Development of ECG and EMG platform with IMU to eliminate the motion artifacts found in measurements

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The long term measurement and analysis of electrophysiological parameters is crucial for diagnosis of chronic diseases, and to monitor critical health parameters. It is also very important to monitor physical fitness improvement, or degradation level, of human beings where physical fitness is entirely critical for their work, or of more vulnerable members of society such as senior citizens and the sick. The state-of-the-art technological developments are leading to the use of artificial intelligence in the continuous monitoring and identification of life-threatening events in the daily life of ordinary people. However, these ambulatory measurements of electrophysiological parameters leads to drastic motion artifacts because of the test subject’s movements. Therefore, there is a dire need for the development of both hardware and software solutions to address this challenge.

The scope of this thesis is to develop a hardware platform, by using off-the-shelf discrete and IC electronic components, to measure two electrophysiological parameters, electrocardiogram (ECG) and electromyogram (EMG), with an additional motion sensor inertial measurement unit (IMU) comprising nine degrees of freedom. The ECG, EMG and IMU data will be collected using the developed measurement platform from various predefined day-to-day routine activity events. A bluetooth interface will be developed to transmit the data wirelessly, and record it on a laptop for further real-time processing. The resources of the electrical workshop and measurement lab at Aalto University will be used for the development, assembly, testing and finally for research of the measurement platform.

The second aspect of the study is to prepare, process and analyze the recorded ECG and EMG data by using MATLAB. Various filtering, denoising, processing and analysis algorithms will be developed and executed to extract the features of the ECG and EMG waveform structures. Finally, graphical representations will be made for the resulting outputs of the aforementioned techniques.

Keywords: ECG, EMG, IMU, Motion artifact
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Muhammad Tanweer
# Contents

Abstract ii

Contents iv

1 Introduction 1

2 Background 4

2.1 Human nervous system and electrophysiology ........................................ 4
  2.1.1 Neurons ........................................... 4
  2.1.2 Membrane potential .................................. 6
  2.1.3 Graded potential ...................................... 9
  2.1.4 Action potential ...................................... 11
  2.1.5 Synapses ........................................... 13
  2.2 EMG (Electromyography) .................................. 18
    2.2.1 History of EMG (Electromyography) ..................... 18
    2.2.2 Somatic nervous system and muscle physiology .......... 19
    2.2.3 EMG measurement techniques .............................. 24
    2.2.4 Recording of surface EMG ................................. 26
  2.3 ECG (Electrocardiography) ................................ 26
    2.3.1 History of Electrocardiography ......................... 27
    2.3.2 Autonomic Nervous system ................................ 28
    2.3.3 Cardiac anatomy ..................................... 30
    2.3.4 Cardiac electrical activity .............................. 32
    2.3.5 ECG wave recording ................................... 36
    2.3.6 Abnormal activity of heart .............................. 39

3 Electrophysiological signal processing 42

3.1 Sources of artifact and noise ........................................ 42
  3.1.1 Artifacts in EMG ....................................... 42
  3.1.2 Artifacts in ECG ....................................... 43
  3.1.3 Artifacts in ECG ....................................... 44
  3.1.4 ECG signal processing ................................... 48
  3.2 Electrode types ........................................... 50
    3.2.1 Non-polarizable electrodes (Conventional wet method) .... 51
    3.2.2 Polarizable electrodes (Dry method) .................... 51
    3.2.3 Capacitively coupled electrodes .......................... 51
  3.3 Applications ............................................. 52
    3.3.1 EMG applications ...................................... 52
    3.3.2 ECG applications ...................................... 53

4 Research materials and methods 55

4.1 Materials ................................................. 55
  4.1.1 ECG hardware platform development ........................ 55
  4.1.2 EMG hardware platform development ....................... 58
4.1.3 IMU hardware platform development ........................................ 61
4.1.4 Combined platform development ............................................. 62
4.2 Methods ................................................................................. 63
  4.2.1 ECG data collection ......................................................... 63
  4.2.2 EMG data collection ......................................................... 64
  4.2.3 Data recording ................................................................. 64
  4.2.4 Filters and algorithms ....................................................... 64

5 Results .................................................................................. 66
  5.1 ECG data processing ........................................................... 66
      5.1.1 ECG data detrending ..................................................... 66
      5.1.2 High frequency noise components removal ..................... 67
      5.1.3 Breathing motion artifact removal .................................. 67
      5.1.4 Sit-stand motion artifact removal .................................. 68
      5.1.5 Walking motion artifact removal .................................... 69
      5.1.6 QRS peaks threshold filter .......................................... 69
      5.1.7 QRS peaks detection ................................................... 70
  5.2 EMG data processing ........................................................... 73
      5.2.1 EMG data detrending ..................................................... 73
      5.2.2 Removal of high frequency noise components ................. 73
      5.2.3 EMG signal processing for event – weight lifting ............. 74
      5.2.4 EMG signal processing for event – heavy weight lifting .... 75

