Design of a MATLAB Graphical User Interface for Advanced PC-based Phonocardiography

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Abstract. In this paper the design of a Graphical User Interface (GUI) is proposed for healthcare applications. The GUI is designed in MATLAB environment and is specifically suitable for advanced phonocardiography (PCG). This diagnostic method is more and more important nowadays, due to the advances in signal processing methods and computer processors performances in order to detect with a simple PC based electronic stethoscope, pathologies otherwise detectable with much more expensive and time-consuming medical techniques and devices.

Keywords: MATLAB, GUI, Phonocardiography.

I. INTRODUCTION

Phonocardiography (PCG) is the study of the graphic representation of heart sounds and murmurs in order to perform diagnosis about earth valves disorders. Heart sounds and murmurs are commonly detected through auscultation, practice used for a long time, and the only instrument needed is the stethoscope. The importance of the auscultation can be explained by the simplicity of the technique and by the great capacity of the human ear to recognize acoustic phenomena. However, to obtain equivalent information with PCG, a single recording is not enough: it is required to have a set of properly processed signals, each of them having higher frequency components gradually emphasized. It is then possible to visually examine the acoustic phenomena in different spectral bands, each of them adapted by an appropriate amplification to provide for the degradation of the intensity of the heart sounds towards high frequencies [1].

Heart sounds and murmurs were recorded for the first time with a modified Frank's capsule: it consists of a thin rubber membrane that seals the ending of a tube connected to the bell of a stethoscope. A small mirror is fixed at the extremity of the membrane and the phonocardiogram is recorded photographing a light beam reflected from the mirror. The apparatus was difficult to use and, since the subsequent membranes were different in thickness, the sensitivity to low frequency vibrations were low [2].

In 1816 René Laennac invented the stethoscope, laying the foundation for modern auscultation [1]. In 1895 Karl Hürthle was the first to record heart sounds on paper with the help of a microphone [1]. In 1907 Willem Einthoven, the inventor of electrocardiography, was the first to record the phonocardiogram with a carbon microphone and a rope galvanometer, [1, 3].

Recent progress in electronics allowed the creation of much more satisfying devices. In 1941 Rappaport and Sprague described the physical law that govern auscultation and phonocardiography, allowing the production of more accurate phonocardiograph. Heart sounds and murmurs are gathered by a crystal microphone and recorded on a multichannel electrocardiograph by a mirror galvanometer [2]. In the second half of the twentieth century studies on the use of ultrasound methods for cardiac examination grew and echocardiography proved to be a valid alternative to PCG [1].

When there is an uncertainty following an auscultation, the phonocardiogram is of great help. This occurs particularly when the murmur is small or unusual, or when tachycardia makes auscultation difficult. However, despite of its great diagnostic capability, the PCG techniques in past years has assumed a role of secondary importance due to the development of techniques of medical imaging. Anyway, these techniques and related medical devices are very expensive, the physician performing the examination and the diagnosis must be expert, and the waiting time required to perform such medical examinations is generally quite long. Nowadays, it is possible to revalue the PCG technique in order to perform a quick and easy first screening diagnosis of cardiac valvulopathy. In fact, it is possible to use an electronic stethoscope together with a proper signal processing algorithm implemented in a personal computer (PC) in order to have an automatic diagnosis of suspected valvulopathies. Details about the algorithm and the stethoscope will be proposed by the authors in a paper in progress.

In order to connect the stethoscope to the PC and in order to implement the auto-diagnostic algorithm, it is necessary to design a simple and easy to manage Graphical User Interface (GUI), allowing the physician, also if not expert, to perform the diagnosis.

To this aim we have designed a GUI, described in this paper. Therefore, in section 2 a deep discussion about the heart sounds is performed; in section 3 valvulopathy characteristics are described in order to emphasize and properly underline the importance of such diagnosis by PCG; in section 4 the GUI is deeply described; in section 5 tests and results are shown; conclusions and final remarks are in section 6.

II. THE HEART AND VALVULOPATHIES

2.1 The cardiac cycle

The heart is a powerful muscle that serves as a pump to push blood through the circulatory system. The contraction of the heart is due to the electric impulse generated by the sinoatrial node. When the heart contracts itself, blood flows through the valves of the atria to the ventricles and finally through the body.

The heart is composed of four cavities (or chambers), two upper and two lowers. The upper cavities are the left atrium and the right atrium, the lower ones are the left ventricle and the right ventricle. Every cavity has four valves that operate as a connection for the chambers. There are two types of valves: atrioventricular valves (tricuspid valve and mitral valve) and semi-lunar (pulmonary valve and aortic valve), that prevent the reverse flow of the blood.

The cardiac cycle includes two essential phases in which the activity of the heart takes place: systole and diastole. During the diastole all the heart is relaxed, allowing blood to flow into the four cavities. Blood flows from the veins into the right atrium and from the pulmonary veins into the left atrium.

The cardiac valves are open at the same time and allow the blood to flow from the atria to the ventricles. The diastole lasts 0,4 seconds circa, enough to allow the ventricles to fill almost completely.

The systole starts with a contraction of the atria, lasting 0,1 seconds circa, that determines the complete filling of the ventricles. Then the ventricles contract for about 0,3 seconds. Their contraction closes the atrioventricular valves and opens the semi-lunar valves; the oxygen-poor blood is pushed towards the lungs, while the oxygen-rich one goes towards the whole body through the aorta.

The state of the cavities of the heart and of the valves during the systole and diastole is described in tables 1 and 2, respectively.

Table 1-State of the cavities of the heart and of the valves during the systole

SYSTOLIC PHASE	Initial	Central	Final
Atria	Empty	Filling	Full
Atrioventricular valves	Closed	Closed	Closed
Ventricles	Full	Emptying	Empty
Semi-lunar valves	Closed	Opened	Opened
Blood flow		From ventricles to the major arteries; from major veins to the atria	

Table 2-State of the cavities of the heart and of the valves during the diastole

DIASTOLIC PHASE	Initial	Central	Final
Atria	Full	Emptying	Empty
Atrioventricular valves	Closed	Opened	Opened
Ventricles	Empty	Filling	Full
Semi-lunar valves	Closed	Closed	Closed
Blood flow		From atria to ventricles	

These cardiac phases can be heard and translated through two different sounds, called cardiac tones. When the ventricles contract, we have the first tone, generated from the vibrations of the atrioventricular valves closing. After the first tones there is a pause during which the ventricles push blood into the arteries. Subsequently the second tone occurs, determined by the vibration of the semi-lunar valves closing. Following the second tone there is a longer pause, with the filling of the ventricles.

In rest condition the heart can pump around 5 liters of blood per minute; this quantity can increase considerably, thanks to compensation mechanisms, following a more or less intense physical activity. Based on the variation of pressure gradients of atria and ventricles (Figure 1), the main phases that constitute the cardiac cycle can be highlighted (Figure 2).

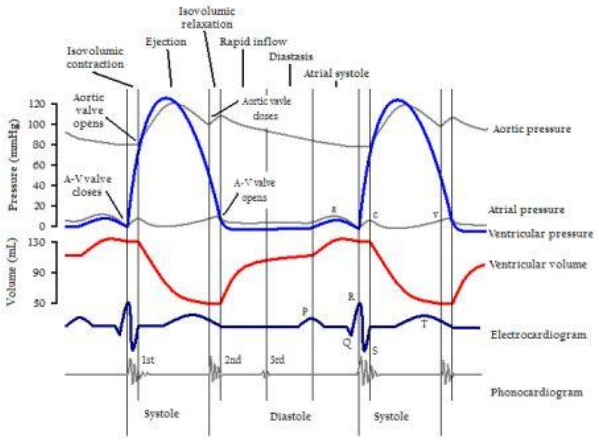

Figure 1- From top to bottom: pressure and volume trend of the heart during the cardiac cycle; electrocardiogram (ECG); phonocardiogram (PCG).

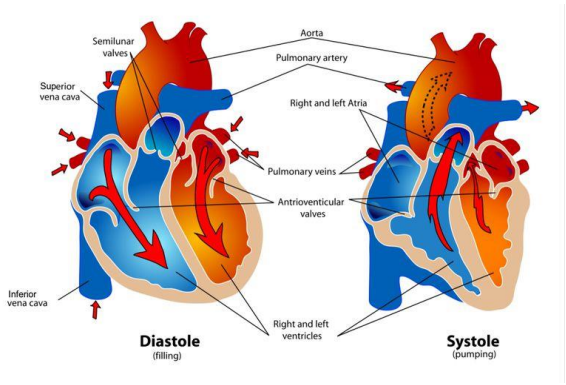

Figure 2-Phases of the cardiac cycle

In the first phase, called atrial systole, the force of the atrial contraction allows the complete emptying of blood from the atria towards the ventricles. During this phase, the atrioventricular valves are opened; the ventricles are released and filled with blood. The semi-lunar valves are closed, to prevent blood from flowing into the pulmonary artery or from the aorta to the ventricles. Afterwards an atrial relaxation occurs, the so-called atrial diastole. The isovolumetric ventricular contraction follows, which occurs between the start of the systole and the opening of the semi-lunar valves; in this phase the ventricular volume remains constant, meanwhile the pressure increases rapidly. This period of contraction lasts for about 0.005 seconds, that is until the semi-lunar valves open and blood is push out of the heart; this occurs when the pressure gradient in the ventricles exceeds the pressure in the pulmonary artery (10 mmHg) and the aorta (80 mmHg). When the semi-lunar valves open, the ventricular ejection phase starts, which is first rapid but then slower as the systole progresses. A considerable quantity of blood, called residual volume, in physiological condition, remains into the ventricles at the end of the ejection period. At the end of the ventricular ejection, the semi-lunar valves close so that the blood, from the large vessels, cannot re-enter the ventricular cavities. The atrioventricular valves remain closed until the pressure into the atrial cavities exceeds the one in the ventricles in diastole. Thus, there is a drastic decrease of intraventricular pressure, but without a variation of volume. Both valve systems are closed, and the ventricles are relaxing. The rapid inflow last 0.1 seconds and gives rise to a drastic increase of ventricular volume. In particular, the phase that describes the slow and delayed ventricular filling at the end of the diastole is called diastase; indeed,

the sudden inflow of blood that occurs immediately after the opening of the atrioventricular valves is followed by a slow but continuous blood flow from the atria toward the ventricles. The diastase lasts for 0.2 seconds and is characterized by a gradual increase of the intraventricular pressure and volume.

2.2 Heart valves

Heart valves are the structure that separate the cardiac chambers, atria and ventricles, and the latter from the large vessels, aorta and pulmonary artery.

There are four heart valves and can open and close in sync with the heartbeat, so that blood can only travel in one direction. Two of them, called atrioventricular (AV) valves, are located at the level of the openings situated between atria and ventricles. The other two, called semi-lunar (SL) valves, are located where the pulmonary artery and the aorta originate from the right and the left ventricle, respectively.

