomic nervous system (ANS), cellular mechanisms for modulating the contraction force, and extrinsic modulation [1].

Modulation by the ANS. The heart is innervated by both adrenergic and cholinergic autonomic efferences. Particularly intense is the innervation of the NSA and NAV. The ANS controls the *chronotropism*, that is the frequency or heart rate, and the *inotropism* of the heart, that is the force of contraction influencing the ventricular pressure, the stroke volume and the cardiac output which also depends on the frequency. In detail, *the parasympathetic control* mediated by acetylcholine slows heart rate. Acetylcholine activates muscarinic cholinergic receptors that influence K^+ and Ca^{2+} channels in the autorhythmic cells. Potassium permeability increases, hyperpolarizing the cell so that the pacemaker potential begins at a more negative value. At the same time, Ca^{2+} permeability of the pacemaker decreases. Decreased Ca^{2+} permeability slows the rate at which the pacemaker potential depolarizes. The combination of the two effects causes the cell to take longer to reach threshold, delaying the onset of the action potential in the pacemaker and slowing the heart rate. *The sympathetic stimulation* of pacemaker cells speeds up heart rate. The catecholamines norepinephrine (from sympathetic neurons) and epinephrine (from the adrenal medulla) increase ion flow through both Na^+ and Ca^{2+} channels. More rapid cation entry speeds up the rate of the pacemaker depolarization, causing the cell to reach threshold faster and increasing the rate of action potential ring. When the pacemaker fires action potentials more rapidly, heart rate increases. *Tonic control* of heart rate is dominated by the parasympathetic branch. This control can be shown experimentally by blocking all autonomic input to the heart. When all sympathetic and parasympathetic input is blocked, the spontaneous depolarization rate of the NSA is 90-100 times per minute. To achieve a resting heart rate of 70 bpm, tonic parasympathetic activity must slow the intrinsic rate down from 90 bpm.

Cellular mechanism for modulating the contraction force. The maximum force developed by the heart muscle depends on the initial length of the fiber (sarcomere). The maximum force (P-pressure) is developed during the isovolumic contraction, this dependence is described by the

Frank-Starling law in a P-V graph (Figure 13). The latter is obtained by interpolating the pressure values reached at various preload volumes (V) at the end of the systole when the muscle stops crouching to reach its functional limit. The Frank-Starling law states that the pressure in the ventricle is directly proportional to the volume that the chamber acquires during preload. Furthermore, the slope of the relation P-V represents the maximum elastance (E). The contraction force can be controlled by modulating elastance in two ways: through changes in the calcium concentration that triggers the contraction or through the variation of the sensitivity of the contractile apparatus to calcium itself.

Extrinsic modulation. The sympathetic nervous system is responsible for the extrinsic modulation of the maximum elastance (inotropic effect), and the rate of relaxation (lusitropic effect) operated by the β-adrenergic receptors. By acting on the NSA, β-adrenergic stimulation increases heart rate and is essential in ensuring that diastolic relaxation is complete even at high rates [1]. The latter modulation is based on the concept of the sensibility of the heart contractive apparatus to the Ca^{2+}: there are several factors that move the relation between contraction and Ca^{2+} concentration.

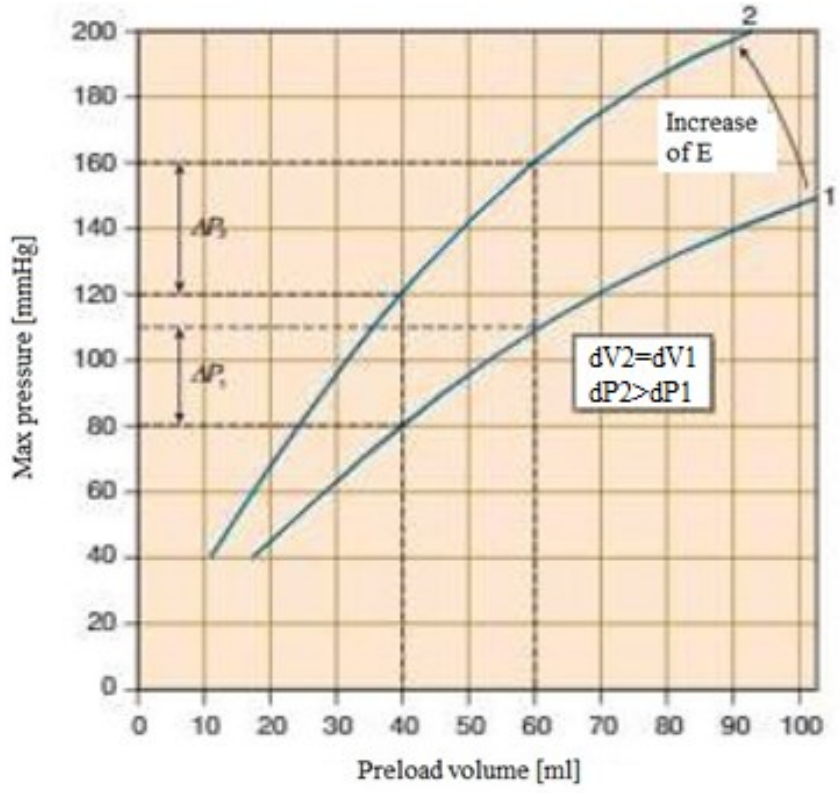

Figure 13: P-V graph showing the Frank-Starling law: developed pressure at different preload volume [2].

III. ATHLETE'S HEART

The development of the athlete's heart is a phenomenon produced by physiologic adaptations of the heart to the increased demands of exercise. The athlete's heart is associated with physiological remodelling as a consequence of repetitive cardiac loading. Athlete's heart is common in athletes who routinely exercise more than an hour a day, and occurs primarily in *endurance aerobic* athletes, then it can occasionally arise in *heavy weight* trainers. The condition is generally considered benign but may occasionally hide serious medical harmful conditions [7]. In the last century, physical activity has been shown to decrease the risk of cardiovascular disease. Nevertheless a small cardiovascular risk remains, as shown by several recent deaths of known athletes that have sensitized physicians to the importance of diagnosing cardiovascular abnormalities in athletes and thereby preventing these exercise complications. Increased physical activity requires increased oxygen delivery to the exercising muscle, which the heart supplies by increasing SV, HR and so CO. The pattern of cardiac changings differs between endurance and strength-trained athletes. The hearts of endurance athletes are characterized primarily by chamber dilation, whereas strength-trained athletes may show increased wall thickness. The degree to which these changes occur is based on the type of training pursued [8].

3. Physiological and Anatomical Changes in Athlete's Heart

The syndrome of athlete's heart (AHS) almost always does not have any pathological symptoms but leads to several changes both in the physiology and anatomy of the heart. Athletes with AHS often are not conscious of having this condition until they undergo specific medical tests. This is because athlete's heart is a normal, physiological adaptation of the body to the stresses of physical conditioning and aerobic, or sometimes anaerobic, exercise. People with AHS show three main changes as signs indicating the AHS that are different with respect to those of regular person: *bradycardia*, *cardiomegaly,* and *cardiac hypertrophy* [7]. Bradycardia is a slower heartbeat with respect to the normal one, ranges around 40–60bpm. Bradycardia is the most frequent change occurring in more than 50% of endurance athletes. Cardiomegaly is the state in which there is an enlarged heart, so an increase of heart chambers dimensions. The wall of the left ventricle increases in size by about 15–20% of its normal capacity. Cardiac hypertrophy concerns with the thickening of the myocardium, in particular the left ventricle. This is due to

a higher requirement of blood and oxygen to the peripherical tissues during an intensive workout. A larger heart results in higher CO, which also allows it to beat more slowly because more blood is pumped out with each beat. A less frequent symptom is the S_3 gallop that is the third heart sound. It is a rare extra heart sound occurring soon after the normal two heart sounds (S_1 and S_2) associated with the closing of heart valves. S_3 sound can be heard when the diastolic pressure, due to irregularly shaped heart, creates a disordered blood flow. However, if an S4 gallop is heard, patients need of immediate attention. An S4 gallop is a stronger and louder sound created by the heart, it is diseased in any way, and it is a typical sign of a serious medical condition [9].

Nevertheless, the dimensions of an athlete's heart, relatively to the body size, rarely exceeds the upper limits of normal. In the majority of athletes and with only rare exception, changes in left ventricular wall thickness, chamber size, and function remain inside the limits of normal. Hence, only some athletes demonstrate cardiac dimensions in the "grey zone" between normal and pathologic cardiac physiology. For this reason, the diagnosis of AHS is an important challenge due to the phenotypic overlap between the cardiac adaptive remodeling and early pathological changes seen in cardiomyopathies [9]. Furthermore, the morphologic changes seen in the highly trained individual reverse with detraining. Table 1 reports the main differences in the heart features between people with AHS and those with cardiomyopathy conditions, moreover, Figure 14 shows the heart of an athlete.

Table 1: Differences in the heart features between people with AHS and those with cardiomyopathy conditions.

Feature	***Athletic heart syndrome***	***Cardiomyopathy***
Left ventricular hypertrophy	< 13 mm	>15 mm
Left ventricular end-diastolic diameter	< 60 mm	>70 mm
Diastolic function	Normal (E/A ratio >1)	Abnormal
Septal hypertrophy	Symmetric	Asymmetric
Family history	None	May be present
BP respose to exercise	Normal	Normal/reduced systolic BP response
Deconditioning	L. ventricular hypertrophy regression	No L. v. hypertrophy regression

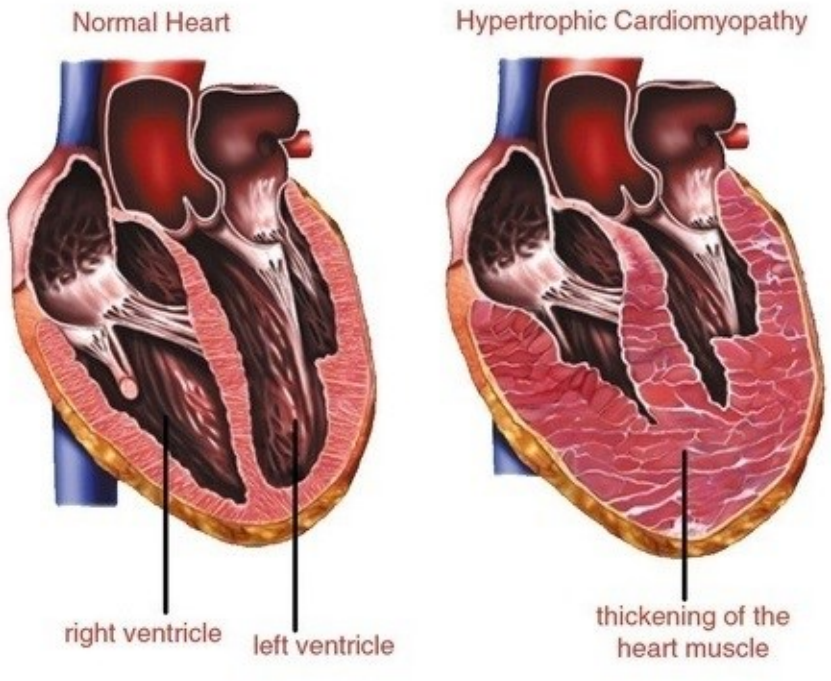

Figure 14: The main differences between a normal heart (on the left) and a hypertrophic one (on the right) [2].

4. Causes of Cardiac Complication During Exercise

Exercise-associated acute cardiac events generally occur in individuals with structural cardiac diseases. Among young individuals, variously defined as aged less than 30 or 40 years, the most frequent pathological findings are hereditary or congenital cardiovascular abnormalities: hypertrophic cardiomyopathy, coronary artery anomalies, aortic stenosis, aortic dissection and rupture, mitral valve prolapse, arrhythmogenic right ventricular cardiomyopathy, myocarditis, and others. In contrast to young subjects, coronary artery disease (CAD) is the most frequent pathological finding among older individuals who die during exertion. The mechanism by which vigorous exercise provokes such events is not defined but suggests that there are some triggering mechanisms as: increased wall stress due to increases in heart rate and blood pressure; exercise-induced coronary artery spasm in diseased artery segments and, finally, increased flexing of epicardial coronary arteries [10]. The annual estimated nontraumatic death rate, within 1 hour of sports participation among high school and college athletes is 1 per 133.000 men and 1 per 769.000 women per year. The first cause of exercise related cardiac death in young subjects is congenital cardiac abnormalities. The rate deaths in adults, is higher

than young one because of the higher prevalence of coronary CAD, and it ranges in 1 death per 15.000-18.000 middle aged men per year among individuals who showed themselves to be healthy [9].