6 Summary ............................................................................... 84

7 Recommendations ................................................................ 86
### Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADC</td>
<td>Analog to digital converter</td>
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<tr>
<td>ANN</td>
<td>Artificial neural networks</td>
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<td>AV</td>
<td>Atrioventricular</td>
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<td>BWT</td>
<td>Bionic wavelet transform</td>
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<td>CC-ECG</td>
<td>Capacitively coupled electrocardiography</td>
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<td>CPU</td>
<td>Central Processing Unit</td>
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<td>cMAP</td>
<td>Compound muscle action potential</td>
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<td>CNS</td>
<td>Central nervous system</td>
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<td>CWT</td>
<td>Continuous wavelet transform</td>
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<td>DB</td>
<td>Daubechies function</td>
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<td>DOF</td>
<td>Degree of freedom</td>
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<td>DWT</td>
<td>Discrete wavelet transform</td>
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<td>ECG</td>
<td>Electrocardiography</td>
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<td>EEG</td>
<td>Electroencephalography</td>
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<td>EMD</td>
<td>Empirical mode decomposition</td>
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<td>EMG</td>
<td>Electromyography</td>
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<td>EPP</td>
<td>End plate potential</td>
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<td>EPSP</td>
<td>Excitatory postsynaptic potential</td>
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<td>FFT</td>
<td>Fast Fourier transform</td>
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<td>FIR</td>
<td>Finite impulse response</td>
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<td>HOS</td>
<td>Higher order statistics</td>
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<td>ICA</td>
<td>Independent component analysis</td>
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<td>IMU</td>
<td>Inertial measurement unit</td>
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<td>IPSP</td>
<td>Inhibitory postsynaptic potential</td>
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<td>ISM</td>
<td>Industrial scientific and medical</td>
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<td>LA</td>
<td>Left arm</td>
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<td>LMS</td>
<td>Least mean square</td>
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<td>MA</td>
<td>Motion artifact</td>
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<td>MUAP</td>
<td>Motor unit action potential</td>
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<td>NS</td>
<td>Nervous system</td>
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<td>PCB</td>
<td>Printed circuit board</td>
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<td>RA</td>
<td>Right arm</td>
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<td>RFI</td>
<td>Radio frequency interference</td>
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<td>Right leg</td>
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<td>RLD</td>
<td>Right leg drive</td>
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<td>SA</td>
<td>Sinoatrial</td>
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<td>sEMG</td>
<td>surface electromyography</td>
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<td>SG</td>
<td>Savitzky Golay</td>
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<td>SNAP</td>
<td>Sensory nerve action potential</td>
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<td>SR</td>
<td>Sarcoplasmic reticulum</td>
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<td>UDWV</td>
<td>Undecimated wavelet transform</td>
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<td>WA</td>
<td>Wavelet analysis</td>
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<td>White Gaussian noise</td>
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<td>WT</td>
<td>Wavelet transform</td>
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<td>WVD</td>
<td>Wigner ville distribution</td>
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1 Introduction

The use of electrophysiological parameters for clinical diagnosis both in animals and humans has already been in practice for more than a century. The research in this field has accelerated over the last two decades because of major improvements in technology. The electric potential activity of neurons and the ionic current in biological cells and tissues are studied in order to develop healthcare technology for living organisms. The recordings of such measurements – such as electrocardiography (ECG), electromyography (EMG) and electroencephalography (EEG) – are very commonly used for clinical healthcare these days.

Electrocardiograms (ECG) are recorded action potentials of millions of individual cells on the surface of the patient’s body due to an electric field established by cardiac beats. The electromotive activity of the heart produces a very weak signal (a couple of millivolts) on the body surface, which is enhanced by using a biomedical instrumentation amplifier [1]. This recording of cardiac electrical activity can be very helpful in identifying the health status of the patient if demonstrated properly [2]. There are several methods used to collect the ECG signals, and various computational methods are used to interpret the state of the patient’s health [3]. Figure 1.1 shows a simple signal-processing algorithm for ECG acquisition [6].

Figure 1.1: A simple ECG acquisition method [6].

Similarly, a neuromuscular activity produces electrical potential which is recorded as an electromyogram (EMG) [4]. Body muscles are made of well organised groups of muscle fibres which use a motor neuron to control the muscle force. The electromyogram (EMG) recordings can reveal significant information about the biomechanics of a patient’s body muscles, and is very useful to assess neuromuscular disorders [5]. Computerized computation of EMG has played a significant role in decision making and quantitative analysis in the field rehabilitation, neurophysiology and sports medicine. Figure 1.2 depicts a simple method for estimation of muscle activity level [4].
The physiological signals from a patient’s body always contain noise components and motion artifacts as well [7]. These extra unwanted signal components makes the situation challenging for physiologists to make proper diagnosis for ECG and EMG. There is a need to effectively remove such unwanted contaminants from the original ECG and EMG signals [8]. Several efforts has been made to remove noise and motion artifacts (MA) from ECG and EMG signals; however these unwanted contaminants still remain one of the major issues in cardiovascular and muscle activity monitoring which can lead to erroneous interpretation of signals in both long and short term recordings [9-11].

There are two major areas where improvements can be made to remove the motion artifacts. One is the hardware involved in sensing the signal where electrodes, signal filters, instrument-amplifiers, analog-to-digital converters – and various interfaces in the chain to the computing element – all have a role to play. The other is the application of innovative computational algorithms to effectively improve the signal quality, and to extract the required signal from the contaminated data set. The objective of this thesis is to design and develop a hardware platform indigenously to record the ECG and EMG data from the patient’s body. The recorded signals are then digitized and transmitted in a real-time environment to the computing unit for further algorithm application to remove the contaminants of the motion artifacts. Also an additional sensor unit is brought onto the same hardware platform for inertial measurement of the patient’s body movement which gives sufficient data for effective motion artifact detection.

The inertial measurement unit (IMU) data is used to remove the part of the signal significantly affected by the motion artifacts (MA). The focus of the thesis is on indigenously development of a multi-sensor hardware platform to record all significant information from the test-body so that the motion artifact (MA) contamination can be removed from the electrophysiological signals, specifically the electrocardiogram (ECG) and electromyogram (EMG). The second goal of the thesis is to apply some algorithms that have been developed precisely for the purpose of processing the signal for MA detection and removal.

The background of this thesis, given in chapter 2, discusses the basic concept, and reviews earlier developments in electrophysiological parameters, specifically ECG and EMG. Motion artifacts, and the different techniques used to improve the signal exaction, are discussed in chapter 3. The research material, the development of the necessary hardware platforms and methods used for this thesis are discussed in chapter 4. The results from data collected from use of the developed platform, and a standard ambulatory holter-monitor for ECG and EMG, are given, presented in chapter 5. A summary of the final assessment of the methodology used is presented,
given in chapter 6. Finally, chapter 7 summarises the recommendations and future developments within the context of this thesis.
2 Background

The human body has a tremendous capability to withstand quite variable environmental conditions, such as hilly regions, sandy deserts, dense cities, or sparsely populated remote areas. Our bodies are able to tolerate the hottest summers in Pakistan or the coldest winters in Finland. When a person jumps into a hot sauna directly from a hole in the ice, the homeostasis system of the body quickly adapts to the new environment by enhancing the blood circulation and sweating in response. In this chapter the basics of human anatomy and electrochemical activities at cellular level will be discussed in order to understand the specific working principles needed for the subject research on development of EMG (Electromyography) and ECG (Electrocardiography) systems using off-the-shelf components. An updated recent source on the principles of human physiology is used as reference [13] or indicated otherwise.