The AV valve that regulates the right atrioventricular orifice, called tricuspid valve, consists of three endocardial leaflets, while the one that controls the left atrioventricular orifice is called the mitral valve and has two leaflets. The constructive modes of both valves allow the blood to flow from atria towards ventricles but prevents backflow from ventricles to atria. The ventricular contractions force the blood that is in the ventricle against the flaps of the cuspid valves on their lower side, causing its closure and ensuring the movement of blood towards the pulmonary artery or the aorta during the ventricular contraction. The SL valve that controls the beginning of the pulmonary artery is the semi-lunar pulmonary valve, while the one at the entrance of the aorta is the semi-lunar aortic valve.

When these two valves are closed, blood fills the spaces between valve leaflets and vessel wall, and each leaflet looks like a little stuffed pocket. On the contrary, the blood flow coming from the ventricle towards the vessel releases the valve leaflets and leads them to collapse with the wall of the blood vessel, thus causing the opening of the valves. The closing of the SL valves, like that of the atrioventricular, immediately prevents the backflow of blood upstream and ensures a unidirectional floe, even when dynamic moments occur which would be able to cause the backflow.

2.3 Heart sounds

There are two audible heart sounds during the heart cycle in normal conditions, the first and second tone. A heart tone is defined as a single acoustic event preceded and followed by a pause.

The bipartition of a tone, instead, occurs when it is possible to distinguish two separate components separated by a short pause, and it is possible to appreciate splitting of about 20-30 ms. That said, it is crucial to know the duration of sounds and pauses in order to filter accordingly the record recorded in phonocardiography: the bipartition of heart tones, for example, is recognizable only in high frequency. The performance achievable with PCG exceed that of the human hearing, allowing to record also low frequency sounds otherwise inaudible to the ear.

Acoustic phenomena produced by the heart are classified in tones and murmurs. Heart tones have a periodic nature, they are brief and originate as a result of sudden events such as the closure of a valve, causing resonant phenomena in the cardiovascular system; murmurs are mainly noisy and of longer duration, they originate from turbulent blood flow.

The 4 heart tones are usually indicated as I, II, III and IV (or also S1, S2, S3, S4). The first two tones are related to the closure of cardiac valve, the third and fourth one, are rather weak and observable only in a small group of people.

2.3.1 Primary heart sounds

First heart tone (S1)

The first tone is generated after the closure of the mitral valve and the tricuspid valve, during the isovolumetric contraction phase of the cardiac cycle and the opening of the aorta valve, and therefore the ejection. Filtering at high frequency, it is possible to observe the splitting of the first tone, with the components due to the closing of the mitral valve (indicated with Is o M1), the closing of the tricuspid valve (Ib o T1), and the opening of the aortic valve. When the heartbeat is quite high, the first tone is higher [1].

Cardiovascular disease can influence the intensity and timing of the first tone components. A wider splitting is observable in the right bundle branch block, the tricuspid stenosis and the atrial septal defect, due to a delay of the Ib component. In the left bundle branch block, instead, the two components Ia and Ib can be overlapping. An attenuated first tone can be found when there is a reduce cardiac contractility (such as myocardial infarction, cardiomyopathy, heart failure), left bundle branch block, mitral insufficiency and aortic stenosis. The first tone appears amplified in case of mitral stenosis and atrial septal defect [1].

When there is a strong systolic or diastolic murmur, the first heart tone may not be distinguishable during auscultation, however the phonocardiograph will show that the first tone vibrations are slightly different from the murmur ones. Sometimes the phonocardiograph shows that the first tone is missing and the protosystolic phase sound is mistaken as the first tone. Occasionally, when a phonogram shows a crescendo first tone, a protosystolic murmur is diagnosed. This error does not occur if the diagnosis of mitral stenosis is made only when a mesodiastolic murmur is heard. In mitral stenosis the first tone is delayed, and the degree of delay measured by the phonogram can indicate the severity of the disease in the future [2].

Second heart tone (S2)

The second heart tone generates at the closure of the aortic valve and, subsequently, the pulmonary valve, to which are associated the two components indicated respectively with IIA (or A2) and IIP (or P2). The bipartition is more noticeable during the inhalation due to the greater difference of the duration of the left and right ventricular systole. The so-called splitting paradox of the second tone happens when the pulmonary component IIP precedes the aortic one IIA; this condition is pathologic and is caused by the delayed closing of the aortic valve. The stiffening of the valve leaflets causes a reduction of the second tone, while its increase is due to larger valves or a lower blood viscosity [1].

A wider bipartition of the second tone may be due to a delayed closure of the pulmonary valve (caused by the right bundle branch block, pulmonary stenosis, pulmonary hypertension, or an atrial septum defect) or an early closure of the aortic valve (caused by a mitral insufficiency or a ventricular septum defect). The splitting paradox may be due to a delayed closure of the aortic valve (caused by a left bundle branch block, aortic stenosis or arteriosclerosis) or the early closure of the pulmonary one (caused by tricuspid insufficiency or early activation of the right ventricle) [1, 2].

Thanks to phonocardiography, it is possible to correctly diagnose the opening tone of the mitral valve rather than the splitting of the second tone in mitral stenosis.

2.3.2 Extra heart sounds

Third heart tone (S3)

The third heart tone occurs during the fast-ventricular filling phase. It is believed that the third tone generates from the sudden deceleration of the blood flow when the ventricle reaches its limit of distensibility, casing vibrations in the ventricular wall. It can be heard in children and teens but can also be recorded in adults at low frequency. The disappearance of the third heart tone through the years is a consequence of the myocardial mass increase having a dumping effect on the vibration. Over the age of 40, the reappearance of the third tone is pathological and is due to mitral insufficiency, aortic stenosis or ischemia [1]. Its presence is rather serious, unless it only occurs with tachycardia, when it may be due to a summing gallop (sum of the protosystolic tone with the protodiastolic one). When the nature of the gallop rhythm is uncertain, the phonocardiograph will clarify the problem [2].

It is difficult to tell the difference between a long third heart tone and a short diastolic murmur during auscultation. The phonocardiogram allows to measure the duration of the vibrations and, if these last more than 0.16 seconds, they constitute a diastolic murmur [2].

Fourth heart tone (S4)

The fourth heart tone coincides with the atrial contraction with and increased blood flood. Its recording is rare, usually only in older people and at low frequencies. The sound is more evident in case of an increased ventricular filling or reduced ventricular distensibility. Pathological causes of the fourth heart tone can be mitral insufficiency, aortic stenosis, hypertension or ischemia [1].

Other pathological sounds that can be detected are ejection clicks (due to congenital valvular aortic stenosis or lungs with narrow valve leaflets) and the non-ejection systolic clicks (caused by the mitral valve prolapse in the left atrium). The opening tone can occur when the mitral valve opens in case of a valvular stenosis [1].

2.3.3 Heart murmurs

Heart murmurs have a different nature than tones. They originate due to the turbulent blood flow. Normally, indeed, the blood flow in the blood vessels is of the laminar type [1]. Murmurs occur when the heart rate increases or the viscosity of the blood decreases. The most common murmurs are proto-systolic and meso-systolic, have a short duration and coincide with the maximum ventricular outflow. At the increase of the blood flow, the turbulence increases and consequently the intensity of the heart murmurs.

The presence of murmurs at non-high flow velocities can be pathological, and its causes can be an irregular opening of the valve, valvular insufficiency or a communication between the left and right side of the heart [1].

Murmurs divide in systolic, diastolic and continuous.

Systolic murmurs

The phonocardiogram id very useful to distinguish systolic ejection murmurs in pulmonary and aortic stenosis from regurgitation murmurs in mitral or tricuspid insufficiency, or on the intraventricular septum defect. Ejection murmurs are diamond shaped and the aortic stenosis one ends before the second heart tone (Figure 3) [2]. The pulmonary stenosis murmur reaches the aortic component of the second tone but ends before the delayed pulmonary component (Figure 4). Regurgitation murmurs in mitral or tricuspid insufficiency and in the intraventricular defect fill the whole systole (Figure 5 and 6). The tricuspid murmurs are of more variable intensity than the others, and often show a noticeable change during respiration [2].

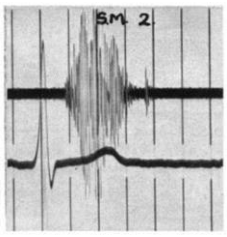

Figure 3- PCG with aortic stenosis [2].

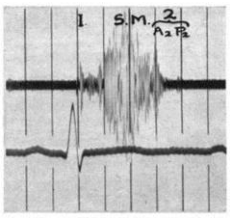

Figure 4-PCG with pulmonary stenosis [2].

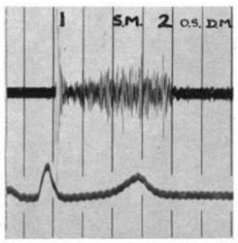

Figure 5-PCG with mitral insufficiency [2].

Diastolic murmurs

When the diastolic murmur from aortic or pulmonary regurgitation is audible at its peak, it can be hard to decide if there is also a diastolic murmur or a Flint murmur [2]. The phonocardiogram of aortic or pulmonary diastolic murmurs has a characteristic trend first in crescendo then in decrescendo (Figure 6). This can be distinguished from the trend of the diastolic trend in mitral stenosis, that starts only after the opening of the atrioventricular valves, and it is made of more irregular vibration (Figure 7) [2]. The first delayed tone and the opening tone of the mitral valve also confirm the presence of mitral stenosis.

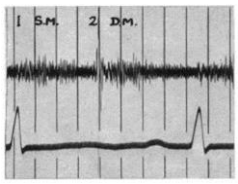

Figure 6-PCG with aortic insufficiency [2].

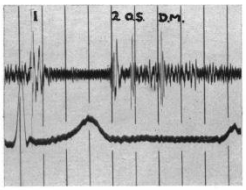

Figure 7-PCG with mitral stenosis [2].

Continuous murmurs

Continuous murmurs are due to the patency of the ductus arteriosus. The phonocardiogram of the murmur of a patent ductus arteriosus shows that the intensity of the vibrations is maximum during the second heart tone and minimum in the beginning of the ventricular systole (Figure 8) [2]. When the murmurs of a pulmonary or aortic disease coexist, the phonocardiogram, often helps to identify them separately. Other continuous murmurs due to conditions as arteriovenous aneurisms or the breaking of the aortic sinus in the right ventricle can show characteristic easily identifiable from the phonocardiogram (Figure 9) [2].

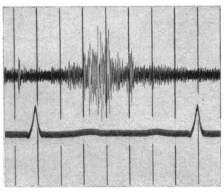

Figure 8-PCG with patent ductus arteriosus [2].

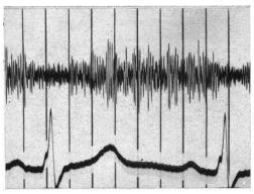

Figure 9-PCG with the breaking of an aneurism of the right sinus of Valsalva [2].

2.3.4 Cardiac vibrations

The mechanical action of the heart can be indicated as a set of time-signals: the most important variables are the blood pressure in cardiac cavities and blood vessels, the tension of the myocardial

wall, the ventricular volume, the speed of the blood flow, the deformation of the heart wall and its movement. Only kinematic information is available on the chest surface, i.e. the movement of the chest surface because of the mechanical action of the heart.