A report from the *National Center for Catastrophic Sports Injury Research* examined the causes of nontraumatic sports deaths in high school and college athletes [11]. About 160 total deaths, in 10 years, were registered. Of these, 100 were attributable to cardiac causes. Hypertrophic cardiomyopathy comprised 56% of cardiac deaths followed by coronary artery anomalies (13%), myocarditis (7%), aortic stenosis (6%), and dilated cardiomyopathy (6%). Less common cardiac causes of death in young athletes included CAD (4%) and aortic rupture (2%). The cause of seven deaths is nowadays unknown and could be associated to abnormalities of the cardiac conduction system that cannot be detected by routine autopsy [11].

IV. TACHOGRAM & HEART RATE VARIABILITY

The tachogram is the series of RR intervals and its analysis allows the quantification of the heart rate variability (HRV). The HRV is the physiological phenomenon of variation in the cardiac frequency in response to several factors, in order to satisfy better the organism requirements [12]. Clinically, an optimal HRV value ensures flexibility and adaptability of the body's crucial regulatory systems. Too much instability, i.e. a high HRV, is detrimental for the efficiency of physiological function and energy usage, while a too low variability indicates pathology as it reflects a reduced regulation capacity. HRV is therefore used as an indicator of heart disease and to predict dysfunctions of the autonomic nervous system (including anxiety and depression) which, as mentioned in the previous chapters, is a regulation system of the heart [13]. The tachogram shows on the ordinate axis the temporal distance between two consecutive beats, that is the useful information, while on the abscissa axis the time or sometimes the number of beats in a certain period. The tachogram is therefore obtained from other signals including the *Electrocardiogram* (ECG) and the *Photopletismogram* (PPG). Figure 15 reports an example of tachogram. Before entering in detail in the different tachogram HRV analyses, it is necessary to describe the signals from which the tachogram is obtained.

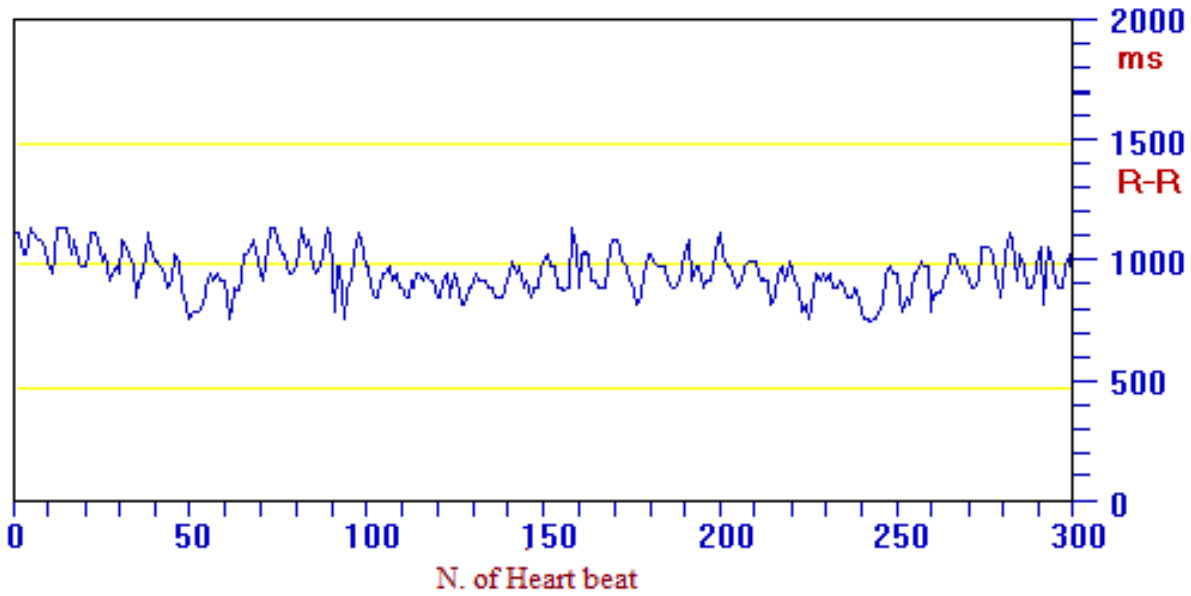

Figure 15: Example of Tachogram [2].

5. Electrocardiogram

In the previous chapter it was explained how the heart can be represented by an electric dipole and, its electric state by a dipole vector. The electric field generated by the cardiac dipole is transmitted to the surface of the body and the variations in potential generated can be recorded by electrodes placed on the skin. The result is a time-varying voltage signal that represents the changes in amplitude and direction of the cardiac dipole during the contraction cycle [1]. The electrocardiogram (ECG) is the graphic reproduction of the electrical activity of the heart recorded at the level of the body surface. Standard electrocardiography involves electrodes recording the dipole signal from 12 leads, of which 6 peripheral (3 bipolar and 3 unipolar) and 6 precordial (unipolar). Figure 16 shows a normal ECG:

- the sequential activity of the atria is visible on the ECG as a single *P wave* representing the depolarization and therefore the contraction of the atria (normally 60-120ms). An inverted P wave indicates a non-sinus origin of atrial activation. Atrioventricular conduction time is measured as a *PQ interval* (typically 120-200ms).
- The *QRS complex* represents the transmural activation and therefore the depolarization/contraction of the ventricles (normally 80-120ms).
- *ST segment* represents the period in which the ventricular cells are all depolarized and therefore no electrical movements are detectable, until the beginning of repolarization. It follows that normally the ST section (typically about 80ms) is isoelectric, that is, placed on the baseline of the path.
- *QT interval* represents electrical systole, that is, the time when ventricular depolarization and repolarization occurs. Its duration varies with the variation of the heart rate, generally it remains between 350 and 440ms. At the end of the repolarization there is a period of electrical inactivity, and the trace of the ECG remains isoelectric until the next pulse originates.
- The T wave represents the repolarization of ventricles [16].

Taken together, the P, Q, R, S and T waves make the so-called *PQRST complex*. Cardiologists call the existing interval between two PQRST complexes as "R-R interval." The R-R interval corresponds to a cardiac cycle, since the normal heart rate at rest is about 72bpm, one cardiac cycle lasts about 800ms. The decision to entrust the R waves of two consecutive PQRST

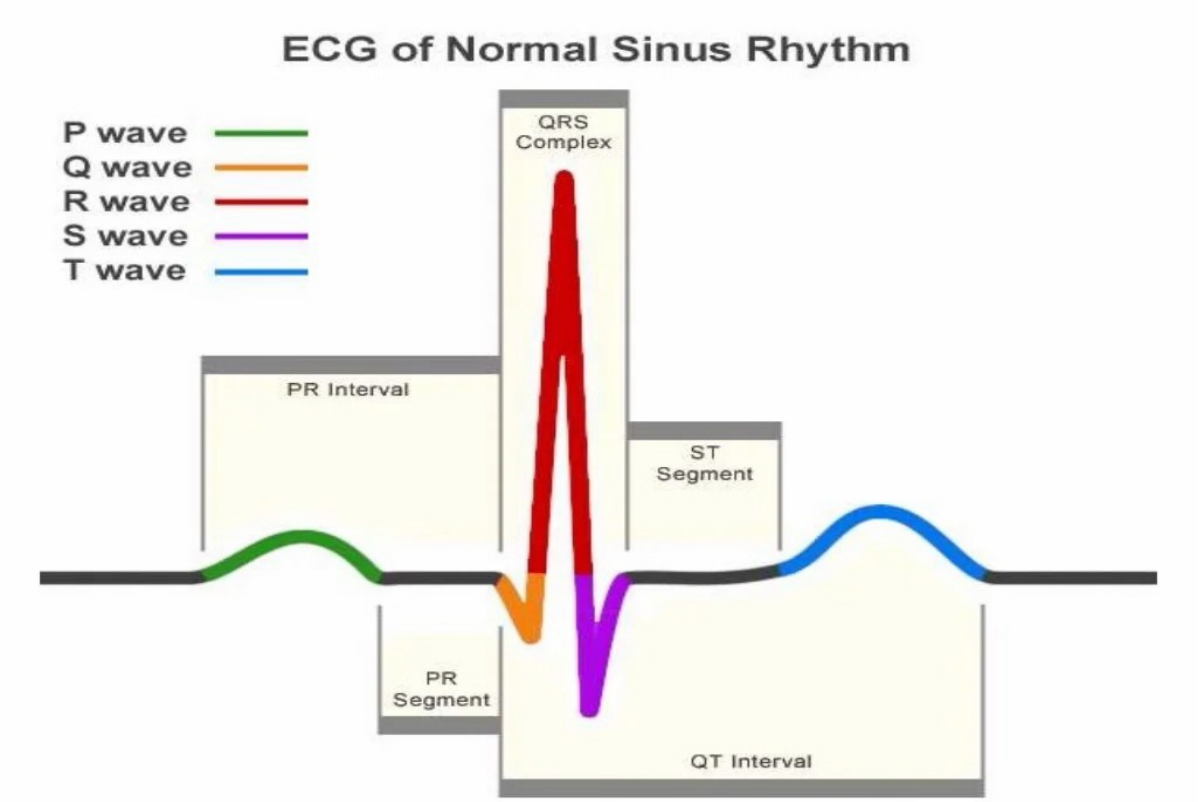

Figure 16: Example of ECG.

complexes with the task of identifying the beginning and end of a cardiac cycle is since the R wave is particularly evident [1].

6. Photoplethysmogram

Another way to detect heart rate and to monitor the cardiac cycle is the *photoplethysmogram* (PPG). PPG is a *plethysmogram* that can be used to detect blood volume changes in the microvascular bed of tissue. The plethysmogram is obtained using the *plethysmograph*, an instrument employed to measure changes in volume in an organ or whole body. The PPG is obtained using a *pulse oximeter* which illuminates the skin and measures changes in light absorption. A pulse oximeter monitors the perfusion of blood to the dermis and subcutaneous tissue of the skin. Heart pumps blood to the periphery for each ventricle contraction causing changes in vessels pressure. Even if pressure pulse is damped by the skin, it is enough to distend the arteries and arterioles in the subcutaneous tissue. If the pulse oximeter is attached without compressing the skin, a pressure pulse can also be seen from the venous plexus, as a small

secondary peak. The change in volume caused by the pressure pulse is detected by illuminating the skin, using a light-emitting diode (LED) and then, measuring the amount of light either transmitted or reflected to a photodiode. Additionally, the shape of the PPG waveform differs from subject to subject and varies with the location and way the pulse oximeter is attached. Since the skin is so richly perfused, detecting the pulsatile component of the cardiac cycle is relatively easy. The direct current (DC) component of the signal concerns with the tonic light absorption of the tissue, while the alternating current (AC) component relays to variation in blood volume in the skin caused by the pressure pulse of the cardiac cycle. The amplitude of AC component is proportional to the pulse pressure that is the difference between the systolic and diastolic pressure in the arteries [14] [15]. Figure 17 shows an example of PPG in which on the x-axis there is the time in seconds and on the y-axis usually mV is reported (changes in pressure lead to changes in light absorption and so to variations in mV output signal of photodiode).

7. Analysis Methods of Tachogram

The tachogram is obtained from the ECG or PPG traces, thanks to software algorithms able to recognize R peaks and of the QRS complex in the case of ECG and peaks in case of PPG signal. Then, the time distance between two consecutive peaks is performed and plotted as function of time or of number or beats in a certain period [13]. Figure 18 reports the extraction of R-R interval on ECG and the creation of tachogram. Moreover, on the ordinate axis, it is possible to put the instantaneous heart rate in beats per minute using the formula $bpm = 60/(R1 - R2)$, where R1 and R2 are the instants of two successive R peaks.