2.1 Human nervous system and electrophysiology

To understand the electromyography and its properties it is necessary to know how electric signals are produced in the human body, and how communication occurs throughout the body to accomplish the sensory and actuation tasks. The human nervous system is responsible for receiving signals from the sensory network, processing the information and making decisions, storing the information in memory, and also sending control commands to all actuators throughout the human body. Figure 2.1 shows a tree of the human nervous system.

The efferent branch of the peripheral nervous system deals with the control commands sent to the actuators in the whole body. The skeleton muscles are controlled by the somatic nervous system of efferent division and it is also called the voluntary control network. Some basic elements of the nervous system are defined below before discussing the working principles of human muscle physiology.

2.1.1 Neurons

The human nervous system has two types of basic functional units called neurons and glial cells. The neurons are excitable cells responsible for action potential generation and electrical signal transmission. The Glial cells are about 90% of the cells in the nervous system and are responsible for neural structure and metabolism activities. Figure 2.2 shows the structure of a neuron [13].

The neuron has a cell body and two neural activity components called dendrites and the axon. The neural cell body performs normal cell operations in a way similar to other body cells. However, dendrites are branches out of the cell body, and are the electric signal receivers from other neurons through a process called synapses. The axon is about a 1mm to 1m long branch which makes the nerve of the nervous system, and works as a transmitter to deliver electric signals to other neurons. Action potential is generated at the axon hillock and travels towards the axon terminal very rapidly. The terminal then releases neurotransmitters at the site of synapses.
Figure 2.1: Human nervous system.

Figure 2.2: Physiology of neuron cell [13].
to transfer the action potential to the postsynaptic neuron. The axon contains a microtubule which supports the quick transport of vesicles with the help of kinesin protein as shown in Figure 2.2.

2.1.2 Membrane potential

There are many ionic solutes throughout human body, and they are responsible for the flow of electric current. The positive ions and negative ions are equal in charge and make the overall body to be neutral. Different ions have different charges and concentration of ions inside and outside the cell membrane. The equilibrium potential of a specific ion can be calculated in mV by using the Nernst equation where $E_I$ is the equilibrium potential of ion (I), $I_o$ and $I_i$ are extracellular and intracellular ionic concentrations respectively, and $z$ is the valence of the ion:

$$E_I = \frac{61mV}{z} \log \frac{[I]_o}{[I]_i}$$  \hspace{1cm} (1)

By taking the concentration of sodium and potassium in extracellular and intracellular fluids, the equilibrium potential for both Sodium $E_{Na}$ and Potassium $E_K$ can be calculated by using the Nernst Equation:

$$E_{Na} = \frac{61}{1} \log \frac{145mM}{15mM} = 60.1mV \approx 60mV$$  \hspace{1cm} (2)

$$E_{Na} = \frac{61}{1} \log \frac{4mM}{140mM} = -94.2mV \approx -94mV$$  \hspace{1cm} (3)

But these ions are uneven in extracellular and intracellular fluids which leads to a potential difference. This potential difference is also called membrane potential because it brings opposite ions close to the membrane and can be measured in millivolts. The intracellular fluids contain more negative ions making the inside charge negative – which is why membrane potential is written with a negative sign and is represented with the symbol $V_m$. Figure 2.3 shows an example of membrane potential.

If the cell is not transmitting or receiving the electrical signals it is considered at rest and the potential of that state is known as resting membrane potential. It ranges from -5mV to -100mV for all body cells. The membrane potential in resting state depends on the concentration gradient of all the ions inside and outside of the membrane. It also depends on membrane permeability, as it is more permeable to the ion in concentration inside of the membrane. For sodium and potassium ions the potential difference is calculated by the Goldman Hodgkin Katz equation:

$$V_m = 61log \frac{P_{Na}[Na^+]+P_k[K^+]_o}{P_{Na}[Na^+]_i+P_k[K^+]_i}$$  \hspace{1cm} (4)

When the neural cell is at rest the number of potassium open channels are 25 times more than sodium open channels, making the membrane more permeable to potassium by setting the resting membrane potential of the neuron to about -70mV.
Figure 2.3: Opposite changes gathered on either sides of cell membrane [13].

\[ V_m = 61\log \left( \frac{(0.04)(145mM) + 4mM}{(0.04)(15mM) + 140mM} \right) = 61\log(0.0697) = -70.6mV \]  

The ions inside the membrane always tend to bring the membrane potential to their equilibrium potentials. When a neuron is in resting state both potassium and sodium are out of their equilibrium level. There is an electrochemical force to move the ions in or out of the membrane depending upon the state of resting potential. In neurons the potassium inside the cell tends to bring membrane potential to its equilibrium potential (-94 mV), and sodium tends to bring it to its own equilibrium potential (+60 mV). Therefore, the electrochemical force to bring more sodium ions inside the cell is way too large by having a potential difference of 130 mV from its own equilibrium potential. The flow of sodium ions towards the inside and potassium ions towards the outside will cause a current flow as defined here:

\[ I_{Na} = g_{Na}(V_m - E_{Na}) \]  

\[ I_K = g_K(V_m - E_K) \]

Where \( g \) is the conductance, \( V_m \) is the membrane potential and \( E \) is the equilibrium potential.

There are three types of ion channels in neurons which can open and close to change the electrical properties of a cell membrane. Leak channels are located everywhere...
on the cell membrane and are open. The channels for sodium and potassium are leak channels. They control the resting potential of the plasma membrane. Ligand-gated channels are controlled by chemical receptors in the membrane and are concentrated at the dendrites. They are responsible for receiving the action potential from the presynaptic neuron. Voltage-gated channels are controlled by membrane potential and are everywhere but densely located in the axon terminal. They are responsible for releasing the neurotransmitters packaged in the vesicles. Voltage-gated channels include potassium channels, sodium channels and calcium channels. These gated channels are controlled by particular stimuli. When they open or close they change the membrane permeability for that particular ion, and the inward or outward flow of that ion is enhanced.