Regarding the heart movement, it is defined in relation of a balance position: we can speak of a vibratory phenomenon. This movement of the chest surface creates an acoustic pressure in the air; it is generally so weak that it can't be perceived by listening to it from a certain distance from the chest. Only if we use closed air cavities the ear can hear sounds: in fact, the closed cavity (as the stethoscope) prevents the acoustic energy dispersion and the attenuation of the acoustic pressure [1].

Since we are in the presence of a vector function, we should consider its three components. Basically, only the component perpendicular to the chest surface is measured and the two tangential components are ignored. A kinematic function can be represented by different temporal representations, such as, for example, the movement, speed and acceleration.

The mechanical effects of the cardiac action on the chest surface are not completely studied by phonocardiography. Historically the frequency spectrum is divided in two parts: low frequencies (up to 20 Hz) are treated by mechanocardiography, high frequencies (over 20 Hz) by phonocardiography. In addition, movements of the chest wall are not exclusively caused by the action of the heart, but also by breathing, which produces low frequency movements (0.2-0.4 Hz), due to the respiratory action itself, and at high frequency, generated by the turbulent air flow in the airways and lungs. From the point of view of phonocardiography, these effects are a source of noise and should be minimized [1].

III. HEART VALVE DISORDERS

Heart valve disorders are called valvopathies and can be of two types: stenosis and insufficiency. Stenosis, which consists of an incomplete opening of the valve leaflets, causes the blood to flow through a smaller orifice compared to normal physiological conditions. It can be of congenital nature, in case of stenosis that occur since birth, or degenerative, in case of stenosis that occur during life, after valve leaflets diseases. If the valve stenosis condition occurs, the heart must provide a higher quantity of energy than it is required during normal conditions, trying to ensure the physiological well-being of the organism. The heart, in fact, developing compensation mechanisms, can produce more work. Heart walls undergo structural changes to allow the fibers that compose it to appropriately increase the ventricular systolic pressure. There is therefore a thickening of the heart walls, hypertrophy and a spherification of the ventricle. Hypertrophy is a compensatory mechanism aimed at giving greater vigour to ventricular contraction, in order to force the anatomical obstacle represented by stenosis. If the stenosis is not critical, hypertrophy is compensatory, and the patient is asymptomatic. Over time, inevitably the symptoms appear, as the valve stenosis progress, caused by the continuous hemodynamic stress, and hypertrophy, like all compensation mechanisms, faces some problems. Valve insufficiency, instead, consists in the incomplete closing of the valve leaflets; this allows the onset of a haematic reflux from the arteries to the ventricles, in case of an insufficient semi-lunar valves, or from the ventricles to the atria, in case of insufficient atrioventricular valves. The incomplete closing is due to leaflets anomalies or of the structures connected to the leaflets themselves. Usually however stenosis and insufficiency coexist, in different degrees, in the same valve, creating the so-called steno insufficiency. As mentioned, valvulopathies can be congenital, if present since birth, or acquired, because they appear during life. The latter may be degenerative, more frequent in elderly subjects, often hypertensive, as they are due to the wear of the valvular structures, infective, such as endocarditis, ischemic, during acute myocardial infarction, traumatic (very rarely) or secondary to conspicuous

dilatation of the ventricle and/or the large vessels. In recent decades, one of the main causes of valvulopathies was rheumatic fever, which arose as a complication of a pharyngitis or tonsillitis, due to streptococcal infection. The valves affected by this infection are damaged progressively deforming. Nowadays, with the improvement of life condition, the reduction of infections and the increase in life span, the most frequent cause of valvulopathy is degenerative, due to the progressive deterioration of the valvular structure that happens with aging. The course of valvulopathies is, in most cases, slowly evolutionary, with a very long phase of complete asymptomaticity. On the other hand, if the valvulopathy arises acutely on a valve, that was normal up to that point, following trauma, myocardial infarction, endocarditis with perforation of valve leaflets, the clinical presentation can be dramatic. The diseases of the right side of the heart (tricuspid and pulmonary), where there is a low pressure, are rare and generally due to congenital problems. On the other hand, mitral and aortic diseases are much more frequent.

Mitral stenosis (MS)

It is defined as an alteration of the mitral system which involves an obstruction to the passage of blood from the atrium to the left ventricle during the diastole.

The normal mitral area is 4-6 cm2; stenosis is defined as mild when the area is approximately 2 cm2, moderate when is about 1.5 cm2, severe when it is about 1 cm2. In most cases mitral stenosis is a rheumatic disease and appears to be more common in female individuals. Valve leaflets in the initial state appear widely thickened and fibrotic with fusion of the joints. With the progression of the disease, areas of calcifications appear, initially located at the joints, later spread to leaflets. The set of all these morphologic alterations implies a reduced excursion of the leaflets, especially in the back leaflet, that creates the stenosis. The obstruction of the blood flow from the atrium to the left ventricle, induced by such pathology, implies a blood stagnation in the left atrium and a consequent increase in pressure and size of the left atrium. The atrial dilatation and the fibrous cloth cause an altered conduction of the cardiac impulse which favours the establishment and the maintenance of atrial fibrillation. Stasis of blood flow in the left atrium, due to the mitral obstruction and the loss of atrial systole caused by the fibrillation, can cause thrombus formation. Thrombi can embolize or more rarely engage in the stenosed mitral orifice. The increase of left atrial pressure has an immediate impact on pulmonary circulation. Lungs face modifications in both vascular and parenchymal components. The required exam to arrive at a certain diagnosis is the echocardiogram. The electrocardiogram and the chest x-ray, once widely used, have now only a minor role, as they are not very specific. There are currently four operations divided in two categories: reparative operations and replacement operations. The type of operation and timing are related. The timing for reparative operations, considering the low risk and the success of the results in the long run, it is earlier than in cases where valve replacement is necessary. The reparative operation is possible when the valve is fibrotic. The valve replacement is indicated in patients with a valve no longer repairable. The surgical operation includes the abscission of the native valve, with the consequent removal of the calcium deposited on the annulus, and finally the implantation of the prosthesis which is sutured to the annulus.

Mitral insufficiency (MR)

While the mitral stenosis has acute joint rheumatism as its sole cause, the peculiarity of mitral insufficiency is the extreme variety of causes (degenerative disease, rheumatic disease, infective endocarditis, dilated myocardiopathy). The most frequent cause today is the myxomatous degenerative mitral disease. The connective tissue that constitutes the leaflets and the tendinous cords undergoes a

structural modification that leads to a greater laxity. The tendinous cords stretch, and the leaflets tend to prolapse in the left atrial cavity. The structurally altered cords, subjected to a higher hemodynamic stress, frequently undergo a break-up increasing the amount of regurgitation. Echocardiographic and radiographic investigations that allow to assess the severity of mitral insufficiency, are not very specific and not very sensitive. In severe forms of mitral insufficiency even severe regurgitation can occur without significant changes in the electrocardiogram and the chest x-ray. Indeed, these two exams cannot provide the essential information for a proper patient management. Currently every attempt is made to repair the valve, instead of replacing it. The main benefits of conservative surgery compared to replacement consist in avoiding problems related to valve replacement (embolisms, thrombosis, malfunction) and in keeping a normal left ventricle function. The results of reparative surgery are extremely positive; the long-term survival of patients undergoing valve repair is greater than that of patients with prostheses ad is about 95% at 5 years.

Aortic stenosis (AS)

The aortic stenosis is the obstruction of the blood flow from the left ventricle in the aorta due to an aortic valve disease. The area of an aortic valve is usually 1.5-2.5 cm2; the stenosis is called critical when the area is less than 0.8 cm2, the medium gradient greater than 50 mmHg and the maximum gradient greater than 75 mmHg. The aortic stenosis is responsible for about 25% of all valvulopathies. Most patients with aortic stenosis (85%) are male. The main forms of aortic stenosis are the congenital form, the rheumatic form and the calcific form. The congenital form is represented as a mono or bicuspid valve and it is the most common cause of aortic stenosis under 30 year. The rheumatic form is due to the localization of the rheumatic disease; often it is also accompanied by valve insufficiency and coexists with a mitral valvulopathy. The degenerative calcific form is proper to the elderly patient and is caused by the calcified degeneration of the valve tissue, accentuated by chronic hemodynamic stress. It is the most frequent cause of aortic stenosis for patients over 70 years. The instrumental techniques used to diagnose aortic stenosis are chest x-ray, that shows the hypertrophy of the left ventricle, and echocardiogram or also the color-doppler. Aside from the simple diagnosis it is also possible to evaluate the severity of the disease and therefore the opportunity of surgical correction. Given the evolutionary nature of the disease, the symptomless patient is controlled annually. The symptomatic patient with severe stenosis is always destined to surgery. The surgical intervention for an aortic stenosis is a valve replacement. The operation is performed by sternotomy in extracorporeal circulation. The valve is replaced with a prosthesis (mechanical or biological) or with a human valve taken from corpse and cryopreserved (allograft). In children with non-calcified aortic stenosis it is possible to dilatate the valve with the percutaneous balloon technique or open the joints with a simple surgical incision in extracorporeal circulation. These are obviously procedures that can be implemented while waiting for surgery.

Aortic insufficiency (AR)

Aortic insufficiency is the incapacity of the valve to maintain diastolic competence. It is due to various causes grouped in to two categories: primitive diseases of the aortic valve and disease of the aortic root. The severe articular rheumatism is still the most common cause of aortic insufficiency. The valve appears thickened and deformed. The patient with chronic rheumatic aortic valve insufficiency remains asymptomatic for a long time. At this stage an unspecific tachycardia sensation may occasionally be present. The first symptoms occur usually around 40-50 years. The onset of symptoms coincides with the ventricular dysfunction phase. A doctor can diagnose such pathology subjecting the patient to a chest x-ray, that shows the enlargement of the left ventricle, and the echocardiogram.

Depending on the cause that caused the aortic insufficiency, the doctor decides the type of surgery to perform. In the forms caused by a primitive valvular disease, the surgery consists in the valve replacement. The replacement is still executed, in most cases, using a mechanical or biological prosthesis. In cases of associated aortic root pathology, it is necessary the removal of the valve and the aortic root, with implantation of a valved vascular prosthesis on which the coronary plugs are re-implanted with different techniques.

Pulmonary stenosis

By stenosis of the pulmonary valve we mean a narrowing of the pulmonary valve orifice which determines an obstacle to the free passage of blood from the right ventricle to the pulmonary artery: the quantity of blood that will reach the small circulation (pulmonary) will be less than at the physiological conditions.

In almost all cases, this cardiopathy is caused by a congenital defect.

The stenosis of the pulmonary valve can be valvular, subvalvular or supravalvular, but most patient fall in the first case. Pulmonary stenosis is in most cases caused by a fusion of the commissures, while in about 20% of the cases the valve is dysplastic, a situation that affects mostly patients with Noonan syndrome. The subvalvular pulmonary stenosis is a muscle pathology (caused by an excess of muscle tissue) and it is almost always associated to other injuries, especially to an intraventricular defect. The supravalvular pulmonary stenosis is almost always part of the clinical picture of multisystem pathologies such as Williams syndrome. Obviously also in this case the valve will be carefully studied during the echocardiographic exam and, depending on some parameters that the echocardiographic cardiologist or the sonographer that performs the exam provides (of these one of the most important is the average pressure gradient), the stenosis of the pulmonary valve will be defined as mild, moderate or severe and depending on its classification a specific therapy will be chosen.