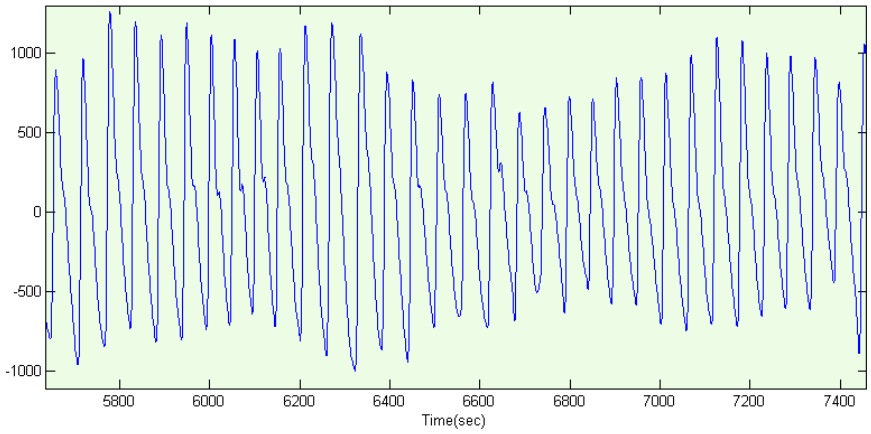

Figure 17: Example of PPG [2].

7.1. Time Domain Analysis

Variations in heart rate may be evaluated by several methods. The time domain measures are the simplest to be performed. Since they are always calculated in the same way, these data can be compared even if collected by different researchers, as well the time length of the ECG trace is the same. This is because, these parameters depend on the length of the recording (traditionally 24h). In a continuous ECG, each QRS complex is detected, and the so-called normal-to-normal (NN), intervals that is all intervals between adjacent QRS complexes, is calculated [18]. From here, simple time-domain variables can include the mean NN interval, the mean heart rate, the difference between the longest and shortest NN interval, the difference between night and day heart rate etc. Usually, the main indices calculated in the time domain are *SDNN*, *SDNN index* and *RMSSD*, while for evaluations on short-term recordings the *pNN50* and the HR_{max}-HR_{min} are also reported [17].

- *SDNN (Standard Deviation of N-N intervals)* parameter provides the standard deviation of the normal interbeat intervals measured in milliseconds and reflects the influence of all factors contributing to HRV (correlated with power in the frequency domain, below).

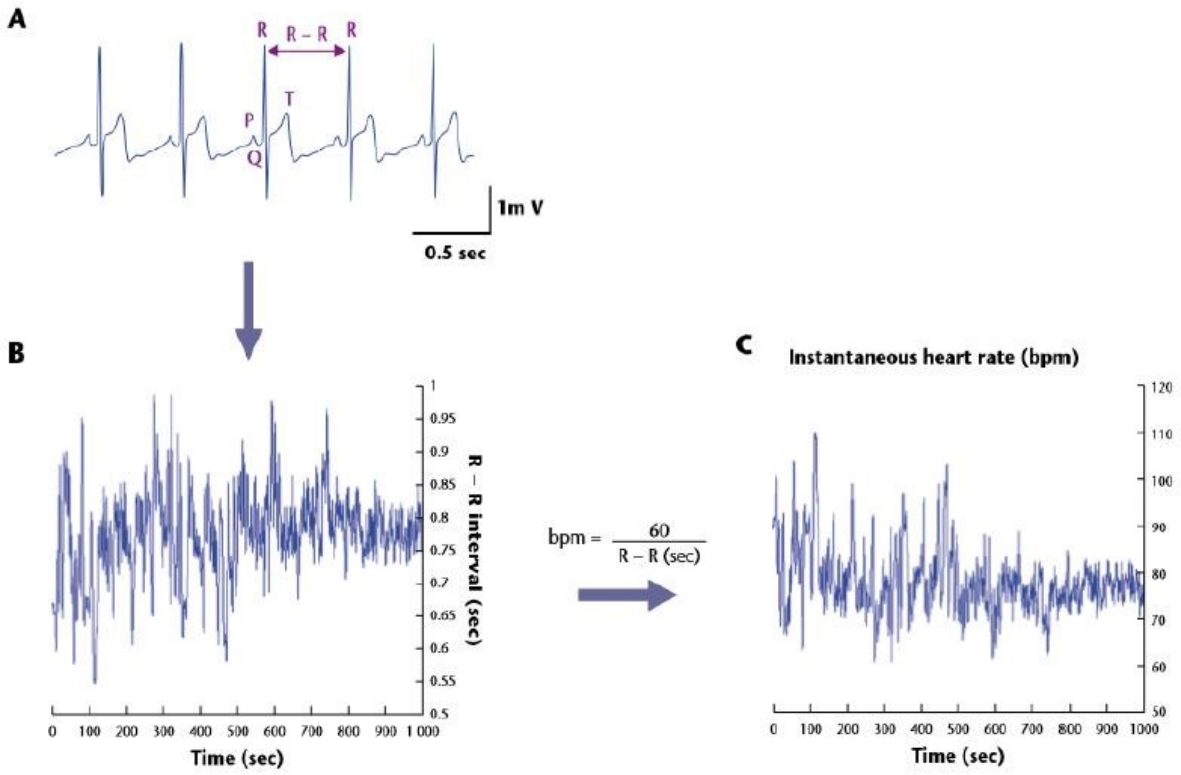

Figure 18: Extraction of R-R interval from the ECG (a), creation of tachogram (b) and usage of instantaneous heart rate (c) [17].

- *SDNN index* obtained in 24-hour acquisitions with the average of the SDNN calculated on the 5-minute intervals into which the total acquisition time is divided.
- *RMSSD (Root of the Mean of the Sum of the Squares of Differences between adjacent NN intervals*) parameter is the root of the square sum of the differences between successive NN beats. The long duration recording (24h) reflects the variability of the heart rate mediated by the parasympathetic nervous system, frequency domain (later) HF band.
- *pNN50 parameter* represents the percentage of adjacent normal beats that differ from each other by 50ms or more and is correlated with the RMSSD.
- HR_{max}-HR_{min}. This parameter is calculated with the average of the difference between the maximum heart rate and its minimum during each respiratory cycle (period in which we calculate the parameter) and is correlated with SDNN and RMSSD [17].

It is inappropriate to compare time-domain measures obtained from recordings of different durations.

7.2. Frequency Domain Analysis

Various spectral methods for the analysis of the tachogram have been employed since the 1960s. The *power spectral density (PSD*) analysis provides the power distribution as a function of frequency and allows to obtain information regarding frequency and amplitude of each specific rhythm present in the HRV waveform [18]. Methods for the calculation of PSD are generally classified as *non-parametric* and *parametric.* The advantages of the non-parametric methods are the simplicity of the algorithm employed (Fast Fourier Transform — FFT) and the high processing speeds. While the advantages of parametric methods are spectral components are smother and can be distinguished without preselecting frequency bands; the spectrum post-processing is easy, low, and high frequency power components are simply calculated, and also the identification of the central frequency of each component is facilitated; PSD estimation is accurate even on a small number of samples. On these samples, the signal is supposed to be stationarity.

The basic disadvantage of parametric methods is the need to verify the suitability of the chosen model and its complexity [17]. The power spectrum is divided into 4 frequency bands: *HF (high-frequency), LF (low-frequency), VLF (very-low-frequency)* and *ULF (ultra-low-frequency*). Figure 19 shows a typical subdivision of the HRV signal into the VLF, LF and HF

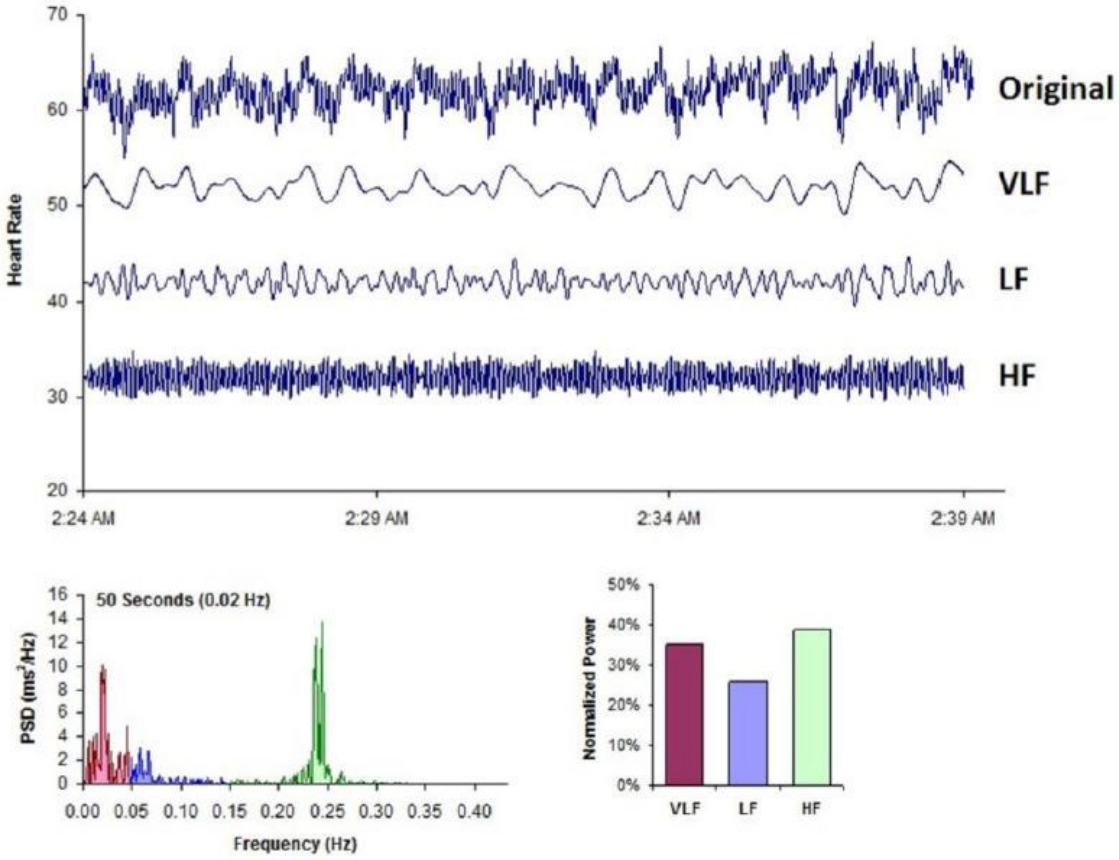

Figure 19: A typical subdivision of the HRV signal into the VLF, LF and HF bands, the power spectral density and the normalized power in each of the three bands [17].

bands, the power spectral density and the normalized power in each of the three bands. In detail, the *HF band* ranges within 0.15-0.4 Hz and reflects the activity of the parasympathetic nervous system. It is also called the respiratory band because it reflects changes in heart rate in relation to the respiratory cycle. During inspiration, the cardiovascular control center inhibits the activity of the parasympathetic system, thus increasing the heart rate. Instead, during the exhalation, the cardiovascular center reports the activity of parasympathetic system at the same level which had at the beginning of inspiration, decreasing the heart rate. A reduction in parasympathetic activity was found in patients suffering from heart disease and in patients under stress or suffering from panic attacks, anxiety or worries. A total block of parasympathetic activity eliminates high frequency oscillations and reduces the power of the LF band [17]. The *LF band* corresponds to the 0.04-0.15 Hz interval. This region is called the "baroreceptor interval". The latter are mechanoreceptors, sensitive to pressure variations, located inside the atria, the ventricles, the carotid sinuses and in the aortic arch. As blood pressure increases, the tissues of the carotid and aorta stretch, causing an increase in surface