During the exchange of ions across the membrane, the resting potential (-70 mV) of the neural membrane is disturbed. Even the resting state of the membrane is called the polarized membrane due to the potential difference on both sides of the membrane. If membrane potential goes more negative than the resting potential it is known as hyperpolarization of the membrane, and if it goes less negative or positive than the resting potential it is known as depolarization of the membrane. When the membrane returns to its resting state from either depolarization or hyperpolarization then it is known as repolarization of the membrane. Figure 2.4 shows all three types of polarizations and the resting state of the neuron cell membrane.

![Figure 2.4: All states of membrane potential [13].](image-url)
2.1.3 Graded potential

As discussed earlier, the gated channels open or close in response to stimuli (neurotransmitters) interacting with the receptors on the membrane. These small changes in the potential of the membrane are called graded potentials, and the strength of the graded potentials depend on the strength of the stimuli. If there is a large number of neurotransmitters interacting with the receptors, then the graded potential will be higher. For example, 10000 neurotransmitters on a gated channel will bring about 2 mV of depolarization to membrane potential. Graded potentials are decremental and lose their strength while traveling away from the site of stimulation because some of the ions leak through the membrane potential on the way. Figure 2.5 shows the transmission of such graded potential, and the loss of its strength, while traveling along the neural membrane.

![Figure 2.5: Neurotransmitter stimulation and strength of graded potential [13].](image)

Graded potentials can hyperpolarize the membrane, and such graded potentials are called inhibitory potentials. Similarly, they can also depolarize the membrane of the neuron and those are called the excitatory graded potentials. Figure 2.6 shows examples of both types of graded potentials. If graded potentials of the same type overlap each other, they can enhance excitatory condition, and if graded potentials of the opposite type overlap they cancel out the effect of each other.

A summation of two graded potentials happening at the same time from the same stimulus source is called temporal summation, and a summation to two graded
Figure 2.6: Hyperpolarization and depolarization of neuron membrane because of graded potentials [13].

Potentials happening at the same time from two different sources is known as spatial summation. Figure 2.7 shows an example of the temporal summation of two graded potentials when stimulation happens from a single source $W$. This depolarization summation leads the membrane potential to its threshold, and further to an action potential which will be discussed in the next section.

Figure 2.7: Temporal summation of graded potentials from same stimulus source [13].

Similarly, Figure 2.8 (a) shows a spatial summation of the same excitatory graded potentials from two different stimulation sources $W$ and $X$, which leads to membrane depolarization and to an action potential. Figure 2.8 (b) shows the summation of inhibitory and excitatory potential overlapping spatially and canceling out the effect.
of each other. There is no change in the overall membrane potential, and it stays in its resting state.

Figure 2.8: (a) Spatial summation of two excitatory graded potentials from different stimulation sources; (b) Spatial summation of an excitatory and an inhibitory graded potential from an independent source of stimulation [13].

2.1.4 Action potential

When a summation of graded potentials reaches the threshold, it leads to an action potential. For neurons the value of the threshold is -55 mV. At this threshold there happens a very fast depolarization of the cell membrane, and it reverses the polarity of membrane potential from negative to positive. The action potentials are generated in nerve cells, normal muscle cells and heart muscle cells. The action potential of nerve cells is discussed here, and the other two types of action potentials will be discussed in later sections of this chapter. During an action potential in neurons, the membrane potential changes from -70 mV to +30 mV. An action potential –
unlike graded potential – is more durable and travels very long distances without losing its strength. There are more leak channels of potassium in the membrane than sodium channels, making the membrane 24 time more permeable to potassium ions. The gated channels can lead to the action potential generation. Figure 2.9 shows an action potential of a neuron having the following phases:

1. **Depolarization:** During the first phase a very rapid depolarization happens and the membrane potential jumps from -70 mV to +30 mV. This happens due to a huge inflow of sodium ions, and shifts the membrane permeability to sodium as the potential has reached the equilibrium potential of sodium.

2. **Repolarization:** During the second phase the membrane permeability shifts back to potassium and the inflow of sodium is reduced rapidly. In this phase the membrane potential returns back to its resting state (-70 mV). It is known as repolarization of the membrane because the polarity is reversed back to negative.

3. **Resting potential:** After the repolarization, the concentration of potassium keeps growing for sometime which leads to hyperpolarization of the membrane closer to the equilibrium potential of potassium (-94 mV) as discussed in an earlier section. The leak channels then bring the potential slowly back to its resting state.

![Figure 2.9: Action potential of nerve cell membrane [13].](image)

The voltage gated sodium and potassium channels are found in the greatest concentration at the hillock of the neuron, and are then evenly distributed along the axon. The voltage gated channels for sodium has two gate types on each channel. One type are called activation gates and the other type are called inactivation gates. The mechanism behind their opening and closing is described in the following three phases, which are also shown in Figure 2.10.
1. When the membrane is at the resting state the inactivation gates of voltage gated sodium channels are open and activation gates are closed. Whenever a stimulus comes it opens the activation gates and allows the sodium flow inside.

2. A rapid inflow of sodium ions depolarizes the membrane and initiates the action potential. When depolarization is triggered a few sodium activation gates are open. The inflow of sodium triggers more activation gates to open regeneratively which causes very rapid depolarization.

3. After a short time – a matter of a millisecond – the saturation of sodium ions inside tends to close the inactivation gates. They remained closed during repolarization and the resting potential is gained. Another stimulation during this phase cannot open the inactivation gates again unless the membrane potential returns to its resting level (-70 mV). The leak channels of the sodium and potassium does the job to depolarize the membrane, keeping it stable in its resting level.

On behalf of potential strength, there are two types of stimulation happening in the neurons. Subthreshold stimulus is the stimulation which is not sufficient to depolarize the membrane upto threshold level. This stimulation opens a few sodium channels but is not enough to trigger the regenerative stimulation process. Suprathreshold stimulus, on the other hand, is stimulation which is sufficient to depolarize the membrane upto threshold and trigger action potential. No matter, how strong the suprathreshold stimulus is, it generates the same level of action potential. The nature of action potential creation of this all-or-nothing principle is shown in Figure 2.11.