The stenosis of the pulmonary valve determines a pressure overload in the right ventricle. Due to this pressure overload (that can increase up to 5 or 6 times compared to the physiological value) the right ventricle hypertrophies (increases its thickness).

When the pulmonary valve stenosis is rather severe the patient may show signs and symptoms of heart failure: this happens when the right ventricle can’t produce such pressure to overcome the obstacle constituted by the valve stenosis: contractility decreases, the ventricle dilates and signs of heart failure appear (hepatomegaly and peripheral edema). The heart failure however is very rare in childhood, whereas it is much more frequent in adulthood if the primum movens is not corrected at the right time.

In mild pulmonary stenosis, in which the right intraventricular pressure is not so high to require a treatment, to the patient is only recommended to perform frequent cardiological and echocardiographic checks. The most frequently encountered symptoms are asthenia, dyspnoea exertional, syncope due to exertion, retrosternal pain.

Pulmonary valve stenosis can be treated either with a percutaneous valvuloplasty or by surgical correction. Percutaneous valvuloplasty with a balloon catheter is chosen if the echocardiogram shows a maximum gradient greater than 40mmHg. In almost 98% of the cases, in fact, through a balloon catheter the stenotic valve dilates and so the obstacle to the ejection of the right ventricle is removed. Such procedure can be done even during neonatal age. If percutaneous valvuloplasty has no positive

effects, surgery is performed in extra-corporeal circulation and involves the surgical opening of the pulmonary valve and its replacement with a biological or mechanical valve. Also, the subvalvular pulmonary stenosis must be treated surgically by removing the muscular structure causing the stenosis itself.

The success rate of percutaneous procedures is about 90% with a mortality risk of 1%. Even in case of surgery there is a very low mortality rate (less than 1%), if we exclude the possible complications induced by being in the neonatal age and by a hypoplastic ventricle.

Tricuspid stenosis (TS)

It is a valvular pathology that determines a shrinkage of the tricuspid valve orifice area which causes an obstruction to the hematic flow from the right atrium to the right ventricle.

The venous oxygen poor blood flow from the atrium to the ventricle through an opening on which three fibrous leaflets are inserted. These leaflets, or cusps, form the right atrioventricular valve also called tricuspid valve. The free ends of the cusps attach to the tendinous cords. These beams originate from the papillary muscles that depart from the inner surface of the right ventricle. This valve, like all the atrioventricular valves, during the period of ventricular relaxation (ventricular diastole) in which the ventricles fill with blood, has to be opened. During the phase of ventricular systole (ventricle contraction) the blood that leaves the ventricle opens the semi-lunar valves, while the blood that flows back to the atria closes the atrioventricular valves.

Tricuspid stenosis is almost always of rheumatic nature. Rarely it is determined by SLE, extrinsic compression, right atrial myxoma, carcinoid syndrome and, even more rarely, can be of congenital nature.

Tricuspid stenosis is classified in mild, moderate and severe depending on different parameters (blood flow velocity, area of the valve orifice, area of the indexed valve orifice, pressure gradient, atrium and ventricle wall thickness during systole and diastole, reduce slope of EF) which are provided by the echocardiographic cardiologist which executes the exam or by the sonographer. The most frequent symptoms in tricuspid stenosis are dyspnoea and orthopnea. They are associated with a sense of discomfort in the neck region, easy fatigability, cold skin and all secondary symptoms of reduced cardiac output (amount of blood expelled from the heart at each systole).

It is fundamental, in tricuspid stenosis, cardiac auscultation which reveals the presence of a heart murmur. The best treatment, according to the most recent guidelines, is surgery with valve replacement. The prognosis of patients with tricuspid stenosis is positive, and the success rate of surgery is close to 88%. Possible complications are due to patient comorbidity (diabetes, hypertension, etc.).

Tricuspid insufficiency (TR)

It is a valvulopathy that leads to a blood reflux from the right ventricle to the right atrium during the systole. The most frequent cause is the dilatation of the right ventricle. The symptomatology is usually absent, but severe tricuspid insufficiency can determine a pulsation of the neck, an olosystolic murmur and a heart failure caused by a disfunction of the right ventricle or atrial fibrillation. The diagnosis is

made through an objective and echocardiographic exam. Tricuspid insufficiency is usually benign and doesn't require a treatment, but some patients need annuloplasty, repair or valve replacement.

Tricuspid insufficiency can be primary or secondary (more common).

Primary insufficiency of the tricuspid is less frequent. It can be due to valve anomalies caused by infective endocarditis in intravenous drug users, carcinoid syndrome, closed thoracic trauma, rheumatic fever, idiopathic myxomatous degeneration, congenital defects. Secondary tricuspid insufficiency is more frequently caused by the dilatation of the right ventricle with the malfunctioning of a normal valve, as occurs in pulmonary hypertension, heart failure due to dysfunction of the right ventricle and in obstruction of the pulmonary outflow tract.

Tricuspid insufficiency is usually asymptomatic, but some patients feel a pulsation of the neck due to the high jugular pressure. Symptoms of severe tricuspid insufficiency include fatigue, abdominal bloating, and anorexia. Patients can also develop symptoms of fibrillation.

At auscultation, the first heart tone (S1) can be normal or barely audible if a there is a tricuspid insufficiency murmur; the second heart tone (S2) can be split or single due to the prompt closure of the pulmonary valve. A third right ventricular tone (S3) can be heard in proximity of the sternum in case of heart failure due to dysfunction of the right ventricle. The tricuspid insufficiency murmur is often inaudible. When present, is a holosystolic murmur better audible with the stethoscope bell at the level of the middle-lower left sternal margin or at the epigastrium, with the patient sitting upright or standing. The murmur can high frequency if the tricuspid insufficiency is very mild and is due to pulmonary hypertension or can be medium frequency if tricuspid insufficiency is severe and due to other causes.

When tricuspid insufficiency is moderate or severe, peak velocity underestimate the pulmonary pressure. Bidimensional echocardiography detects structural anomalies present in primary tricuspid insufficiency. Cardiac MRI is currently the method of choice to evaluate the size and right ventricle function, which generally must be performed when the quality of the echocardiographic image is inadequate.

The ECG and the chest x-ray are also often performed.

The ECG is usually normal, but in advanced cases, can show high P-waves, at peak, caused by the dilatation of the right ventricle, a high R or QR wave in V1 characteristic of right ventricular hypertrophy, or atrial fibrillation.

The chest x-ray is usually normal, but in advanced cases with right ventricular hypertrophy or with right heart failure, it may show a dilatation of the superior vena cava, a silhouette of the right ventricle or the enlarged right atrium (behind the sternal handlebar in lateral projection) or the presence of a pleural effusion. Severe tricuspid insufficiency has an inauspicious diagnosis, although initially well tolerated. As for valvular insufficiencies of the left heart, the ventricle subjected to volume overload eventually irreversibly decomposes.

Very mild tricuspid insufficiency is a normal finding and doesn't require any action.

Patients with a very severe tricuspid insufficiency must undergo surgery as soon as symptoms appear despite medical treatment or when there is a moderate and progressive enlargement or dysfunction of the right ventricle. During surgery for left cardiac lesions, moderate or mild tricuspid insufficiency with dilatation of the ring <40 mm must undergo repair.

Surgical options include annuloplasty, valve repair and valve replacement.

Recording of heart sounds

In auscultation, the doctor uses a stethoscope as a practical alternative to place the ear in direct contact with the chest. The patient is laying on the table with the chest uncovered, on which the doctor places a small microphone. The variation of the vibrations caused by the noise and the eventual murmurs are sent to the recorder. The exam lasts for about 10-20 minutes. The reading of the track is instantaneous, and the results are therefore immediately known. The exam doesn't involve any secondary effect.

The recording of hear sounds is a problem of vibrations measurement. That implies the necessity of a sensor, appropriate amplifier and filter, a memorization and visualization tool for the collected data. The useful band width is 20-100 Hz. The sensor must be a vibrations transducer (vibration pickup); an alternative is a stethoscope with a microphone, the electronic stethoscope. What is usually used is a sensor, a PC with a data acquisition card and a signal processing software [1].

Since the vibratory phenomenon of the chest wall is represented by a space-time kinematic function, one can act in two ways: sampling over time, as a set of images of the movement of the chest wall, or sampling in space, as a set of time signals obtained with multiple sensors. In addition to the temporal analysis, the one in the frequency domain is also very important. Spectral analysis of hear murmurs is able to detect differences in transvalvular pressure in patients with aortic valve stenosis, monitor the conditions of bioprosthetic valve, distinguish the components of the first two heart tones [1].

Figures from 10 to 18 show the temporal and spectral graphs obtained by PCG of some of the major valvulopathies.

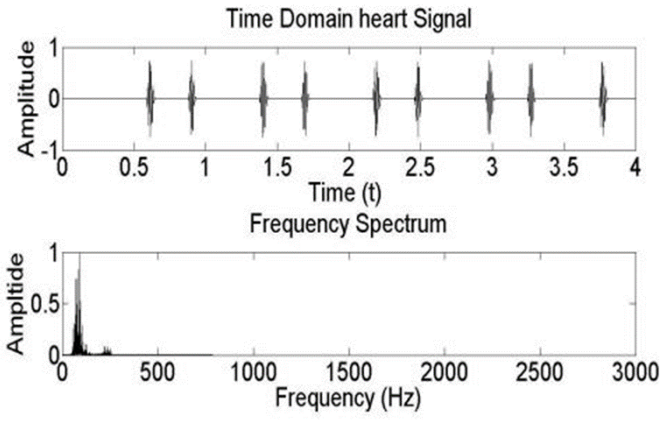

Figure 10-PGC of a normal heart signal (top) and its spectrum (bottom)

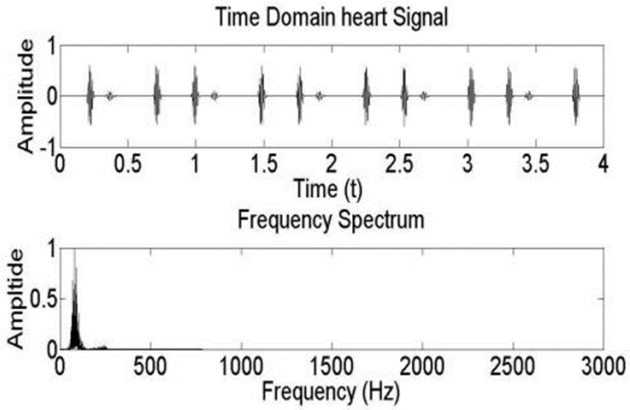

Figure 11-PCG of a heart signal with a third tone (top) and its spectrum (bottom)