tension and in the activity of the baroreceptors. Normally the baroreceptors are tonically active, but in case of an increase in pressure in the areas mentioned above, they generate action potentials with a greater frequency. This frequency will increase with increasing pressure. The activation of the baroreceptors determines the inhibition of the sympathetic nervous system and the activation of the parasympathetic nervous system. The combined effect of the two phenomena leads to a sudden decrease in blood pressure: the inhibition of the sympathetic system decreases the peripheral vascular resistance, the heart rate, and the contractility; at the same time the activation of the parasympathetic leads to the same effects. In turn, a decrease in blood pressure determines the opposite effect and so the activation of the sympathetic system, and the inhibition of the parasympathetic. For this reason, an increase in peripheral vascular resistance, heart rate and myocardial contractility is shown, and the blood pressure increases again. Contrary to HF band, the LF band shown both the activity of the sympathetic and parasympathetic system mediated by baroreceptors. The sympathetic activity generates oscillations in heart rhythms which fall into the LF band, and which correspond to physical activity or psychological stress. During slow breathing below 8.5 breaths per minute, or during sighs and deep breaths, parasympathetic activity generates oscillations that fall into the LF band [18]. The *LF / HF ratio* means the LF band power (the area under the PSD curve in the interval 0.04-0.15 Hz) divided by the HF band power (the area under the PSD curve between 0.15-0.4 Hz). This ratio is considered an *index* of the *sympatho-vagal balance*. A low value of the LF/HF ratio represents a prevalence of parasympathetic activity w.r.t. sympathetic one, due to an imbalance between the two branches of the autonomic nervous system in favor of parasympathetic activity at rest. On the contrary, a high value of this ratio can be seen as a prevalence of sympathetic activity over parasympathetic activity, which can be observed in subjects involved in demanding tasks as athletes during training or competition when the sympathetic activity increases. This relationship is much more complex since both sympathetic and parasympathetic activity is present in the LF band. Therefore, this ratio cannot be seen, in an absolute way, as a balance between the activities of the two branches of the autonomous system. The considerations based on this relationship must be made knowing the LF band generation mechanism and the context in which the acquisitions are made [18]. The *VLF band* is included in the frequency range 0.0033-0.04 Hz. Long-term regulatory mechanisms such as thermoregulation, the renin-angiotensin system, humoral factors can contribute to this band. Some evidence experiments suggest that the amplitude and frequency of oscillations in this

range are modulated by the sympathetic system. More specifically, VLF oscillations can be generated by the stimulation of afferent sensory neurons present in the heart, which activate feedback mechanisms at different levels of the heart nervous system, but also mechanisms present between the heart and the vertebral column. An increase in power in this band could reflect an increase in the activity of the sympathetic branch. The modulation of the oscillations in the VLF band by the sympathetic system, due to physical activity, stress or other factors that can increase sympathetic activity, causes them to cross over into the LF band [18]. The *ULF band* falls below 0.0033 Hz, thus with periods longer than 5 min. The fluctuations in heart rhythms within this band are mainly due to circadian fluctuations: physiological rhythm characterized by a period of 24 hours in which some metabolisms of the body are repeated equally even by varying the external conditions. However, there are also contributions due to very slow regulatory processes, such as body temperature regulation, metabolism, and the renin-angiotensin system. Given the very low frequency of the oscillations, the contribution of the ULF band can only be appreciated in 24-hour acquisitions [18]. All these evaluations begin from the computation of PSD but, independently of the method employed, only an estimate of the true PSD of the signals can be obtained by proper mathematical algorithms.

7.3. Non-Linear Methods Analysis

Non-linear methods are employed to quantify the structure and complexity of HRV. This signal is non-stationary and non-linear in nature. Analysis of HRV dynamics is based on observation suggesting that the mechanisms involved in cardiovascular regulation likely interact with each other in a non-linear manner. The most important indexes which describes non-linear HR dynamics are the *short-term fractal scaling exponent* measured by *detrended fluctuation analysis (DFA)*, the *approximate entropy (ApEn)*, the *Lyapunov exponent (LE)* [19] [20].

DFA is a fractal-related method. The fractal analysis consists of several methods aimed to assign a *fractal dimension* to a dataset that can be a theoretical dataset, or a signal extracted from some phenomena (HRV power spectrum in this case). The fractal dimension is a ratio providing a statistical index of complexity comparing how details in a pattern change with the scale used to measured it. DFA is a method employed to define the statistical *self-affinity* or *similarity* of a signal. A self-similar object is totally or partially similar to a part of itself: parts of it show the same statistical properties at many scales. It is useful for analyzing time series that appear to be long-memory processes. At the end of DFA the *scaling exponent* (α, the slope

of the power spectrum in this case) is calculated and it provides an estimation about the statistical self-affinity of the signal [21]. The *approximate entropy* is a technique used to quantify the amount of regularity and the unpredictability of fluctuations of a time-series data. The presence of repetitive patterns of fluctuation in a time series gets it more predictable than a time series in which these patterns are absent. The ApEn is calculated using an algorithm. A low value of ApEn means a time series containing many repetitive patterns, thus more probability to be predicted. Instead a high *ApEn* value concerns with a less predictable process dynamical system [22]. The *Lyapunov exponent* of a dynamical system is a quantity representing the rate of separation of infinitesimally close trajectories in the *phase space*. It is a space in which all possible states of a system are represented, and each state corresponds to one unique point in the phase space. Two trajectories in phase space with initial separation vector Z_0, diverge at a rate given by $Z(t) = e^{\lambda t} * |Zo|$, where λ is the Lyapunov exponent. For different orientations of Z_0, the rate of separation may change, thus, there is a spectrum of Lyapunov exponents, equal in number to the dimension of the phase space. The most common parameter used here is the *maximal Lyapunov exponent (MLE)* because it gives information about the predictability of a dynamical system. A positive *MLE* is usually an indication that the system is chaotic [23]. The single HRV time series is the output of a complex dynamical system that is the human body. The reconstruction of the system trajectory in the space state uses time delayed versions of the observed scalar quantities:

$$x(to + n\Delta t) = x(n) \tag{4}$$

where *to* is the initial time and Δt is the sampling interval (in HRV series Δt is = to 1, as HRV is a beat-to-beat event series). These new points become the coordinates to reconstruct the system dynamics in an embedding space. The multivariate vectors are obtained in a *m*-dimensional space, and defined as:

$$y(n) = \{x(n), x(n + \tau) \dots \dots x(n + (m - 1)\tau)\} \tag{5}$$

where $n = 1; N - (m - 1)\tau$, τ is an appropriate time lag and *m* is the dimension of the embedding space [20]. The *False Nearest Neighbors (FNN) method*, increasing the reconstruction dimension, allows estimating the true *m* value removing false intersection of the reconstructed trajectories [20]. The τ parameter can be calculated as the first local minimum of the *Average Mutual Information I(t)* [20].

7.4. Symbolic Data Analysis

The symbolic dynamics is a recent index of HRV, used to determine the prevalence of sympathetic or parasympathetic cardiac modulation. It consists in creating symbols, or better series of number, starting from a tachogram with at least 300 consecutive beats reported. Usually the full range of the sequences, so the y-axis of tachogram where duration of each R-R interval is reported, is divided in 6 levels (from 0 to 5) and patterns of length equal to three are created [24]. All possible patterns are divided in three categories:

- *0V* represents patterns with no variation, so *non-variable*. The three symbols are equal between them.
- *1V* pattern has one only variation so two symbols are equal, and one is different. It is the *variable* pattern.
- *2V* patters show 2 variations, so all symbols are different from the previous one. It represents the *very-variable* patterns.

Figure 20 shows the process of creation and classification of symbols in a R-R time series. After classifying each three-length pattern in one of the three types (0V, 1V, 2V), the percentage of incidence of each type is calculated. It is shown that high percentage of 0V incidence refers to a predominance of sympathetic system, instead high percentage of 2V incidence concerns with a parasympathetic cardiac autonomic modulation [24].

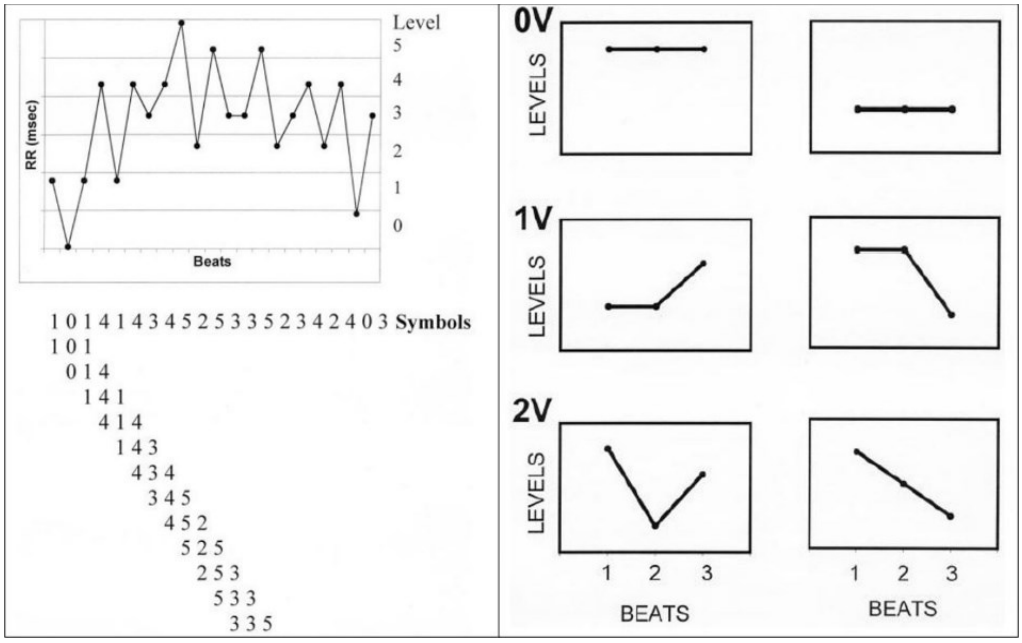

Figure 20: An example of how creating and classifying symbols in a R-R time series [24].

V. PSYCHOPHYSICAL STRESS DURING PHYSICAL EXERCISE

In recent decades, athletes and coaches have tried to find creative and innovative methods to improve the quality and quantity of athletes' training. These efforts have consistently faced obstacles concern with the stress load accumulated by athletes, that does not allow them to have always very high-performance during training and competitions [25]. Stress occurs under two different forms strictly correlated between them: *physic stress* and *psychological/mental stress*. Maintaining this stress distinction, the physic type stress includes problems of a strictly physical nature as *overtraining, fatigue, injuries.* Instead in mental stress, *illness* and *burnout* are the major found diseases. The term burnout, according to the World Health Organization (WHO), represents a syndrome resulting from chronic stress characterized by a series of phenomena of fatigue, disappointment, and low productivity that result in exhaustion and disinterest in one's professional activity. In order to avoid the unleashing of events leading to a compromised mental state, the first step is the prevention of overtraining. Athletes should be able to balance the physic stress of training and an appropriate recovery time. During overtraining, athletes find themselves on a chronic performance plateau that cannot be simply solve by short amounts of rest and recovery. Symptoms of overtraining are the "bridge factor" between physic and mental stress including depression, general apathy, emotional instability, impaired performance, restlessness, irritability, disturbed sleep, weight loss, increased resting heart rate, increased vulnerability to injury, hormonal changes, and lack of overcompensation. Thus, in a competitive level sport, when a condition of chronic stress occurs, optimizing the *recovery-stress state* is fundamental. The recovery is defined as an inter-individual and intra-individual multi-level (e.g. psychological, physiological, social) process in time for the re-establishment of performance abilities [25]. Effective recovery from intense training loads, often faced by elite athletes, can many times determine sporting success or failure. If, from one point of view, the physic stress is the first issue with which athletes have to faced trying to increase their performance, it can lead to the incoming of a psychological stress that cannot be overlooked. Mr. Janke (1976) identifies 5 categories of stressors relating to the sports field:

- External stressors: related to the environment (water sports; sports in extreme environments), sensory deprivation (headphones in skeet shooting), the risk of injury (in artistic gymnastic).

- Stressor due to the deprivation of basic needs (time zone can disturb sleep; climatic conditions).
- Performance stressor: excessive physical and but more mental pressure; excessive monotony and repetitiveness of training, performance anxiety and failures.
- Social stressors: conflicts (with coaches, teammates, parents, other reference figures), social isolation (continuous travel, many commitments can lead to the neglect of loved ones).
- Other stressors: difficult decision-making processes; uncertainties about their competitive future, etc. [26]

One of the psychological responses mainly to the last three stressors is *anxiety*. Many of the ways to cope with stress are aimed precisely at reducing anxiety, which can be so intense that it in turn becomes a source of stress. Anxiety is nothing more than the psychological reaction of fear towards events perceived as stressful and threatening. This mechanism is part of a particular automatic response to physical dangers, the so-called "fight or flight" response, normally present in all animals. Anxiety disorder occurs when the fight or flight response is regularly triggered by stimuli or situations that are not very dangerous and certainly do not represent a threat to survival. In this case, the athlete's perception to certain situation and self-state makes the differences and trigger anxiety disorder. Thus, the fight or flight response is an automatic response to the perception of a serious threat and cannot be changed. Instead, the way of interpreting situations and events can be changed, and it is the main purpose of mental training to which athletes are subjected [26].