The period when the membrane goes through repolarization – and until it retains its resting state – is called the refractory period. During this period a second stimulus cannot generate another action potential, and action potentials cannot overlap. After initiation of action potential at the axon of the neuron, it is propagated rapidly towards the axon terminal, and it does not lose its strength on the way. The propagation of action potential in the axon is shown in Figure 2.12 [13]. Once the action potential is initiated, it depolarizes the membrane region adjacent to the region of initiation. Hence, depolarization of one after another region propagates the action potential without losing the strength. Electromyography (EMG) is only recorded for myelinated nerve fibres which have a myelin sheath on the axon. The myelin sheath also works as insulation for the axon, and action potential propagates rapidly. The conduction velocity is proportional to the efficiency and length of the myelin sheath [19].

2.1.5 Synapses

In the human nervous system the action potentials are transferred from one neuron to another through a process called synapses. There are two types of synapse in the nervous system. In electrical synapses, electrical signals are passed by the flow of ions through the gap junctions between two neurons. Communication in electrical synapses is very fast and also bidirectional. The gap junctions between
communicating cells also offer a path for the second messengers to flow through. Some electrical synapses are always active and others are active only if some certain conditions are met. The electrical synapses happen in the brain stem and the cortex of eyes, and serve an effective role in communication.

On the other side, in chemical synapses stimuli (neurotransmitters) are released by one neuron to pass the signal to the other neuron. The neuron passing the signal is called a presynaptic neuron, and the neuron which receives the signal is called a postsynaptic neuron. These are the majority of synapses present throughout the human nervous system, and are discussed here in detail. There are many arrangements of pre- and postsynaptic neurons: if the axon of a presynaptic neuron meets the dendrite of a postsynaptic neuron it is called axodendritic synaptic; if the axon meets the cell body of a postsynaptic neuron it is called axosomatic synaptic, and; if the axon meets the axon of postsynaptic neuron it is called axoaxonic synaptic. The small space of 30-50 nm width, where chemical synapses happen is called a synaptic

Figure 2.10: The mechanism of activation and inactivation gates of voltage-gated sodium channel [13].
There are enzymes in an axon of a neuron, which are responsible for creation of neurotransmitters. These neurotransmitters are packed in small packets called synaptic vesicles. Once an action potential arrives at the axon, these neurotransmitters are released in the synaptic cleft to stimulate the receptors of a postsynaptic cell. Figure 2.13 shows the synaptic cleft of a presynaptic neuron at rest where voltage gated calcium channels are closed.

On the arrival of action potential at the axon of a presynaptic cell, the voltage gated calcium channels are triggered to open. The inflow of calcium increases the systolic calcium level which leads to the release of neurotransmitters from the synaptic vesicles in the synaptic cleft by an exocytosis process as shown stepwise in Figure 2.14. A higher frequency of action potential arrival opens the calcium channels for a longer period of time. The elevated level of systolic calcium causes the release of even more neurotransmitters. It takes only a couple of milliseconds to release the neurotransmitter after the arrival of an action potential.

The neurotransmitters in the synaptic cleft bind to the receptors of the postsynaptic neuron, and initiate the action potential in the postsynaptic neuron as well. The neurotransmitters in the cleft are actively transported back to the synaptic vesicles of the presynaptic neuron with the help of reuptake molecules. Other neurotransmitters are degraded by enzymes present in the postsynaptic neurons. This removal of neurotransmitters from the cleft prevents the continual stimulation reaction on the postsynaptic neuron. The communication signal is transmitted to the next neuron within 0.5-5 milliseconds, and this time span being referred to as synaptic delay.
If a synapse is strong enough to depolarize the postsynaptic neuron membrane to its threshold it is called an excitatory synapse, and leads to initiation of action potential in a postsynaptic neuron. The potential of the postsynaptic membrane in response to the excitatory synapse is called excitatory postsynaptic potential (EPSP). Similarly, if a synapse hyperpolarizes the membrane of a postsynaptic neuron instead of depolarization it is called an inhibitory synapse, and the potential that it generates in the membrane of postsynaptic neuron is called inhibitory postsynaptic potential (IPSP). Both EPSP and IPSP are graded potentials and can be summed if stimuli overlap. The summation can be temporal or spatial as discussed earlier in section.
Figure 2.13: Presynaptic neuron is at rest and waiting for action potential to arrive [13].

Figure 2.14: An active presynaptic neuron releasing the neurotransmitters in synaptic cleft [13].

2.1.3.
2.2  EMG (Electromyography)

Electromyography (EMG) is an electrical signal generated by neuromuscular actuation when a muscle contracts. This signal has enormous importance in the analysis of the anatomical and physiological properties of human muscles, the central nervous system which controls the muscle, and also the sensory network which brings feedback to the brain. Figure 2.15 shows the simplest form of an EMG signal read from a muscle [18]. A brief history of EMG development is presented in the next section, and in the following sections EMG-related human physiology, the somatic nervous system and its principles are discussed.

Figure 2.15: EMG Signal read from human body muscle [18].

2.2.1 History of EMG (Electromyography)

An Italian physicist and biologist Luigi Galvani, who had been teaching obstetrics in the University of Bologna in 1770s onwards, discovered “animal electricity” when he hanged the fresh body of a dead frog, on a copper hook with a steel wire during a thunderstorm, and noticed contraction of muscles. He observed that an electrostatic charge applied to a nerve provokes muscle contraction when his assistant touched the motor nerve with an electrostatically-charged scalpel [13,14]. In the 1840s Carlo Matteucci repeated Galvani’s experiment and endorsed his discovery. Later on the development of the galvanometer led to the detection of an electric signal running on the muscle surface during contraction, and this opened a new horizon in research into human electrophysiology as well [15].

In the 1850s Guillaume Benjamin Duchenne developed electrodes and simulation equipment which he used to find out that different muscles could be innervated by applying an electric signal to specific surface points. [15]. Later, the invention of the string galvanometer, the cathode ray tube, and other safety equipment, led electromyography (EMG) to clinical grade. The Nobel Prize winners Joseph Erlanger and William Osler discovered that the conduction velocity of nerve fibers depends on their physical diameter. In the twentieth century, Fritz Buchthal recorded the action potential of signal muscle fibre by developing microelectrodes. During second
World War, Martin Glover Larrabee was working at Framingham Hospital and, with his team, he measured compound muscle action potential (cMAP) from the muscle surface. In the meantime George Dawson developed signal averaging and photographic superimposition techniques, and recorded the sensory nerve action potentials (SNAPs) [15, 16].