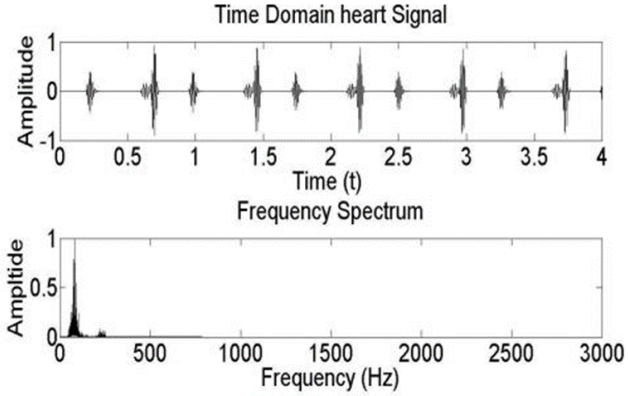

Figure 12-PCG of a heart tone with a fourth tone (top) and its spectrum (bottom)

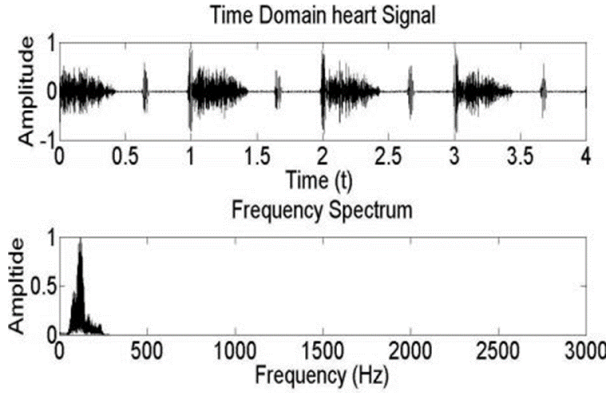

Figure 13-PCG of a heart signal with aortic insufficiency (top) and its spectrum (bottom)

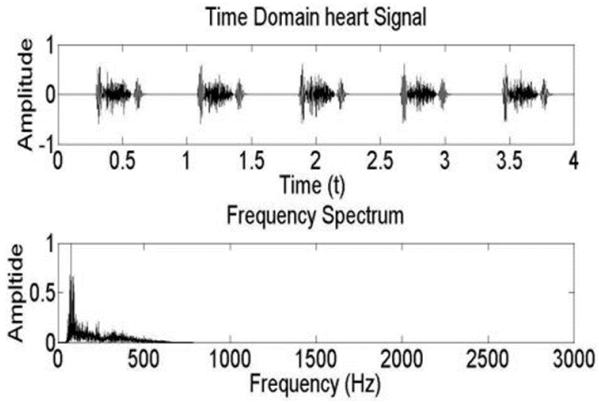

Figure 14-PCG of a heart tone with aortic stenosis (top) and its spectrum (bottom)

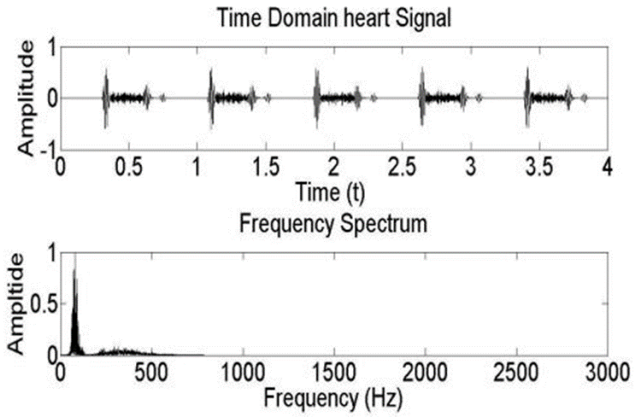

Figure 15.PCG of a heart signal with mitral insufficiency (top) and its spectrum (bottom)

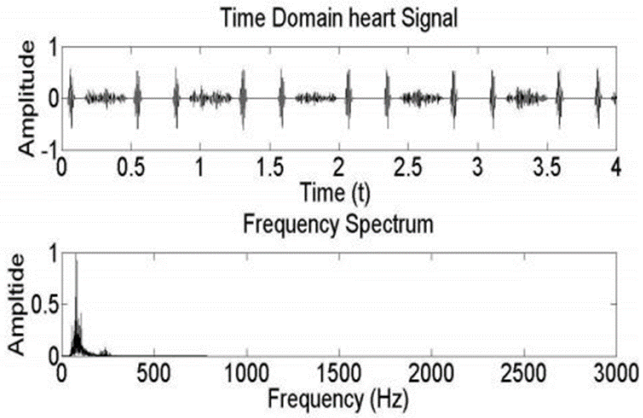

Figure 16-PCG of a heart signal with mitral stenosis (top) and its spectrum (bottom)

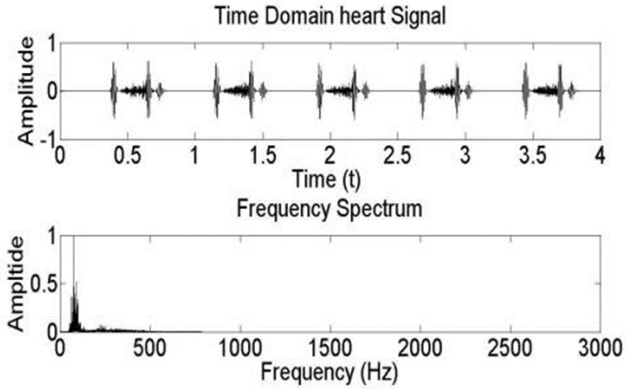

Figure 17-PCG of a heart signal with pulmonary stenosis (top) and its spectrum (bottom)

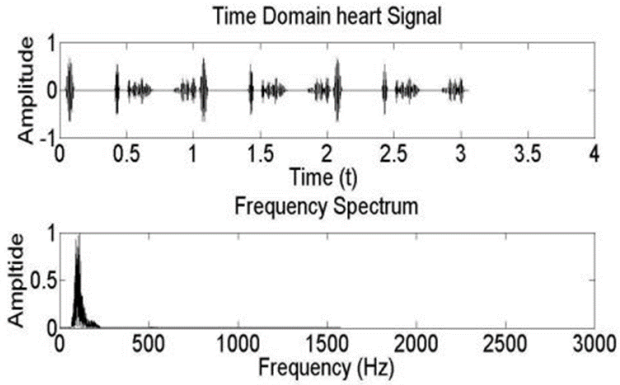

Figure 18-PCG of a heart signal with tricuspid stenosis (top) and its spectrum (bottom)

IV. DESCRIPTION OF THE GUI FOR PCG

In order to develop a new generation PC-based electronic stethoscope able to provide an automatic self-diagnosis of valvulopathies performing an advanced PCG, we have designed a GUI (Graphical User Interface) through which it would be possible to enter patient's personal data, proceed to acquire biological signal through audio devices (cabled or wireless) or by loading them from files and perform proper processing for automatic diagnosis.

A paper dealing with the signal processing algorithm for automatic diagnosis is in progress; in this paper we focus about the GUI, giving also an overview about data acquisition and first-processing algorithms implemented. A graphical user interface is a way for humans to interact with computer, made with windows, menus and icons that can be controlled by a mouse and a keyboard [3]. One of the benefits of using a GUI is that they make computer operations more intuitive and therefore easier to learn and use.

That is why a GUI can facilitate the acquisition and the analysis of patient's data also fostering a faster and more accurate diagnosis. The realization of this interface took place through GUIDE, a design environment provided by MATLAB. Once the layout was designed through the Layout Editor, its behaviour has been programmed. The Data Acquisition Toolbox was used to acquire data via hardware, thus allowing the connection between the device and MATLAB.

The interface can be opened by typing its name into the MATLAB prompt. The GUI is divided in sections according to the functions it performs, as shown in Figure 19.

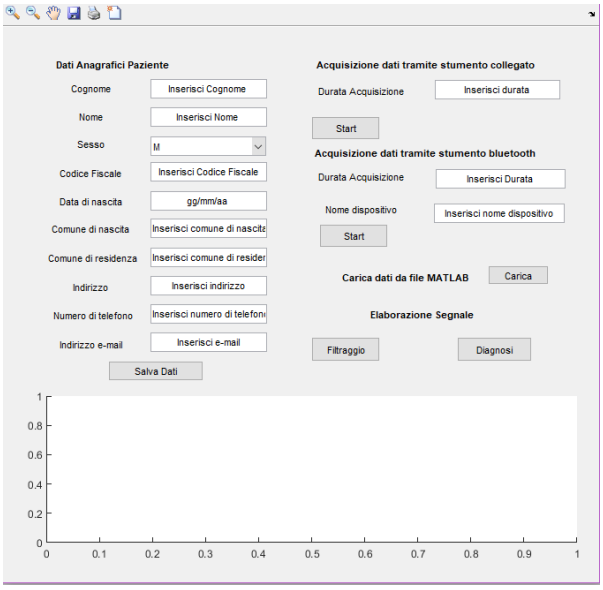

Figure 19-GUI main window

In the following, all sections are detailed with the relevant MATLAB code used.

4.1 Entering personal data

One of the sections of the interface is reserved for entering patient's personal data. It is made by 9 text boxes, 1 pop up menu and 1 push button to save the data. This section is highlighted in Figure 20 and zoomed in Figure 21

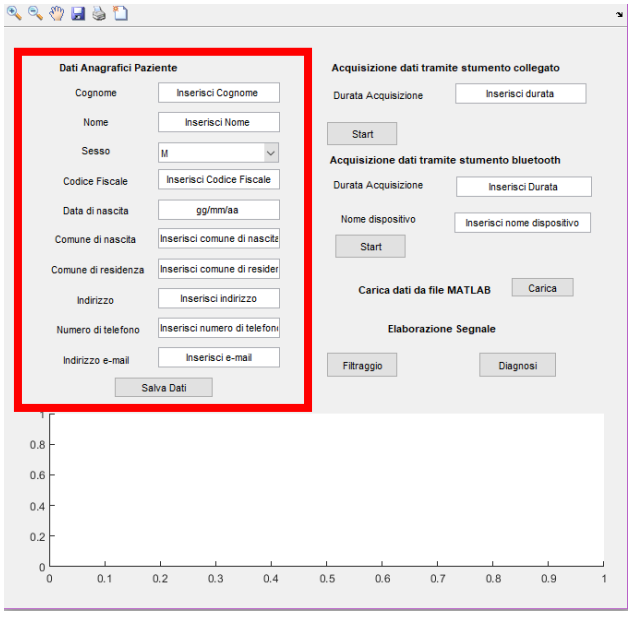

Figure 20-Section dedicated to data entry

The MATLAB call-back functions for the boxes indicated in Figure 21 are:

```
function cognome_Callback(hObject, eventdata, handles)
% hObject    handle to cognome (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
% handles    structure with handles and user data (see GUIDATA)
cognome_paziente=get(hObject,'String');
handles.cp=cognome_paziente;
guidata(hObject,handles);
```

The string entered by the user is saved in a variable (in this case "cognome_paziente") through the get() function. This variable is then added to the struct handles with the line handles.cp=cognome_paziente and save the change with guidata(hObject,handles), allowing the use of the variables saved, even outside of the functions in which they were created [4].