8. Tools for Stress Evaluation

Evaluation and monitoring tools for the stress are important for assessing the individual's mood, his need for recovery and current living conditions. The advantage of psychometric tools is that they provide information quickly, while common physiological monitoring (blood analysis and specific medical diagnostics) can take hours to days for feedback. The following evaluation and monitoring tools can be applied in all sports. The *Profile of Mood States* (POMS) provides a self-assessment of mood and affective states and is often used in the psychological monitoring of training / overtraining / under recovery. The POMS is a 65-questions questionnaire in which responses are rated on a 5-point- Likert scale (Figure 21). It provides a measure of total mood

disorders and six moods (Tension, Depression, Anger, Vigor, Fatigue, Confusion) and is useful for detecting mood fluctuations during exercise. However, the POMS does not give information on the causes of overtraining. The first POMS questionnaire was developed in 1971 by Douglas M. McNair with Maurice Lorr and Leo F. Droppleman [27]. Currently, the most used is the POMS 2, which is available for adults aged 18 years and older (POMS 2–A) and another for adolescents 13 to 17 years of age (POMS 2–Y). The *Borg's Rating of Perceived Exertion* (RPE) is used for a wide variety of sports and exercise approaches, to measure the level of perceived individual exertion. The scale is a psychobiological measure used for monitoring the internal load of training sessions by means of a numerical and visual scale proposed individually to each athlete [28]. With it, athletes can identify the subjective perception of the effort regarding the session just carried out by answering a simple question *"How tiring was your training?* ". There is a well-established relationship between the training load (TL) and perceived effort: TL value, is obtained by multiplying the value indicated by the athlete, by the duration of the training session. This value allows an estimate of the commitment perceived and the monitoring of athletes' level of fatigue, avoiding the incurrence of overtraining syndromes. The most used scales to ask for the RPE athletes are shown in Figure 22.

Name: _____ Date:

Below is a list of words that describe feelings people have. Please **CIRCLE THE NUMBE**
THAT BEST DESCRIBES HOW YOU FEEL RIGHT NOW.

	Not At All	A Little	Moderately	Quite a lot	Extremely
Tense	0	1	2	3	4
Angry	0	1	2	3	4
Worn Out	0	1	2	3	4
Unhappy	0	1	2	3	4
Proud	0	1	2	3	4
Lively	0	1	2	3	4
Confused	0	1	2	3	4
Sad	0	1	2	3	4
Active	0	1	2	3	4
On-edge	0	1	2	3	4
Grouchy	0	1	2	3	4
Ashamed	0	1	2	3	4
Energetic	0	1	2	3	4
Hopeless	0	1	2	3	4
Uneasy	0	1	2	3	4
Restless	0	1	2	3	4
Unable to concentrate	0	1	2	3	4
Fatigued	0	1	2	3	4
Competent	0	1	2	3	4
Annoyed	0	1	2	3	4
Discouraged	0	1	2	3	4
Resentful	0	1	2	3	4
Nervous	0	1	2	3	4
Miserable	0	1	2	3	4

PLEASE CONTINUE WITH THE ITEMS ON THE NEXT PAGE

Figure 21: An example of a page of POMS with evaluation on a 5-point- Likert scale [27].

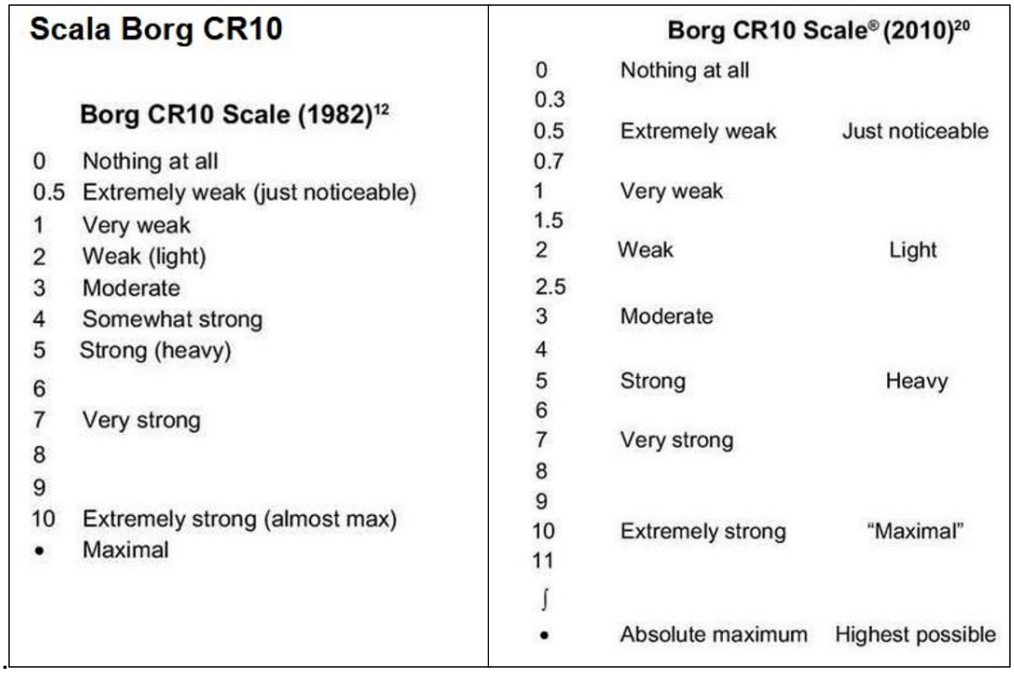

Figure 22: The most used scales for RPE answers [28].

Recently, Kentta¨ and Hassme´n have introduced *Total Quality Recovery (TQR)* which highlights the relationship between training and recovery. Its structure is similar to the RPE one, and the aim of this approach is to evaluate both recovery and under recovery [29]. Athletes using the TQR method collect points in a 24-hour period. 20 points are available divided in: 10 nutrition points, 4 sleep and rest points, 3 relaxation and emotional support points and finally 3 stretching and warm down points. A score of 13 is considered the minimum score so anything below this value represents an incomplete recovery.

Another approach, established by Hanin (2002), proposes that athletes possess an optimal functioning zone in which performance is maximized when an individual's subjective and emotional experience falls within this zone. Since the most common emotion in competition is anxiety, the *Individual Zones of Optimal Functioning (IZOF)* proposes that there are individual differences in the way people react to anxiety. Therefore, each person has his own preferred level of anxiety that allows them to perform at their optimum. When athletes are in their *optimal performance zone* this means that they are in they preferred level of anxiety. In addition to anxiety, IZOF allows the description of several emotional states which could be helpful or not. For example, some athletes may notice that feeling excited is not conductive to performing well, while others would say that feeling angry helps them to reach their optimal performance state. Thus, the IZOF model suggests that each athlete could find out his or her optimal combination of useful emotions and learn, how to reach this unique state prior to competitions.

The main criticism of the IZOF model is that it does not explain why some people perform better when in certain emotional states and others do not. A common way to find out the individual optimal performance zone is *individualized emotion profiling* a reconstruction of athlete's emotional experiences related to successful and poor performances [30].

The *RESTQ-Sport* (Kellmann & Kallus, 2001) systematically evaluates the recovery-stress state of an athlete. It indicates the state to which an individual is physically and / or mentally stressed and if the athletes is able to apply recovery strategies. The RESTQ-Sport evaluates the usage of these strategies on a Likert-type scale with values ranging from 0 (never) to 6 (always) indicating how often the participant has taken part in various activities over the past three days / nights. The RESTQ-Sport consists of 77 items (19 scales) to which the participants respond. High scores on the stress-associated activity scales reflect intense subjective stress, while high scores on the recovery-oriented scales indicate good recovery activities. The RESTQ-Sport consists of 7 general stress scales: general stress, emotional stress, social stress, pressure, fatigue, lack of energy, physical complaints; 5 general recovery scales: success, social recovery, physical recovery, general wellbeing, sleep quality; 3 sport-specific stress scales: obstructed pause, emotional exhaustion, injury; 4 sport-specific recovery scales: being fit, personal realization, self-efficacy, self-regulation. The high validity of the test-retest shows safe results in relation to short-term changes in the recovery-stress state and functional fluctuations [31].

Thus, as these monitoring and evaluation tools show, sport is not only a physical issue but also a mental one. The psychology of sport provides the athlete with tools that allow them to improve their performance, combining the indispensable physical training and the improvement of the athletic gesture, the use of techniques that work on the psychological dimension to obtain a better management of energy and emotionality in a training and sports competition situation.

9. Physiological Parameter for Stress Evaluation

The monitoring and evaluation of the physical and mental athletes' stress is not entrusted just to individual investigative tools such as questionnaires and, in general, tools described in the previous paragraph. A huge contribute to create a complete picture of the psychophysical condition of the athlete is given by the investigation of physiological parameters and in general biofeedback at rest and during activity. The most monitored physiological parameters and biofeedback are:

- *Surface electromyography* (EMG) records the electrical activity produced by a muscle when it contracts. The electrical signal recorded on the skin surface is the result of the simultaneous activity of numerous motor units. These motor units are under the voluntary control of the athlete; thus the EMG signal is easily controlled voluntarily. This investigation gives an assessment on the contraction muscle capacity that can be not maximum if some problems, both of mental and physical nature, occur in the athletes [32].
- *The heart rate* is how many times a heart beats per minute (bpm). The average bpm for a man or women who does not exercise is 70 bpm. Heart rate varies between people because of fitness, age and genetics.
- *The temperature measurement of the skin* in the peripheral area reflects the blood flow through the blood vessels under the skin. The change in this measurement is associated with sympathetic activity: the activity of the sympathetic nervous system increases peripheral vasoconstriction (the decrease in the diameter of the blood vessels induces a decrease in blood flow, with consequent lowering of temperature) while a reduction in the activity of the sympathetic nervous system and \ or an increase in that of the parasympathetic system leads to peripheral dilation (an increase in the diameter of the arteries leads to an increase in blood flow and therefore in skin temperature). It is an indicator of physiological flexibility and health. Working on this parameter helps relaxation and stress management by improving blood circulation [32].
- *The skin conductance measurements* reflect changes in the activity of the sweat glands caused by the activity of the postganglionic cholinergic fibers in the sympathetic nervous system. When sympathetic activation occurs, the sweat glands begin to secrete sweat with a proportional increase in the electrical conductance of the skin measured by a skin conductance sensor. It is usually associated with the activation at the cortical level of anxious thoughts, worries and other stressful thoughts so can represent an indicator of the psychological state of athlete [32].
- *The change in blood flow volum*e is recorded with a photoplethysmograph. It returns information regarding heart rate by calculating the volume changes of blood flow in the arteries, capillaries and any other tissue that follow each beat. It is therefore used as an index of blood perfusion and the elasticity of the arteries and blood pressure, therefore as an index of cardiovascular health. Changes in heart rate are measured by calculating

the time between one beat and the next leading to an assessment of the HRV. Cardiac variability is an index of physiological well-being, a large cardiac variability is characteristic of a physiological system that easily adapts to stressful situations. The two parameters considered in HRV are amplitude (which represents quantity) and consistency (which represents quality). [32].

- *Breathing*. The way in which the individual breathes influences and reflects his psychological and physiological state. Breathing occurs automatically during daily activities. It is controlled by the automatic nervous system and there is no awareness about the breathing patterns except in difficult situations. In these cases, bad breathing can be reflected in low performance as it affects not only the physical performance but also the mental one, hindering the search for the optimal strategy to deal with a given situation. Despite this, it is possible to voluntarily influence breathing by increasing self-awareness of respiratory functioning through self-observation. Thus, breathing changes are expected during times of physical or mental stress. Two common parameters are the respiratory rate (RR), which is the number of breaths taken in one minute and minute ventilation and the volume of air that is inhaled or exhaled in one minute. Sinus respiratory arrhythmia (RSA), a process in which the heart rate changes according to respiratory activity, is the mechanism that most affects cardiac variability. In particular, during exhalation the heart rate decreases, while during inspiration the heart rate increases. Furthermore, difficult mental tasks are associated with reductions in oxygen saturation (SO_2) which is the proportion of oxygenated haemoglobin to total haemoglobin in the blood [32].