In 1943 Edward Lambert developed the first electromyography (EMG) laboratory at Mayo Clinic in United States. Later on, Erik Stålberg investigated the electrical signal velocities through muscle fibres. He also found normative mean jitter values in different muscles which led to the electromyography (EMG) of a single fibre, and thus it became more practical to diagnose disorders in neuromuscular transmission. In 1948 three scientists, named James A. Fizzel, James Golseth and Herbert Jasper, developed the first commercial EMG machine for neuromuscular disorder diagnosis [17]. Figure 2.16 shows James Golseth demonstrating the first EMG machine for clinical purposes [15].

Figure 2.16: James Golseth demonstrating the first EMG machine available commercially [15].

2.2.2 Somatic nervous system and muscle physiology

The somatic nervous system controls only skeletal muscles with the help of only one type of neuron called motor neurons. The somatic nervous system is also known as
the voluntary nervous system because one can make a conscious decision to contract or relax a skeletal muscle. Muscle cells are called muscle fibres because of their physical fibrous shape, and these are innervated by a single motor neuron running from the spinal cord of the central nervous system (CNS) all the way to muscle fibres, and is capable of controlling several muscle fibres. A group of muscle fibres and the single motor neuron innervating them make a motor unit. Figure 2.17 shows how motor units are packed together in a muscle.

Figure 2.17: Motor neurons and groups of muscles forming motor units [13].

The axon of a motor neuron is called a terminal bouton and it lands on a muscle fibre at a very specific place called a motor-end-plate to make a neuromuscular junction. Only one type of neurotransmitters – called acetylcholine – are released in the junction to stimulate the nicotinic cholinergic receptors present in vast numbers on a motor-end-plate in the synaptic cleft. One action potential in a motor neuron stimulates all the muscle fibres connected to the neuron. A neuromuscular synapse is similar to a neuron-to-neuron synapse as discussed earlier. The synapse produces depolarization potential in the motor-end-plate, called end-plate-potential (EPP), which further leads to muscle contraction. Because there is only one motor neuron controlling the muscle fibre, and there is only one type of neurotransmitter, therefore the quantity of neurotransmitters in a synaptic cleft needs to be reduced for muscle relaxation.
The skeletal muscles are sometimes connected to bones from both sides with the help of elastic tissues called tendons, for example, biceps and triceps. Some muscles are connected to skin-like facial muscles, or to cartilage, like larynx, or to other muscles in the anal sphincter. The tendons are connected to the meaty part of the muscle which generates the force by contraction. The out-covering tissue of muscle is known as epimysium – having perimysium inside which is further divided into bundles called fascicles. Each fascicle consists of hundreds and sometimes thousands of muscle cells called muscle fibres. The membrane of muscle fibre is sarcolemma, which contains semifluid sarcoplasm packed with mitochondria and hundreds of rod-like banded elements called myofibrils as shown in Figure 2.18. Myofibrils are the main element to produce contraction force, and they are surrounded by a membranous structure, sarcoplasmic reticulum (SR), which works for storing calcium. SR has lateral sacs with transverse tubules (T tubules) running though to make a triad which serves an important role in action potential propagation. Figure 2.18 explains the overall structure of skeletal muscle fibre.

Figure 2.18: Structure of skeletal muscle fibre with terminal boutons of motor neuron [13].
The myofibrils are built up of a fundamental unit sarcomere, which repeats over and over throughout the length. It contains two types of the fibres in the longer axis, called thick filament, made up of myosin protein, and thin filaments made up of actin protein, as depicted in Figure 2.19. Actin monomers have myosin binding sites where thick filament binds and generates force. There is also a long fibre tropomyosin in the thin filaments which moves to either cover or expose the myosin-binding sites, and hence controls the contraction and relaxation. Thin filaments also have a complex of three proteins troponin, one binds the actin, the second binds the tropomyosin, and third has space for calcium binding. On the other hand, the thick filament consists of hundreds of myosin protruding bud-like structures found in pairs, and extends from long tails wrapped around each other. The bud structures of the thick filament are joined back to back from the tails and are called crossbridges which bind to the thin filament binding sites, and pull the thick filament inwards to produce the contraction force. Figure 2.19 shows the structure of thin and thick filaments, and also the sarcomere consisting of both. Thick filaments are connected to the z-line with an elastic protein titin, which can be stretched to three times of its length. The titin helps to relax the muscles when force is removed.

The action potential at the terminal bouton depolarizes the sarcolemma to its threshold level by releasing acetylcholine, triggering the action potential in a muscle cell. This action potential runs rapidly through the T tubules, and triggers the release of calcium from the SR stores. An increase of calcium in the cytosol triggers more calcium channels in the SR causing further release of calcium. Then calcium binds to the troponin complex and shifts the tropomyosin away from the actin binding sites, therefore triggering the crossbridge cycle, and ends up in the thick filament bud binding to the actin sites and pulling it to create the contraction force. The strength of this contraction force depends on the cytosolic calcium level. A continuous arrival of serial action potentials is required to maintain a force. If there is no action potential coming in, the calcium-pumps in the SR pump calcium back to its stores, hence dropping the cytosolic calcium level. Tropomyosin again covers the actin binding sites to stop the crossbridge cycle, and the muscle goes into relaxing state. The whole process is explained in Figure 2.20. Whenever a single action potential comes to a motor unit, a peak force is produced for a small fraction of time, and then falls to zero. This event is called twitch, which is a mechanical output of a motor unit produced in response to one action potential.

A single isolated twitch can only be produced in a laboratory. In practice, twitches exist in series, and overlap with twitches of other motor units in the same muscle. A twitch has three phases: the first is the latent delay of a few milliseconds between the stimulus time and contraction; the second phase is the contraction itself, which varies from 10 to 100 milliseconds depending upon muscle type, and; the third phase is relaxation. These phases of the twitch are shown in Figure 2.21. When a muscle generates a force greater than the opposing force such a twitch is known as isotonic contraction, and if the muscle generates a force but cannot contract because of a bigger opposing force, such a twitch is known as isometric contraction.

In order to increase the force of the overall muscle, the frequency of action potentials is increased to increase the twitches of a single motor unit. The brain also recruits
Figure 2.19: (a) Structure of thin filament. (b) Structure of thick filament. (c) Sarcomere structure consisting of thin and thick filaments [13].

more motor units present in the same muscle to enhance the force. Once all the
muscles are recruited that’s the maximum force one muscle can produce.