Figure 21 – Zoom of the GUI section for patient's data entry

Regarding the pop-up menu for selecting the gender of the patients, the callback function is:

```
% --- Executes on selection change in sesso.
function sesso_Callback(hObject, eventdata, handles)
% hObject    handle to sesso (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
% handles    structure with handles and user data (see GUIDATA)str = get(hObject, 'String');
str = get(hObject, 'String');
val = get(hObject,'Value');
% Saves the data depending on the choice of the user
switch str{val};
case 'M' % The user selects 'M'.
handles.sesso = 'Maschile';
```



```
case 'F' % The user selects 'F'
handles.sesso = 'Femminile';
end
guidata(hObject,handles)
```

In the variable str is saved the string ‘M’ or ‘F’ depending on the choice of the user, and in val either 1 per ‘M’ or ‘2’ for ‘F’. Through a switch struct, according to the gender selected by the user, in handles.sesso is saved the string “Maschile”(Male) or “Femminile” (Female).

The callback function for the box used to insert the fiscal code differs from the previous ones because, once called, must create two new folders:

```
function cod_fis_Callback(hObject, eventdata, handles)
% hObject    handle to cod_fis (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
% handles    structure with handles and user data (see GUIDATA)
cf_paziente=get(hObject,'String');
mkdir date;    %Creates the folder named as the date of the acquisition
[status,msg,msg_ID]=mkdir(date,cf_paziente);  %Creates the subfolder into the folder
named as the date
  % named as the fiscal code of the patient
If strcmp(msg_ID,'MATLAB:MKDIR:DirectoryExists')==1
      s2='(1)';
 %If the folder is already there, creates a new one
   s=strcat(cf_paziente,s2)
%with the same name and adding ‘(1)’
   mkdir(date,s);
   cf_paziente=s;
end
handles.cf=cf_paziente;
guidata(hObject,handles);
```

Once the fiscal code is added, besides saving it in the handles struct, a new path is created in the current one by making a new folder named as the date (using the function date), and a subfolder named as the fiscal code of the patient. In this new path will the saved all the files of the patients created by the interface.

To create the two folders, the command used is mkdir, in the case of the subfolder however, the output variables are status, msg and msg_ID. The reason why is that, in case of two patients registered in the same day that have the same fiscal code, we want to concatenate the string cf_paziente (that has the fiscal code of the patient) and ‘(1)’ , so that the data is not overwritten. In fact when the folder cannot be created because it is already present, the variable msg_ID contains the string 'MATLAB: MKDIR:DirectoryExists' and, through a conditional statement.

Once all the data is entered, it is saved by pressing the button “Salva Data”, which calls the function:

```
function salva_dati_Callback(hObject, eventdata, handles)
% hObject    handle to salva_dati (see GCBO)
```



```
% eventdata  reserved - to be defined in a future version of MATLAB
% handles    structure with handles and user data (see GUIDATA)
cf=handles.cf;
cognome_paziente=handles.cp;
nome_paziente=handles.np;
sesso_paziente=handles.sesso;
data_nascita=handles.dn;
comune_nascita=handles.cn;
comune_residenza=handles.cr;
indirizzo=handles.ind;
numero_tel=handles.nt;
email=handles.email;
%Creates, in the new path created, a text file .txt in which all the personal data of the patient
is saved
mypath=strcat(pwd,'\',date,'\',cf)
file=fullfile(mypath,'Dati_anagrafici_paziente.txt');
fileID=fopen(file,'wt');
fprintf(fileID,'Nome:              \t%s\n',nome_paziente);
fprintf(fileID,'Cognome:           \t %s \n',cognome_paziente);
fprintf(fileID,'Codice Fiscale:    \t %s \n',cf);
fprintf(fileID,'Sesso:             \t %s \n',sesso_paziente);
fprintf(fileID,'Data di nascita:   \t %s \n',data_nascita);
fprintf(fileID,'Comune di nascita: \t %s \n',comune_nascita);
fprintf(fileID,'Comune di residenza: \t %s \n',comune_residenza);
fprintf(fileID,'Indirizzo:         \t %s \n',indirizzo);
fprintf(fileID,'Numero di telefono: \t %s \n',numero_tel);
fprintf(fileID,'Email:             \t %s \n',email);
fclose(fileID);
```

This function creates a text file ('Dati_anagrafici_paziente.txt') in which all the personal data entered through the interface, is saved. The file is saved in the new path created.

4.2 Data acquisition

The section highlighted in Figure 22 is reserved for the data acquisition through a device (cabled or wireless) or through a MATLAB, mp3 or wave file.

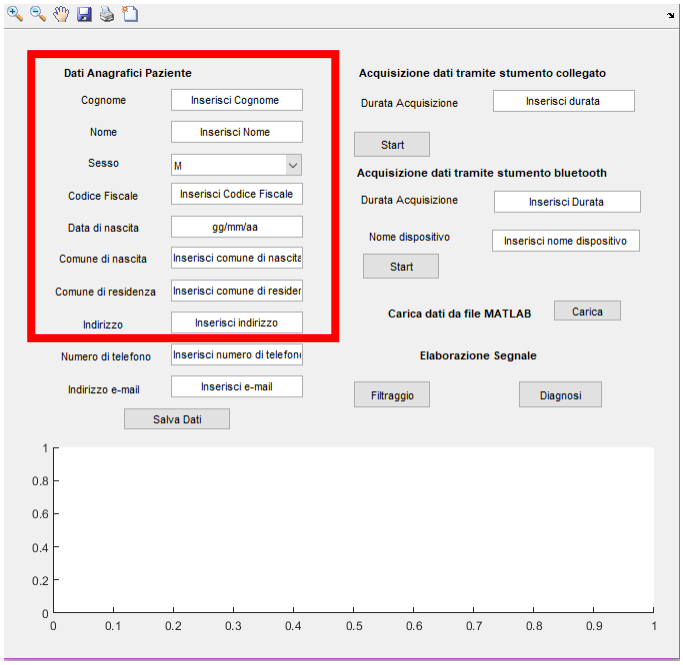

Figure 22-Section dedicated to data acquisition

In the section for the acquisition with a device, it is possible to enter the desired duration of the acquisition and a button to start it. In the section used for the acquisition with a wireless device, there is also a box to enter the name of the device.

The component used for both section for entering the duration in seconds of the acquisition is coded with this function:

```
function durata_Callback(hObject, eventdata, handles)
% hObject    handle to durata (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
% handles    structure with handles and user data (see GUIDATA)
t=str2num(get(hObject,'String'))
handles.t=t;
guidata(hObject,handles);
```

The string entered is turned into a numeric vector with the function str2num; and saved in the struct handles.

The “Start” button of the section reserved for the acquisition through a cabled device calls the following function:

```
% --- Executes on button press in pushbutton1.
function pushbutton1_Callback(hObject, eventdata, handles)
% hObject    handle to pushbutton1 (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
% handles    structure with handles and user data (see GUIDATA)
t=handles.t;
a = daq.createSession('directsound');
```



```
%Creates a session for the acquisition of data
addAudioInputChannel(a, 'Audio0', '1');
a.DurationInSeconds=t;
data=startForeground(a);
N=length(data);
Fs=44100;
%Sampling frequency
dt=1/Fs;
t1=dt*(0:(N-1))';
plot(t1,data,'r');
handles.data=data;
guidata(hObject,handles);
%Sets the path as the new one created previously
cf=handles.cf;
mypath=strcat(pwd,'\',date,'\',cf);
file=fullfile(mypath,'Dati_bin.dat');
%Saves the data in a binary file
fileID=fopen(file,'wt');
fwrite(fileID,data,'double');
fclose(fileID);
%Saves the data in an audio file
file=fullfile(mypath,'Registrazione_audio.wav');
audiowrite(file,data,44100);
%Saves the data in a MATLAB file
file=fullfile(mypath,'Dati_MATLAB.mat');
save(file,'data');
```

To acquire the data a session object is created for the audio board and an input channel with the lines a = daq.createSession('directsound') and addAudioInputChannel(a, 'Audio0', '1') [5].

Once the session is created and the channel is added, the duration of the acquisition is changed with a.DurationInSeconds=t where the variable t contains the value chosen by the user.

Now the data acquisition can start, and, once it's over, the data can be saved in a variable with the function data=startForeground(a) [5].

The acquired data is then saved, in the patient's personal folder, in three types of file:

- Binary ("Dati_bin.dat")
- MATLAB ("Dati_MATLAB.mat")
- Audio ("Registrazione_audio.wav")

Respectively with the functions fwrite(), save() ed audiowrite(). Once the name of the wireless device is entered, the function that is called is:

```
function nome_disp_bt_Callback(hObject, eventdata, handles)
% hObject    handle to nome_disp_bt (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
```



```
% handles    structure with handles and user data (see GUIDATA)
nome_disp_bt=get(hObject,'String');
BT=Bluetooth(nome_disp_bt,1);
ID=audiodevinfo(1,nome_disp_bt);
fopen(BT);
handles.ID=ID;
guidata(hObject,handles);
```

In these lines a Bluetooth object is created, so that it is possible to communicate with it and to acquire data. The callback function for the "Start" button of the section for the data acquisition with the wireless device is:

```
% --- Executes on button press in start_bt.
function start_bt_Callback(hObject, eventdata, handles)
% hObject    handle to start_bt (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
% handles    structure with handles and user data (see GUIDATA)
Fs=44100;                %Sampling frequency
nBits=64;                %Number of bits
ID=handles.ID
recorder=audiorecorder(Fs,nBits,1,ID);
%Sets the device specified as the audio input
t=handles.t
recordblocking(recorder,t);
%Records the audio for t seconds
data=getaudiodata(recorder);
%creates a vector which stores the values of the acquired signal
N=length(data);
dt=1/Fs;
t1=dt*(0:(N-1))';
plot(t1,data,'b');
handles.data=data;
guidata(hObject,handles);
%Sets the folder in which the data has to be saved
cf=handles.cf;
mypath=strcat(pwd,'\',date,'\',cf)
 %Saves the data in a binary file
file=fullfile(mypath,'Dati_bin_BT.dat');
fileID=fopen(file,'wt');
fwrite(fileID,data,'double');
fclose(fileID);
%Saves the data in an audio file
file=fullfile(mypath,'Registrazione_audio_BT.wav');
audiowrite(file,data,44100);
 %Saves the data in a MATLAB file
file=fullfile(mypath,'Dati_MATLAB_BT.mat');
save(file,'data');
```

The function sets the device specified as an input audio with audiorecorder(), stores the recorded audio in a vector call data and plots it.

As the previous case, the data is saved in three files:

- Binary (“Dati_bin_BT.dat”)
- MATLAB (“Dati_MATLAB_BT.mat”)
- Audio (“Registrazione_audio_BT.wav”)

The data can be acquired also by selecting a file pressing the button “Carica” that calls the following function

```
% --- Executes on button press in carica_dati.
function carica_dati_Callback(hObject, eventdata, handles)
% hObject    handle to carica_dati (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
% handles    structure with handles and user data (see GUIDATA)
[file,newpath]=uigetfile('*.mat;*.wav;*.mp3');
file=fullfile(newpath,file);
[newpath,name,ext]=fileparts(file)
if ext=='.mat'
   data=importdata(file);
else
  [data,Fs]=audioread(file);
end
Fs=44100;
N=length(data);
dt=1/Fs;
t1=dt*(0:(N-1))';
plot(t1,data,'b');
handles.data=data;
guidata(hObject,handles);
```

Once the button is pressed, with the execution of the function uigetfile(‘*mat;*wav;*mp3’) , a window is opened to select the MATLAB, mp3 or wave file as we can see in Figure 23.