Lastly, the activity *of hypothalamic–pituitary–adrenocortical system* (*HPA*) with the secretion of cortisol, and that of *sympathetic adrenomedullary system* (*SAM*), with the secretion of alpha-amylase, have been largely used as objective markers of psychophysiological performance stress. Cortisol is a good indicator of stress. This steroid hormone plays a central role in the physiological and behavioural response to stress. In detail, salivary cortisol (SC), as a representative marker of circulating free cortisol, has been recommended as an index of training stress. An increase in this hormone is important during preparing for mental and physical competitive demands: greater cortisol levels before competition exhibited a greater performance. However, extremal high level of cortisol may lead to poor performance because it may interfere with some cognitive processes [33].

10. Stress Differences between Training and Competition

Previous studies on stress responses to competition and training suggests a high psychophysiological demand reflected on psychological (rate of perceived exertion 'RPE', anxiety, etc) and physiological (heart rate 'HR', cortisol) response. These studies have shown that psychophysiological challenges associated with training and competition settings can induce different responses, especially in the case of hormonal parameters [33][34]. Psychological and physiological stress components seem to be more incisive during the competition match day (MD) compared to the training day (TD). In particular, the study of J. Fernandez-Fernandez et al. [34] is reported to confirm the findings mentioned above. The study compares the psychophysiological stress responses during a real competitive match and a training session in a group of high-level young female tennis players. The first finding is that MD induces higher stress responses compared to TD. Moreover, results show a significant effect of winning or losing on SC levels in athletes, with the highest concentrations observed for the losers. However, the latter is in opposition to previous research in other sports that reported no differences in cortisol increases between losers and winners suggesting that the specific athletes' characteristics and/or the physical demands of the sport could also influence SC responses [34]. Regarding the anxiety components, significant differences are found comparing MD and TD, noting higher anxiety components during TD. Thus, SC concentrations before competition on MD were significantly and positively correlated to cognitive and somatic anxiety scores which agree with previous research. In contrast, this relationship was not observed on TD. These results suggest that neuroendocrine responses and anxiety scores are related only under the competitive match play, also supported by the relationship between workload parameters (i.e., HR, RPE) and questionnaire scores before and after competition. In summary, the study confirms the different psychophysiological responses of young tennis players regarding the importance of competition (MD vs. TD). Real competitive environments stimulate a higher psychophysiological response in players over the entire day, especially in losers. Moreover, the relationship among hormonal level, self-reported stress scores and some physiological parameters during matches is more clearly linked during real competitions [34]. The reported study provides a general picture on the literature state of art about the main differences stress responses between competition and training [34].

VI. MATERIALS and METHODS

11. Data Acquisition

Data for the analysis conducted and reported in this thesis were provided by Cardiovascular Bioengineering lab. Data are a collection of 200 electrocardiographic traces acquired by 10 different subjects, all practicing the same sport: athletics. The acquisitions were made via *Alivecor's Kardia Single Lead* which is part of a range of clinical quality mobile ECG recorders (Figure 23), during two different workouts (one strength and one speed) and in competition.

Figure 23: Alivecor's Kardia Single Lead, a part of a range of clinical quality mobile ECG recorders.

The *speed training* was composed of a first part of warm-up, a speed test central part and a final part in which the subjects cool down, running in the most possible unconstricted way to relax the muscles and, subsequently, doing stretching exercises. In details, in the warm-up, the subjects run mildly for 10 minutes; subsequently they carry out a series of joint mobility exercises to warm up all the joints subjected to prolonged stress. This first part of warm-up lasted about 30 minutes. The second part of the warm-up involves more dynamic exercises carried out for about another 30 minutes, such as running with high knees in place, running back kick, running under kick and two accelerations on 20/30 meters. After this warm-up part, three speed tests were carried out. In particular, the subjects have to run two tests of 120 meters in length in which 40 meters were run in a rather controlled way so as to assume a correct running attitude, and the remaining 80 meters were all run at maximum speed and then, a final test of 150 meters. This part takes about 30 minutes. In the last part, the athletes concluded the training with a very light run of 5 minutes and with stretching exercises. During the speed training, 7 ECGs were acquired. The first represents the resting ECG, the second identifies the

ECG acquired after 5 minutes of rest from the end of the warm-up; the third and fourth are the acquisitions relating to specific training exercises containing respectively the cardiac data acquired after running the second test, or the 120m and those obtained at the end of the third test, or after the 150m. The last three ECGs were acquired during the rest period 5, 10 and 15 minutes after the end of the training.

Strength training begins with a first part of warm-up, in which the subjects carry out joint mobility exercises and subsequently, a series of exercises on the spot to warm up all the muscles of the lower limbs such as squats, squat jumps, lunges back and forth. Subsequently, they perform 10 repetitions of each of the two exercises on which the training is focused, namely the rapid continuous half squat jump and the step jump on a 40 cm step. This first part of warm-up last about 45 minutes. Subsequently, in the central part of the training, which lasts approximately 30 minutes, 5 series of the two exercises mentioned above are carried out, each of the series consisting of 6 repetitions. Between the series, the subjects recover 2 minutes, while between one exercise and another the recovery is 4 minutes. Finally, after the strength exercises, the athletes conclude with stretching exercises. During the strength training, 7 ECGs were acquired. The first represents the resting ECG, the second identifies the ECG acquired after 5 minutes of rest from the end of the warm-up; the third and fourth are the acquisitions relating to specific training exercises containing respectively the cardiac data acquired after the first and the exercise. The last three ECGs are acquired during the rest period 5, 10 and 15 minutes after the end of the training.

The race includes an initial part of warm-up with about 7 minutes of light running followed by joint mobility. After this first part of warm-up, another one follows in which all the more dynamic exercises are carried out. It ends with some extensions, running in the most relaxed way possible for about 80 meters 3 or 4 times, and, finally, accelerations of about 15/20 meters are made to try the start. This whole part of the pre-race general warm-up lasts approximately 1 hour and 10 minutes. Subsequently, the subjects take part in the competition by running a 200-meter test. At the end of the competition, after having partially recovered, the athletes cool down with a light 5-minute run followed by stretching exercises. During the competition, 6 ECGs were acquired. The first represents the resting ECG, the second identifies the ECG acquired after 5 minutes of rest from the end of the warm-up; the third is the acquisitions

immediately after the race. The last three ECGs are acquired during the rest period, after 5, 10 and 15 minutes from the end of the race.

12. Population

The database comprises 10 subject, 9 female and 1 male, aged between 15 and 22 years old, practicing the same sport: athletics. The median weight is 59 kg, and the mean height is 166,5 cm. All the anthropometric data are shown in Table 2. All subjects were asked to fill a questionnaire which revealed no subject is a smoker or chronically uses alcohol; furthermore, the subjects do not suffer from any cardiovascular disease and any autonomic control impairment. No one takes drugs and they are all in good or excellent physical shape. All subjects train at least 4 times per week with an average duration of 2 hours per workout.

Table 2: Anthropometric data in median and percentiles (25-75), gender shows M/F. M stands for male, F for female.

	Age (year)	***Weight (kg)***	***Height (cm)***	***Gender***
Subject1	22	54	156	F
Subject2	16	53	165	F
Subject3	16	58	164	F
Subject4	15	58	172	F
Subject5	17	60	163	F
Subject6	16	54	160	F
Subject7	20	60	168	F
Subject8	21	60	185	M
Subject9	18	62	174	F
Subject10	19	70	180	F
Total	17,5 [16,0; 19,9]	59 [55,0; 60,0]	166,5 [163,3; 173,5]	1/9

13. Extraction of RR series

Data were analysed in Matlab® (The MathWorks, Natick, MA, USA) environment which allowed the identification of R peaks using the *pan_tompkins* function. Since the acquisitions were saved in EDF format, they were converted in MAT format using Python environment in order to get they ready to be loaded. All ECG signals were subjected to a filtering operation in which two filters were applied: first a high-pass filter with cut-off frequency f1 = 5 Hz which

served to eliminate the baseline, and then a low-pass filter with f2 = 15Hz which allowed the elimination of line noise. Subsequently, the pan_tompkins function was applied on ECGs to allows the identification of R peaks. Once peaks were identified, some adjustments were performed to allow that the found peaks coincide with each maximum of the QRS complex. To do this the *max* function was applied considering a fraction of the ECG for time giving the position of the maximum in that fraction of time. In particular, this function considers a round of a delta amplitude equals, in samples, to the delay introduced by pan_tompkins filtering. At this point, in order to know the correct position of R peak, the delta was subtracted from the R-position previously calculated by the algorithm, the value of the position obtained from the max function was added and, finally, one sample was subtracted. With this operation, the correct positions of the R peaks have been obtained for each ECG and saved. The '*diff*' function has been applied to the peak position vectors saved for each ECG, in order to extract the temporal distance between one peak and the next: the RR series or Tachogram (Fig 24). To remedy some artifacts, present in the data acquisition, the tachogram has been adjusted by imposing two conditions: if the tachogram has two consecutive samples one greater and the other lower than 25% of the mean, both are replaced with two samples of equal amplitude to the mean. The second condition requires that all samples with an amplitude equal to or greater than twice the average of the signal are split into two distinct samples with the same amplitude. For each tachogram the percentage of adjusted values does not exceed 25% of the total values so as not to change the initial tachogram too much.

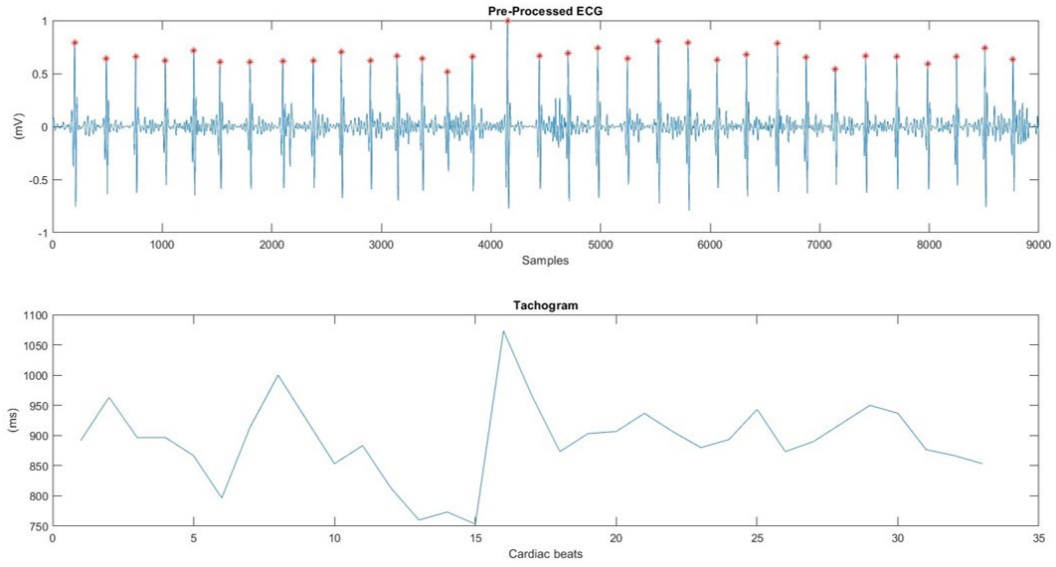

Figure 24: Example of tachogram extraction from a series of R peaks in an ECG.

14. Symbolic Analysis

The symbolic analysis applied in this study is the same explained and detailed in *Porta et al., 2007* [35]. A quantization procedure of £ symbols was applied. Each RR series was divided in £ layers of amplitude equal to the difference between the RR maximum and minimum divided by £. Any RR value falling into a given layers was substituted with a symbol ranging between 0 and £-1. This procedure allows to turn the RR sequence into a series of symbols. Starting from the symbolic series patterns formed by L consecutive symbols are created. Traditional setting suggests a value of £= 6 and L = 3 [35][36]. Patterns were classified according to the significance and sign of the variations computed over two consecutive symbols. Four types of patterns are extracted: the stable patterns with no variation (V0) that have same symbols; patterns with one variation (V1) so two consecutive equal symbols and the other different, and patterns with two variations (V2). Here a distinction is made. Two like variations (LV2) presenting symbols that were all different between them but the sign of the two variations was equal (both increasing or decreasing) and patterns with two unlike variations (UV2) showing variations between two consecutive symbols having opposite sign. The results of symbolic analysis were expressed as percentages of patterns belonging to a type, over the total number of patterns. Moreover, literature shows that the increase of 0V% concerns with situations of augmented sympathetic control and vagal withdrawal, while the 2UV% raising indicates an increment of vagal control and a sympathetic withdrawal. Finally, the mean of each RR series is calculated.