If an electrode is connected to the motor neuron, it picks up the action potential
traveling through it. The same action potential can also be measured at the terminal
bouton or directly from a depolarizing sarcolemma. However, performing such single
nerve measurements is neither practical nor feasible. For this reason, the compound
muscle action potentials (CMAPs) are measured for all motor nerves of a single
muscle. When a motor nerve is electrically stimulated at any point, the depolarization
wave travels in both directions, and a nerve conduction can be measured by using
the surface electrodes on the skin if the nerve is superficial enough. This method of
measuring electrical activity is more common and technically feasible [19]. Figure
2.22 shows how the action potentials of several motor units combine to make the
CMAPs, and finally EMG [20].
2.2.3 EMG measurement techniques

The measurement of EMG signals involves electronic equipment with electrodes to collect the CMAPs from a muscle. There are two techniques used for EMG measurement [21].

**Invasive measurement technique:** In this technique needle-type electrodes are inserted into the muscles to collect the EMG signal from the depolarizing membrane of the muscle fibres. This technique is however uncomfortable and painful for patients, and that’s why it is only used for clinical purposes. It does, however, also give more precise results for motor unit action potentials (MUAPs). Figure 2.23 shows how
the MUAP is recorded for four muscle fibres using the invasive technique [21].

**Non-invasive measurement technique:** In the noninvasive technique, electrodes are placed on the skin surface above the muscle, and the EMG signal is recorded as a gross activity of the muscle. This technique is widely used because it is not painful and is more comfortable for patients. The technique is also called surface EMG (sEMG) and it is the focus of this thesis. The spatial resolution is more limited in surface EMG than in the invasive technique, and also the MUAP high frequency parts are smoothed in surface EMG. However, it brings challenges in signal processing due to poor signal recording and more environmental noise.
2.2.4 Recording of surface EMG

The sEMG technique can measure the trains of the MUAP at low level contractions; however MUAPs of an individual nerve cannot be detected. Multichannels and the use of several electrodes make it possible to measure the generation and extinction of action potentials, and their conduction velocity in muscle. The sEMG is recorded at sampling rates lower than 500 Hz because the skin tissue acts as a low-pass filter while performing surface EMG recording [21].

In order to record the EMG signal electrodes, active, reference and ground, are used. An active electrode is placed directly at the entry of the motor nerve to collect the action potential of the motor neuron. A reference electrode is placed close to bone, and it is used to record the compound muscle action potentials. A ground electrode is usually larger in size of area to provide more provision for artifact rejection. If an external stimulator is used then the ground electrode is placed in between the recording and stimulator electrodes [20].

2.3 ECG (Electrocardiography)

The human heart beats about seventy times per minute on average when the body is at rest. The cardiac beat increases during activity because the heart is responsible for supplying all crucial nutrients and oxygen by propelling blood to all organs of the body. Electrocardiography (ECG) is the recording to the heart’s electrical activity. This electrical activity makes the heart beat in a rhythmic way to pump the blood into the whole body. A human heart normally pumps blood by contracting for three billion times in its average life. A brief history of ECG is discussed in the next section, and the anatomy of heart with the autonomic nervous system are both discussed in
2.3.1 History of Electrocardiography

After the discovery of animal electricity in a frog by an Italian anatomist Luigi Galvani, as discussed in an earlier section, an era of research and development was triggered in the field of medicine with connection to electricity. Charles Kite won a Silver Medal for his article on electricity used for diagnosis of a human who had recently died as a result of drowning. He concluded that electricity is a wonderful tool for finding out whether a person is dead or not [25].

In the mid nineteenth century Hoffa experimented on the hearts of cats and dogs by applying a strong electric current, and concluded that fibrillation can be induced by a single electric pulse. Rudolph von Kolliker and Heinrich Muller measured the electric current pulses from an exposed ventricle using a galvanometer. He observed the twitch of a cardiac muscle before and after cardiac systole. Later on, these twitches were recognised as QRS complex and T waves in an ECG signal. In 1878 these two waves were verified by John Burdon Sanderson and Frederick Page when they recorded the electric current of a frog [26]. In 1887 a British physiologist Augustus Waller recorded for the first time human ECG by using a capillary electrometer [27]. In 1891 William Bayliss and Edward Starling measured three phases of human ECG from a single cardiac beat by placing electrodes on the right hand and the apex. These phases were recognised as the P, QRS and T waves of ECG.

In 1893 a Dutch physiologist Willem Einthoven introduced the electrocardiogram term for the very first time, and he also used PQRST alphabets to describe the ECG waveform of a single beat [28]. He also invented a string galvanometer and produced it on a commercial scale, which was more sensitive than earlier versions. Figure 2.24 shows a galvanometer used to record ECG of a human subject [28]. In 1912 Einthoven introduced his equilateral triangle formation of standard leads I, II and III. He was awarded the Nobel Prize in 1924 for his work on ECG.

In 1931 Alvery Hyman patented the first artificial cardiac pacemaker with the aim of fitting the pacemaker in a doctor’s bag. It was used on 43 patients, continuing to function successfully for 14 cases [29]. In 1934 Fran Wilson introduced one indifferent electrode by placing electrodes on the right arm (VR), left arm (VL) and left foot (VF) with 5000 ohm resistors. Later on, this electrode was known as “Wilson’s central-terminal”, and a combined lead acted as the ground terminal [30]. In 1938 the British Cardiac Society and American Heart Association standardized the position of the chest leads from V1 to V6. Later Emanuel Goldberger increased the voltages of Wilson’s leads by 50%, and introduced augmented limb leads which he referred to as aVR, aVL, aVF. Wilson’s three leads, Einthoven’s three leads, and standard 6 chest leads were all combined to form 12-leads ECG, which is still in practice today. In 1949 Norman Jeff Holter invented the first ambulatory ECG monitoring device, which was named the Holter Monitor after him [31]. There has been a gradual development in the ECG recording equipment and further elaboration of recordings to diagnose different disorders and diseases related to the heart. With the introduction of wireless technology in 1999, the first ECG signal was transmitted and received wirelessly,
which was good enough to be interpreted by cardiologists. In 2005 the clinicians made a decision on the wirelessly-received ECG signals of a patient which were transmitted from an ambulance [32].