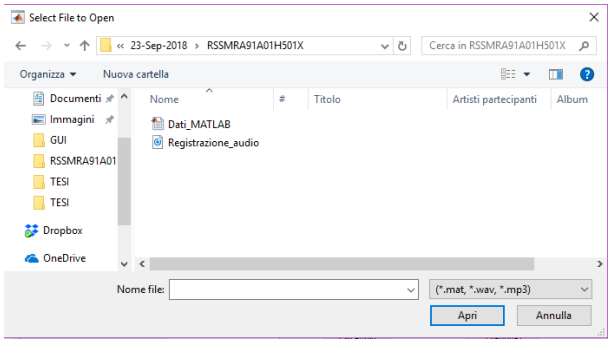

Figure 23-Interface shown to select the file to open

If the file selected by the user has mp3 or wave extension, the data is converted into a vector, ready to be processed.

4.3 Data pre-processing

Once the data has been acquired, it is possible to process it by pressing the two buttons “Filtraggio” and “Diagnosi”, shown in figure 24, which respectively perform a low pass filtering and a Fast Fourier Transform of the signal. These operations are pre-processing steps preceding further signal processing for the automatic diagnosis of valvulopathies.

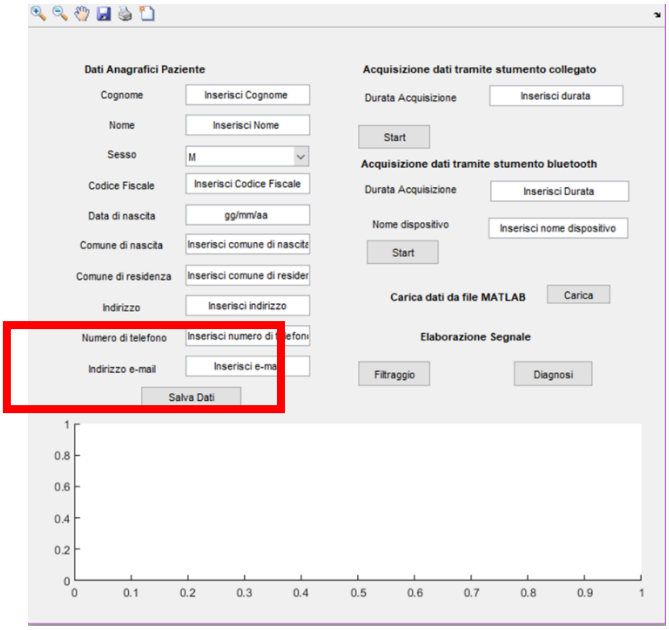

Figure 24-Section dedicated to data processing

The button “Filtraggio” calls back the function:

```
% --- Executes on button press in filtro.
function filtro_Callback(hObject, eventdata, handles)
% hObject    handle to filtro (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
```



```
% handles    structure with handles and user data (see GUIDATA)
%Low pass filtering
fc=20000;          %Cut off frequency
data=handles.data;
Fs=44100;
Wn = fc/(Fs/2);
[b,a] = butter(5,Wn,'low');
yb = filter(b,a,data);
N=length(data);
dt=1/Fs;
t1=dt*(0:(N-1))';
plot(t1,yb,'g');
cf=handles.cf;
mypath=strcat(pwd,'\',date,'\',cf)

%Saves the data in a binary file .dat
 file=fullfile(mypath,'Segnale_Filtrato.dat');
fileID=fopen(file,'wt');
fwrite(fileID,yb,'double');
fclose(fileID);
%Saves the data in a MATLAB file .mat
file=fullfile(mypath,'Segnale_Filtrato.mat');
save(file,'yb');
```

The function filters the signal saved in handles.data with a Butterworth filter; the line [b,a] = butter(5,Wn,'low') saves in b and a the Butterworth transfer function's coefficients having a normalized cutoff frequency equal to Wn, and with the line yb = filter(b,a,data) the signal is filtered and saved in yb.

The filtered signal is also saved into the patien's personal folder with 2 extention:

- "Segnale_filtrato.dat"
- "Segnale_filtrato.mat"

For the "Diagnosi" button che implemented function code is:

```
% --- Executes on button press in diagnosi.
function diagnosi_Callback(hObject, eventdata, handles)
% hObject    handle to diagnosi (see GCBO)
% eventdata  reserved - to be defined in a future version of MATLAB
% handles    structure with handles and user data (see GUIDATA)
%FFT of the signal
data=handles.data;
Y=fft(data);
Fs=44100;
N=length(data);
amp=abs(Y(1:floor(N/2)+1));
f=(0:N/2)*Fs/N;
```



```
figure;
plot(f,amp)
cf=handles.cf;
mypath=strcat(pwd,'\',date,'\',cf)
%Saves the data in a binary file .dat
file=fullfile(mypath,'FFT_segnale.dat');
fileID=fopen(file,'wt');
fwrite(fileID,amp,'double');
fclose(fileID);
%Saves the data in a MATLAB file .mat
file=fullfile(mypath,'FFT_segnale.mat');
save(file,'amp');
```

The function performs the Fast Fourier Transform (FFT) of the signal with the command fft() and plots the result.

It is also saved in:

- "FFT_segnale.dat"
- "FFT_segnale.mat"

V. TESTS AND RESULTS

In this paragraph it will be carried out a practical test of the operation of the interface, using an electronic stethoscope. Once the sensor is connected, we can open the interface by typing its name in the command window (Figure 25).

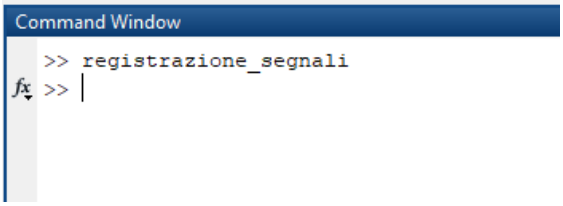

Figure 25-Command window

The window that will open is that showed in Figure 26:

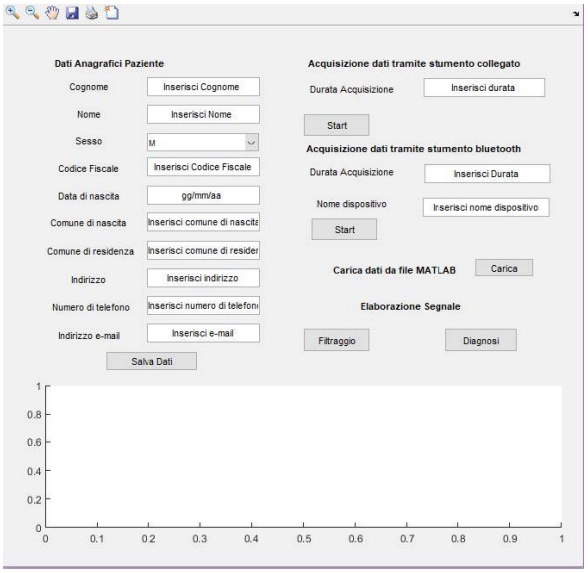

Figure 26-GUI

We can now proceed by entering the patient's personal data.

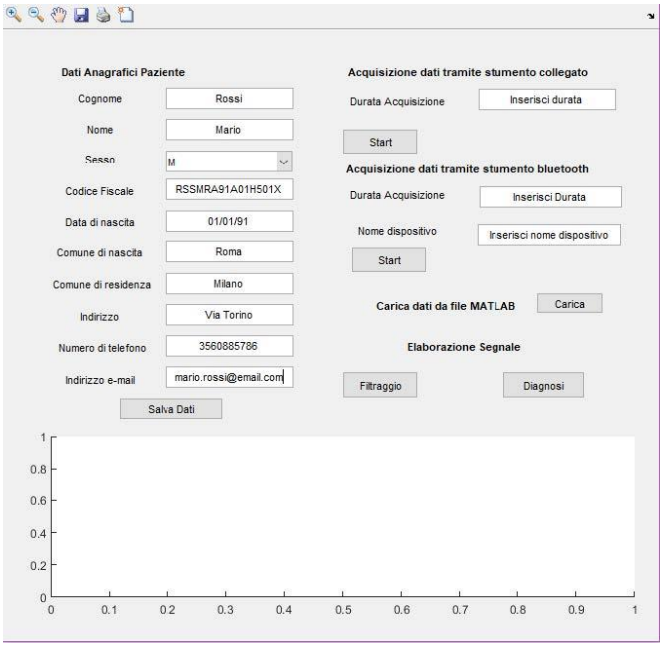

Figure 27-Entering personal data

Following the insertion of the patient's fiscal code, there is the creation of two folders as shown in Figure 28 and 29:

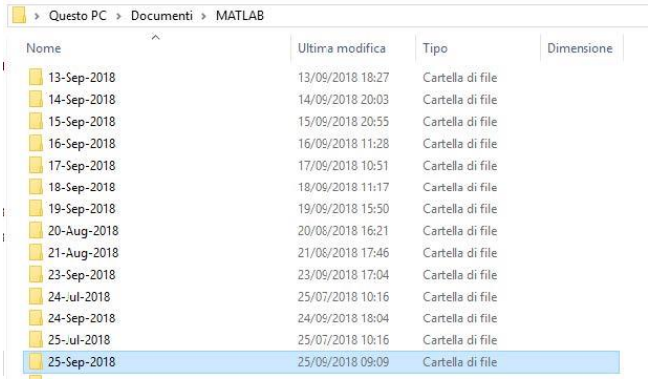

Figure 28-Recording date folder

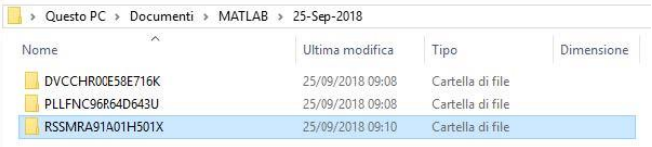

Figure 29-Patient's fiscal code folder

Once all data is entered and the "Salva dati" button is pressed, a text file containing them is created:

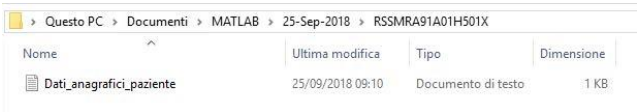

Figure 30-Personal data file

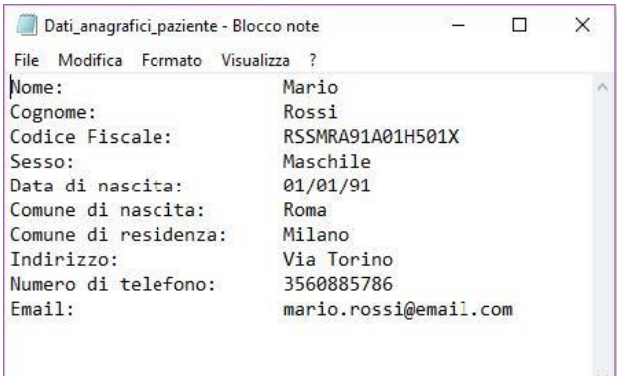

Figure 31-Patient's personal data

Let's now turn to data acquisition via electronic stethoscope. First, we enter the duration of the acquisition (for example 15 seconds):