15. Statistical Analysis

In order to decide the statistical test to be employed in this analysis, the *Lilliefors test* is performed on the results of symbolic analysis. The Lilliefors test detect if a distribution of data is normal or not and it is implemented in Matlab environment using the *lillietest* function. Since some distribution is not normal, so reject the null hypothesis, *median* and 25^{th} and 75^{th} *percentiles* are calculated to represent the distribution of the results. Finally, the distribution of V0, V1, LV2, UV2 incidence and RRmean are compared between them in the following way: the main distinction is between ‘strength, speed and competition’; then six conditions are considered (the first exercise in strength and speed is delated to compare only the last exercise both in training and competition) and the values of features (V0, V1, LV2, UV2, RR-mean) are

compared with the corresponding ones in the same condition but in different type of physical activity (strength, speed, competition). Thus, the total number of comparisons is 90. Since the data distribution is not normal, all the matches are performed employing Wilcoxon rank sum nonparametric test in Matlab environment with the *ranksum* function. The null hypothesis of test is 'medians are equal' that can be rejected or not. Between 90 comparisons, 16 reject a null hypothesis with a p-value lower than 0.05. At the end, one of these 16 has been discarded for a movement artifact in the acquisition of original signal.

VII. RESULTS

Symbolic analysis results are reported in Tables 3-4 showing the distributions of V0, V1, LV2, UV2 incidences and RRmean in terms of their own measures of centrality (median) and dispersion (25^{th} and 75^{th} percentile). Thus, each table has 7 rows and 10 columns. Columns are arranged in 3 main group representing the phase in with signals were acquired, then each group is divided in other 3 columns representing the type of training or competition: 'V' stands for speed training, 'G' for competition and 'F' for strength training. In rows there are the incidences of the four patterns provided by symbolic analysis and the mean of each tachogram, expressed as median and 25^{th} and 75^{th} percentile. Figures 25-29 show the same data of tables above with error bar graphs. Moreover, results of non-parametric test, performed on the 90 comparisons and described in '*Statistical Analysis*' chapter, are represented in figures by a horizontal line and an asterisk indicating which comparison reports a p-value lower than 0,05. Between the 90 comparisons, 15 of them show a significant statistical difference.

Table 3: Median and percentiles of the four symbolic markers and RR mean in the first three phases of both training sessions and of the competition

	Pre-warm-up			***Post-warm-up***			***Post Exercise***		
	V	***G***	***F***	***V***	***G***	***F***	***V***	***G***	***F***
RRmean [ms]	712,7 [662,0; 804,4]	653,1 [611,7; 718,0]	731,9 [693,3; 761,4]	611,0 [554,5; 683,9]	592,7 [564,3; 641,2]	628,8 [567,2; 656,3]	410,8 [401,6; 436,3]	450,5 [432,5; 495,5]	525,6 [517,4; 539,8]
V0 [%]	27,0 [10,3; 34,3]	31,0 [24,0; 41,8]	18,5 [12,8; 30,8]	45,5 [19,8; 51,5]	43,0 [29,3; 61,5]	41,5 [23,3; 51,5]	54,0 [45,8; 69,5]	38,5 [18,5; 54,5]	35,5 [19,3; 60,5]
V1 [%]	44,0 [36,0; 50,3]	36,5 [30,0; 43,0]	45,0 [41,5; 48,8]	32,0 [21,3;43,5]	32,0 [25,8; 37,5]	33,5 [28,0; 45,3]	25,0 [18,0; 28,8]	32,5 [24,5; 38,8]	36,0 [28,5; 44,5]
LV2 [%]	11,5 [6,5; 24,8]	11,5 [8,8; 13,0]	13,0 [10,3; 17,5]	9,0 [3,5; 14,0]	6,0 [2,3; 12,0]	7,0 [3,3; 13,8]	2,5 [1,3; 5,5]	4,5 [3,0; 9,5]	9,5 [0,0; 12,3]
UV2 [%]	18,0 [7,5; 21,0]	21,5 [15,3; 25,5]	20,0 [15,0; 23,8]	15,5 [11,8; 20,3]	13,5 [10,0; 20,0]	15,0 [8,5; 16,8]	15,5 [8,0; 20,8]	23,0 [13,8; 29,8]	12,0 [9,5; 20,5]

Table 4: Median and percentiles of the four symbolic markers and RR mean in the last three phases of both training sessions and of the competition.

	Post 5 minute of rest			***Post 10 minute of rest***			***Post 15 minute of rest***		
	V	***G***	***F***	***V***	***G***	***F***	***V***	***G***	***F***
RRmean [ms]	539,9 [525,1; 542,8]	547,0 [530,9; 563,2]	666,5 [622,9; 729,7]	554,5 [499,8; 589,9]	555,2 [510,7; 573,1]	658,7 [630,3; 732,5]	560,0 [551,9; 638,9]	589,8 [502,7; 679,0]	705,5 [641,6; 731,5]
V0 [%]	44,5 [33,0; 64,5]	54,0 [33,3; 71,8]	30,5 [19,8; 35,3]	37,5 [27,5; 56,5]	33,5 [28,5; 47,3]	21,0 [18,0; 30,8]	50,0 [32,8; 61,3]	51,5 [24,8; 63,5]	42,5 [21,5; 51,0]
V1 [%]	35,0 [19,5; 46,5]	24,0 [12,0; 34,8]	39,0 [33,8; 51,0]	33,0 [26,3; 35,5]	39,5 [35,8; 43,8]	45,0 [37,0; 51,3]	31,5 [24,8; 45,0]	32,5 [23,8; 41,5]	36,0 [24,0; 37,5]
LV2 [%]	4,5 [2,5; 8,0]	5,0 [1,0; 7,8]	9,5 [7,3; 11,8]	8,5 [4,0; 11,5]	5,5 [4,0; 9,3]	9,5 [6,3; 15,3]	7,5 [4,5; 9,0]	3,5 [2,0; 5,0]	4,5 [3,0; 9,5]
UV2 [%]	11,0 [5,3; 16,5]	17,0 [12,3; 19,8]	22,0 [16,3; 27,5]	13,0 [9,0; 24,5]	19,0 [13,0; 24,0]	18,5 [17,0; 24,5]	9,5 [5,0; 16,5]	12,0 [9,3; 26,3]	24,0 [13,5; 25,8]

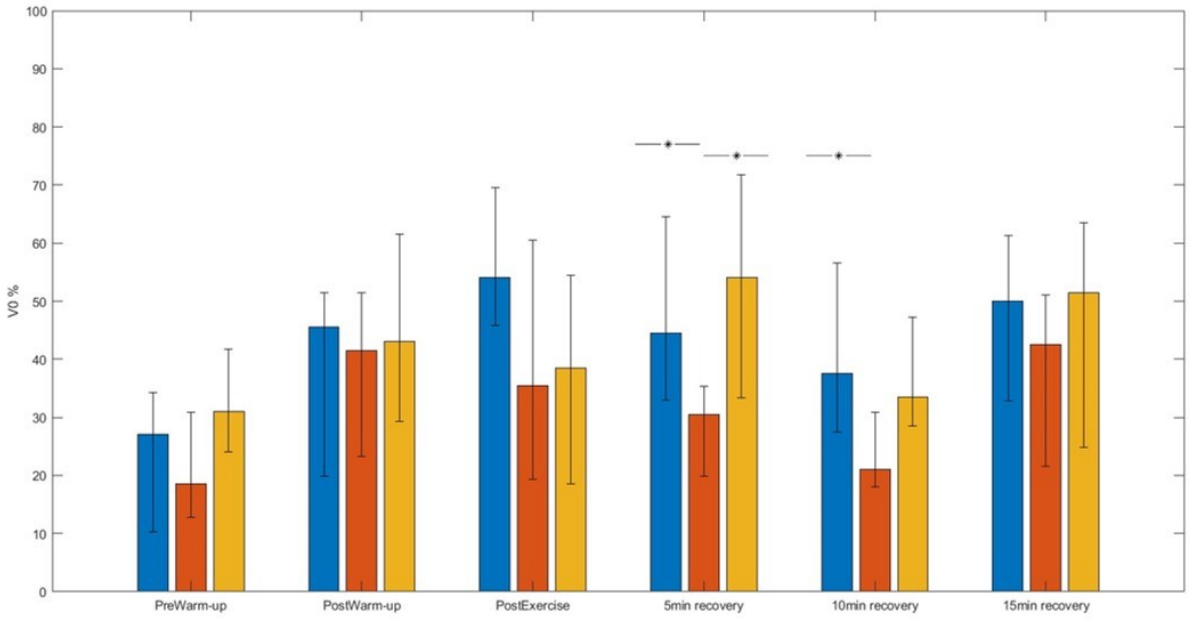

Figure 25: The error bar graphs show the symbolic feature V0 % incidence. Data are reported as median plus 25th and 75th percentile. The symbol * indicates p-value<0.05 between analysed comparisons as result of Wilcoxon rank sum test.

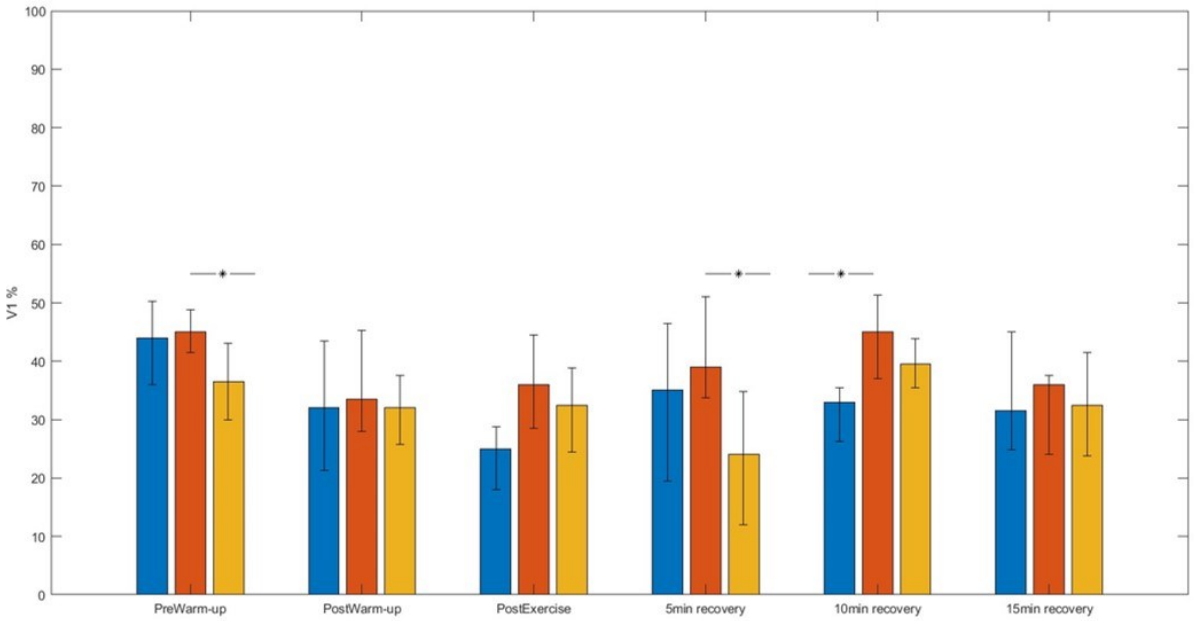

Figure 26: The error bar graphs show the symbolic feature V1 % incidence. Data are reported as median plus 25th and 75th percentile. The symbol * indicates p-value<0.05 between analysed comparisons as result of Wilcoxon rank sum test.