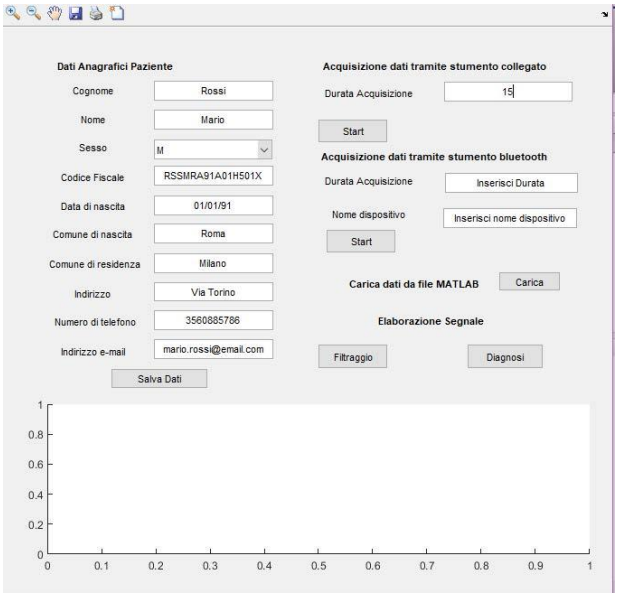

Figure 32-Entering the duration of the acquisition

Subsequently, by pressing the "Start" button, we can start the data acquisition, after which we can see the graphical representation through the interface itself like in Figure 33:

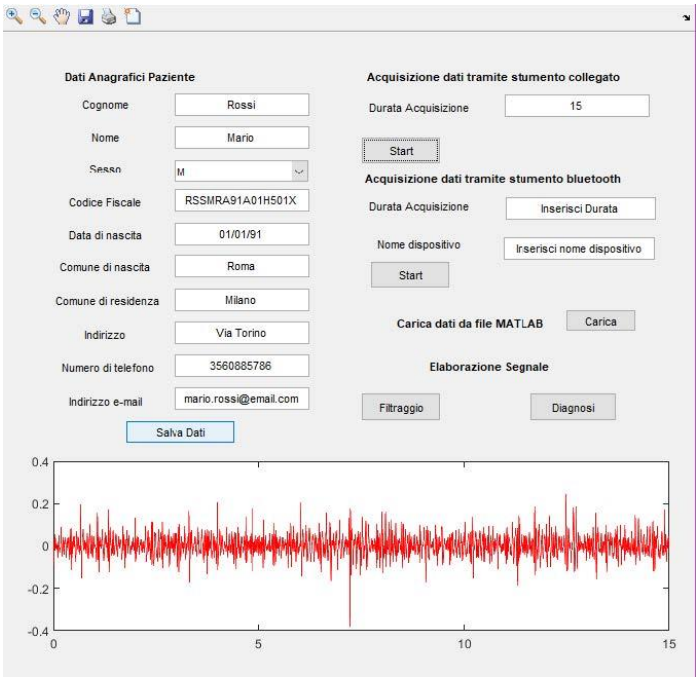

Figure 33-Graphical representation of the acquired data

In Figure 34 we can see that the data is also saved in binary, MATLAB and audio formats, so they can be processed or listened to later.

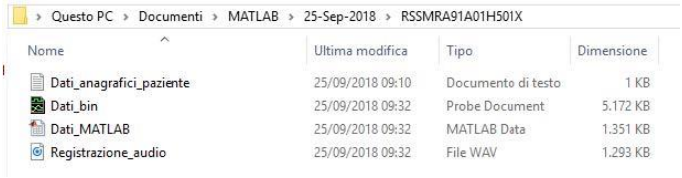

Figure 34-Saved acquired data

The recorded signal can now be processed through the two functions associated with the buttons "Filtraggio" and "Diagnosi"; we can see in Figure 35, what happens after pressing the first:

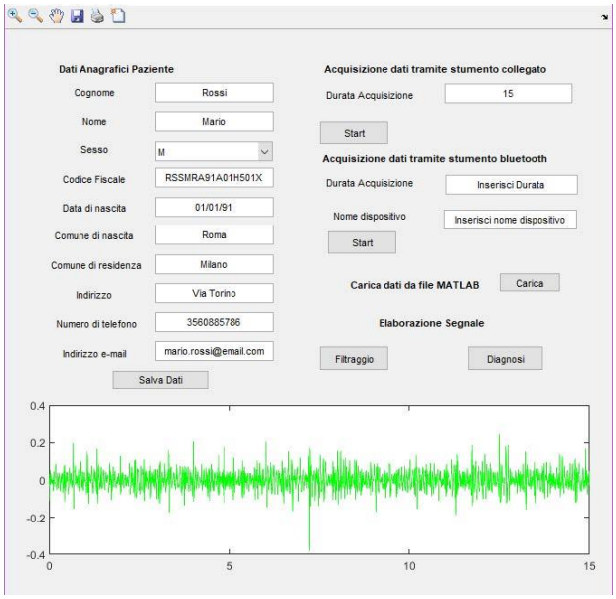

Figure 35-Graphical representation of the filtered data

The signal was then filtered correcly, the result is saved in the "Segnale_filtrato.dat" and "Segnale_filtrato.mat" files.

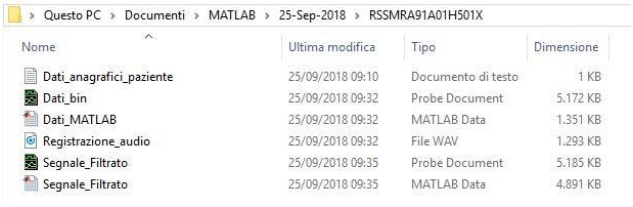

Figure 36-Filtered signal files

Processing the signal by pressing the "Dignosi" button, it is transformed through the Fourier Transform:

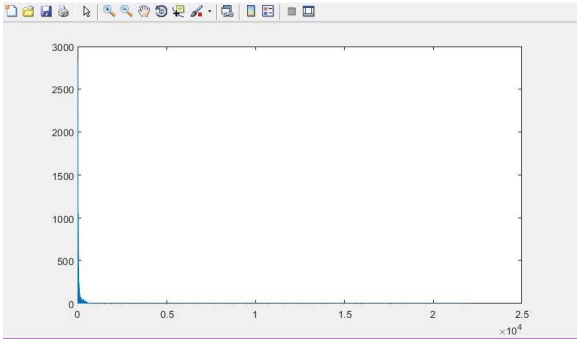

Figure 37-Signal FFT

Also in this case, the result is saved into the files:

Figure 38-Signal FFT files

The processing canalso be performed on previuosly recorded signals by loading them using the dedicated button; in fact it allows to select MATLAB, WAV or MP3 files:

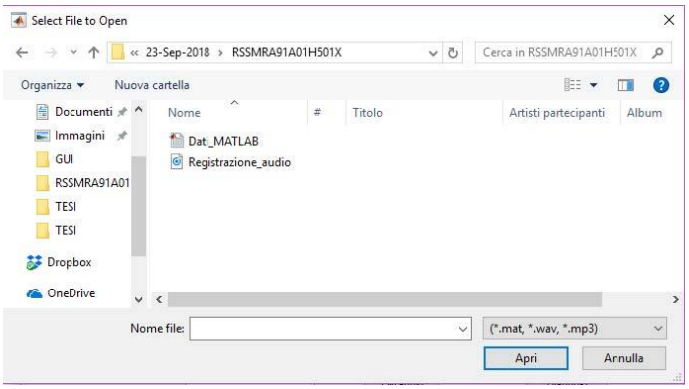

Figure 39-File selection to load

In Figure 40 we can see that once the data is load, its graphical representation will appear on the interface:

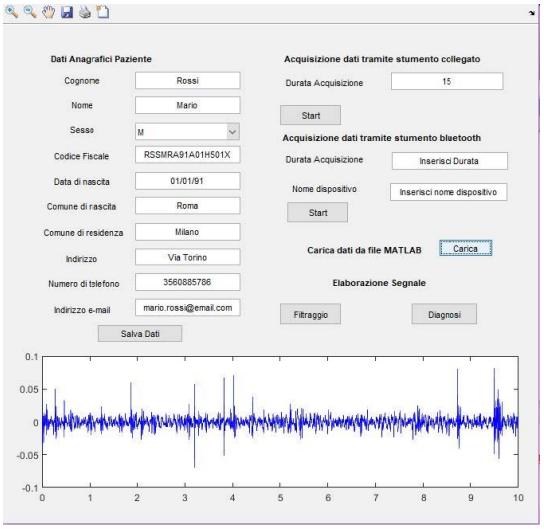

Figure 40-Graphical representation of the loaded data

VI. CONCLUSIONS AND FUTURE DEVELOPMENT

Conventional PCG is a simple but very effective medical examination performed by using only a stethoscope. However, PCG has lost interest due to a number of reasons. First, the vibratory signals are complex and therefore difficult to interpret; they are characterized by a wide range of frequencies with consequent various temporal representations. Getting high quality recordings with a high signal to noise ratio is difficult. Furthermore, the availability of technologies such as Doppler echocardiography and heart imaging techniques, which provide more direct and precise information on the functioning of the heart, has reduced the use of PCG. Finally, the diagnosis is strongly dependent on the physician capability and human hear sensibility to ear heart sounds and murmurs.

The auscultation, however, being simple, economic and not limited to hospital environments, has maintained its position as a diagnostic tool for the general physician and for the cardiologist. Therefore, an advanced PCG technique can be implemented by using electronic stethoscopes coupled with a PC with specific software for automatic diagnosis. Such an advanced PCG could become increasingly important avoiding for a first screening and diagnosis the use of more complex to use and expensive medical devices in order to detect heart valves pathologies.

Therefore, we have designed a MATLAB GUI useful for the acquisition and processing of heart sounds and murmurs on a PC in order to give an automatic diagnosis not affected by the limitations of the human ear and of the physician capabilities.

In this paper the GUI has been deeply described providing also the relevant MATLAB code, after having made a brief description of the heart and heart sounds and the main heart valve diseases. The results presented in this paper aim to inspire future works aimed at the classification of a greater number of cardiac anomalies and at the developing of a some-sort of smart electronic stethoscope. The developed GUI can be helpful in the sanitary field. In fact, the memorization of the acquired data, both biological and personal, can help the information exchange between medical staff.

The memorization happens in multiple formats, so that the data can be read by other type of device or processed later by the same interface. Other functions can be implemented in the interface, for example

to easily find fundamental parameters of the signal, depending on its origin or to have an automatic diagnosis of other cardiac pathology.

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