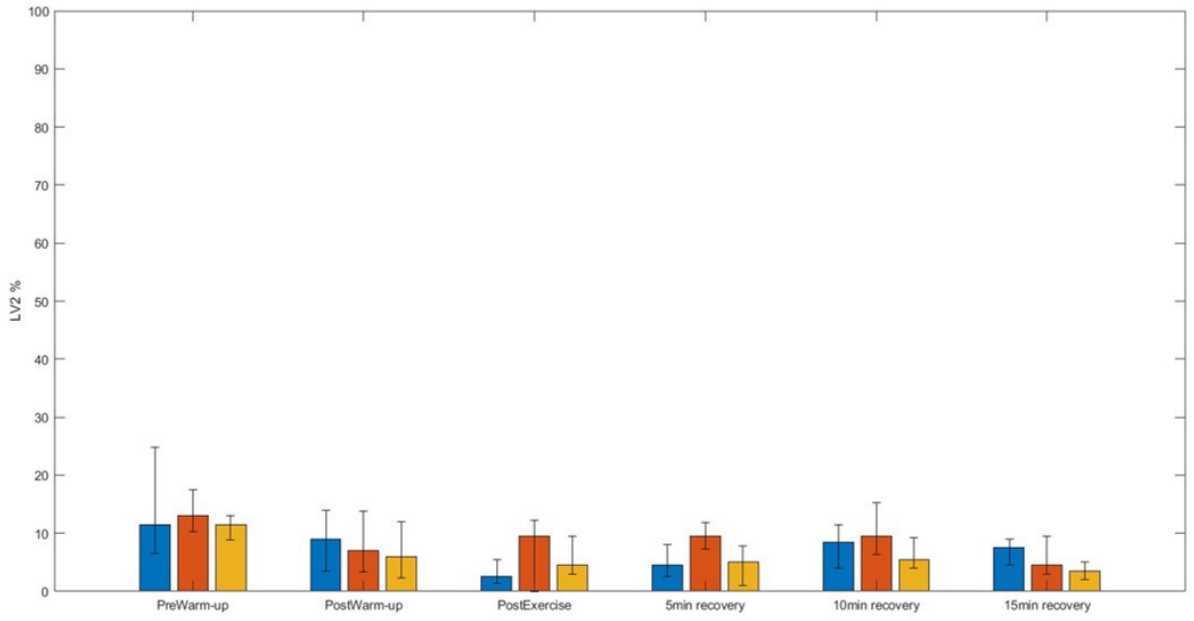

Figure 27: The error bar graphs show the symbolic feature LV2 % incidence. Data are reported as median plus 25th and 75th percentile.

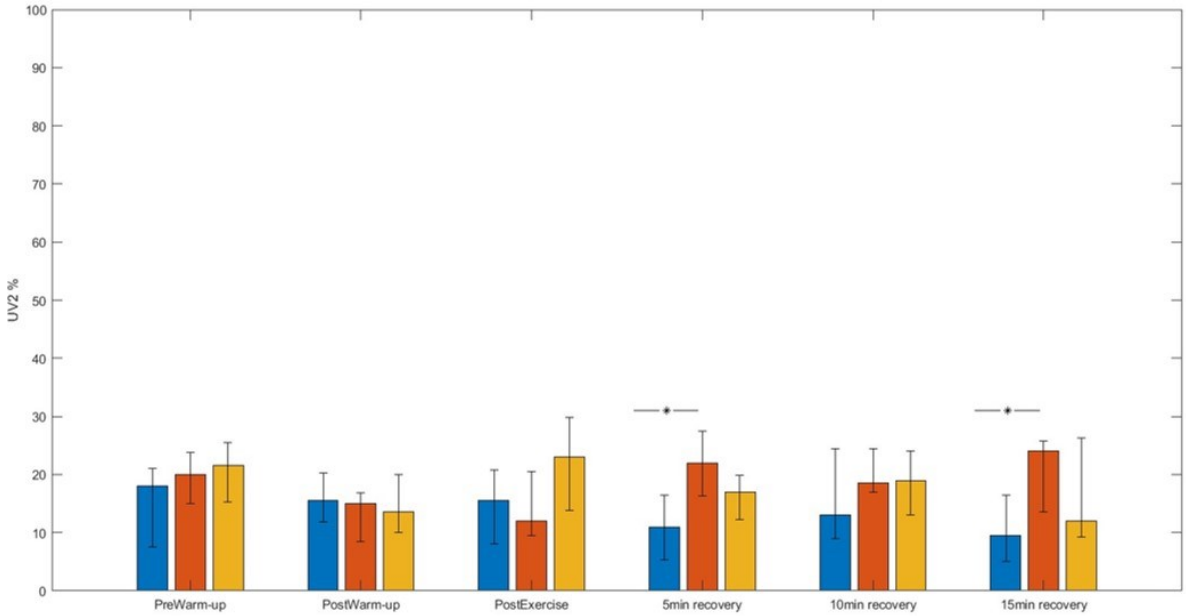

Figure 28: The error bar graphs show the symbolic feature UV2 % incidence. Data are reported as median plus 25th and 75th percentile. The symbol * indicates p-value<0.05 between analysed comparisons as result of Wilcoxon rank sum test.

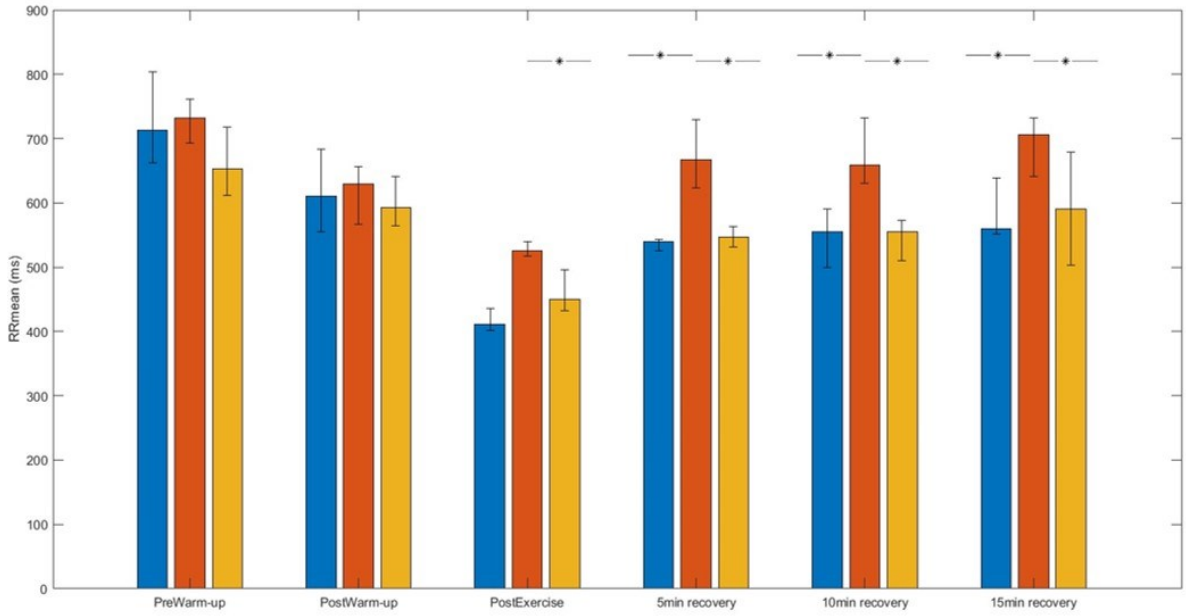

Figure 29: The error bar graphs show the parameters RR mean. Data are reported as median plus 25th and 75th percentile. The symbol * indicates p-value<0.05 between analysed comparisons as result of Wilcoxon rank sum test.

VIII. DISCUSSION

This work evaluates sport-related psychophysical stress during athletics training and competition exploiting symbolic analysis. Symbolic analysis already has widespread application in many sectors, especially in the economic and financial fields and in the last 20 years, its use in the medical field, nowadays limited to research, has increased a lot. The main purpose of introducing this tool as support to doctors in the diagnostic step is to unmask certain pathological heart conditions not visible in a simple ECG especially in athletes. Indeed, all the features characterizing the athlete's heart may hide abnormal values of heart parameters, so pathological conditions would be not detected, and the probability of sudden death during sport increases. In this study, no significant differences outcome from the comparison of features extracted in speed training and in competition, thus only the other two matches show statistical differences. This suggests that similar sporting gestures, such as those in a running training and a 200-meters race, do not provide noteworthy differences in terms of sympathetic or parasympathetic control on the heart, although the psychological approach dedicated to a competition is different from that of a workout. This finding is confirmed by the similarity among the features trends extracted in speed training and competition, and by the differences with those extracted during strength training. Thus, can be concluded that the type of sporting gesture, aerobic or anaerobic, affects the heart presumably more than the mental condition. Secondly, the incidence value of V0 is major in speed training and competition than in strength training, in particular after 5 and 10 minutes of rest. Since V0 is related to sympathetic activity [24], this result suggests that greater sympathetic control is found during a short run at maximum speed (training or competition) than in strength workout. In addition to the sport differences described above (anaerobic and aerobic), a psychological factor can be identified. All subjects under study are sprinters, thus, significantly increase the level of attention and 'positive anxiety' in the race and in simulating the sporting gesture (speed training) with respect to the strength exercises. This leads to greater activity of the sympathetic system, that releasing more adrenaline and noradrenaline [1][2], maintains a psychophysical state of maximum attention. Indeed, the values of RRmean parameters confirms a high sympathetic activity in competition and speed workout than in strength training. In matches where the V0 parameter is higher, the RRmean parameter is lower, as expected since it is known as an increase in sympathetic activity (represented by a high V0) increases the heart rate and so makes the mean of tachogram lower. The other parameter to be analyzed is UV2.

From a statistical point of view, as reported in Figure 28, the value of this parameter is higher in strength training than in other condition after 5 and 15 minutes. Since UV2 reflects the activity of the parasympathetic nervous system and a sympathetic withdrawal, its value in strength training, where there is a minor contribution of the sympathetic system (low V0), should be higher than in speed training and competition. Human body always tries to maintain a sort of balance between sympathetic and parasympathetic control, thus normally, as one increases, the other decreases. In certain conditions, however, when one of the two activities rises a lot, the other follows the trend to maintain balance. For this reason, the symbolic features used in this study, provide the possibility to detect nonreciprocal changes in sympathetic and parasympathetic modulations or reciprocal changes with different magnitudes [36]. For what concern the V1, it is not clear how this parameter is correlated to the nervous system in literature, thus, no physiological assessments can be done in those matches showing statistical differences in V1 trends. Similar considerations are found about LV2 since this parameter does not show any significant differences in matches but, however the UV2 incidence represents a reliable parameter for the estimation of the parasympathetic activity much more than LV2. Another interesting finding is that about 93% of the matches that report statistical differences, are located in the phases following physical effort (post exercise and in rest time) and report a general level of sympathetic activity higher in competition than in training. To explain this finding, it could be considered that there may be a psychological component, linked to the great investment, in terms of time and dedication, and to the great importance that the subject gives to the race. The lack of relaxation from a mental point of view, after the race, could lead to different activity of sympathetic system with higher value of V0 incidence in race than training. Despite the interesting results obtained in the study, it is not possible to prove with certainty that the increase in sympathetic activity is due to psychological stress rather than to a physical one, probably both. Indeed, no psychometric tests were administered to the athletes during the acquisition, so no data were collected to better explain the rule of psychological stress in the sympathetic activity. Thus, it is recommended to implement this type of analysis and data collection in future studies in order to enrich the evaluation and make it more reliable. Moreover, a larger dataset, coming from a larger population of athletes, would perhaps have provided clearer results, a normal distribution of the incidence of V0, V1, LV2, UV2 among the subjects, and the study in general would have been more robust and reliable.

IX. CONCLUSION

The study suggests that symbolic analysis is an optimal tool for evaluating the control of the sympathetic and parasympathetic nervous system on the heart, while subjects are practicing sports that do not allow for long ECG recordings. Furthermore, the symbolic analysis of the tachogram provides information about psychophysical athletes' state and can be used as a complementary tool to stress assessment tests. The results suggest a general greater sympathetic nervous system contribution in competition than in training, as expected. If this finding is strictly due to the difference between sports gestures (aerobic and anaerobic) or to a different mental condition cannot be certainly stated, probably both of these factors make their contribution. Results of this thesis will be deepened in the future to improve its limits so that this type of analysis becomes applicable and useful not only in the case of athletes, but in all those cases in which it is not possible to register long-term ECGs. Moreover, future studies could be focused on the development of a neural network using symbol patterns as features to detect anomalies that could hide heart or even psychological diseases. Finally, this technology could be implemented in sports clinics, giving support to the medical team for a more meticulous diagnosis on athletes, trying to reduce or delete the cases of 'sudden cardiac death in athletes' a problem that is currently still open.

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