2.3.2 Autonomic Nervous system

The autonomic nervous system in human beings is responsible for controlling all the smooth muscles, cardiac muscles, visceral organs, glands and adipose tissue at subconscious level; that is why it is also called the involuntary system. The autonomic system has two branches which innervate the same organs but in the opposite way.

The parasympathetic nervous system is active when the body is at rest, and this condition is referred to as “rest and digest” because the digestive system is active in this phase. On the contrary the sympathetic nervous system is most active when the body is physically active, and this condition is called “fight or flight” because the body is active to respond to any threatening situation. The autonomic system regulates the push-and-pull control mechanism of the effector organs for both the sympathetic and parasympathetic systems. The autonomic nervous system (NS) involves the ganglionic network which has preganglionic neurons and postganglionic neurons. A single preganglionic neuron interacts with multiple postganglionic neurons through synapses. Figure 2.25 shows the ganglionic network and its components in the autonomic nervous system.

A neurotransmitter acetylcholine is released by the preganglionic neurons of both sympathetic and parasympathetic branches, and also by the parasympathetic postganglionic neurons. Norepinephrine is released by the sympathetic postganglionic
neurons. During a neural synapse norepinephrine and acetylcholine bind to different types of receptors. The muscarinic cholinergic receptors are found on effector organs such as the heart, the smooth muscles of the eye pupil, and the smooth muscles in the digestive tract. Both sodium and potassium channels are operated by the acetylcholine receptors. Figure 2.26 shows the neurotransmitters and receptors in the autonomic nervous system. On stimulation the sodium moves in faster than potassium is able to move out, hence leading to the depolarization of the postsynaptic neuronal membrane. On the other hand, muscarinic cholinergic receptors are linked to G proteins to produce second messengers.

The site where the efferent neuron synapses the effector organ is called the neuroeffector junction, and this synapse is different from the neuron-to-neuron synapse because of a unique postganglionic axon which has bud-like structure called viscosities. The voltage-gated calcium channels at the viscosity end are operated on arrival of the action potential, and the increase in systolic calcium triggers the release of neurotransmitters stored in vesicles by an exocytosis process. The distance between the viscosities and the effector organs is bigger than the synaptic cleft so it disperses the neurotransmitters over a larger area. Figure 2.27 shows the viscosities of the postganglionic neuron and the neuroeffector junction during action potential.

The autonomic nervous system is regulated by the hypothalamus, pons and medulla oblongata. The fight-to-flight mode of the sympathetic nervous system is controlled by the hypothalamus which controls the temperature, food intake and water balance as well. The heart beat rate, blood vessels and the smooth muscles of the respiratory system are controlled by the medulla oblongata and pons.

The emotions play a crucial role in the control of the autonomic nervous system and the organs controlled by it. The emotional response of the autonomic nervous system could be a racing heartbeat, an upset stomach, blushing, fainting or sweating.
2.3.3 Cardiac anatomy

The system responsible for providing the required energy and oxygen to every cell of the body, and also to remove carbon dioxide and waste products from them, is called the cardiovascular system. It contains three components: (i) a muscular pump, the heart, which pumps blood through vessels, (ii) blood vessels, the conduits which carry blood, and blood, a fluid which carries all the material to and from the body cells. The heart also provides endocrine and sensory functions to regulate the blood volume and pressure. The heart is a muscular organ which generates the force to propel the blood, and contains two upper chambers, called atria, and two lower chambers, called ventricles. Figure 2.28 shows a cross section of the human heart and all its components.

The heart can be divided into a pair of pumps separated vertically by a layer called the septum. Each pump contains an atrium and a ventricular. The right part deals with deoxygenated blood and the left part deals with oxygenated blood. The whole network of blood vessels in the human body is called the vasculature which is a closed loop network. It starts from large vessels known as arteries taking blood out of the heart. Then those arteries divide into smaller vessels called arterioles, which are further divided into the smallest vessels called capillaries. The exchange of nutrients, gases and hormones happen at the capillary level. These capillaries then join up together to form the bigger vessels called venules and which are joined to form the biggest vessels veins bringing the blood back to the heart.

The cardiovascular system consists of two blood circulation circuits. The Pulmonary circuit involves the lungs which exchange the gases in the blood. The pulmonary circuit takes deoxygenated blood from the right ventricular through pulmonary arteries and passes through the lung, and brings back the oxygenated blood through
Figure 2.27: Neurotransmitters release on arrival of action potential at viscosities of the postganglionic neuron [13].

pulmonary veins to the left atrium of the heart. The systemic circuit stake oxygenated blood from the left ventricular, and supplies it to whole body through arteries and arterioles. It brings deoxygenated blood back from the whole body through veins to the right atrium. The heart nourishes itself from blood that passes through the coronary arteries.

The heart sits in the thoracic cavity separated from the abdominal cavity by the diaphragm. A thin membrane called the pericardium surrounds the heart and lubricates the heart with pericardial fluid. The walls of the heart are made of three layers: (i) the outermost layer, made of connective tissues, is called epicardium; (ii) a middle layer made of cardiac muscles which is called myocardium, and; (iii) the innermost layer made of epithelial cells which is called the endocardium. The myocardium is mainly responsible for the rhythmic contractions which squeeze the blood out of chambers with high pressure, and myocardial relaxation lets the blood flow into the chamber. The myocardium of the ventricles is thicker than that of the atria, and specially, the left ventricular myocardium is thickest because it is
Figure 2.28: Cross-section of the human heart and its components [13].

The blood circulation in the heart is unidirectional, being maintained by four valves in the heart. The atrioventricular valves separate the atria and ventricle, and prevent the backflow of blood. These valves open and close passively due to the pressure difference created by the heart beats. The AV valves open due to higher atrial pressure during atrial contraction, and close when the ventricles contract to make pressure in the ventricles higher than in the atria. Two other valves, called semilunar valves, separate the ventricle and arteries. These also ensure that the backflow of blood into the ventricle is inhibited when the ventricles relax to fill with blood from the atria.

### 2.3.4 Cardiac electrical activity

The atria contract together, followed by the contractions of both ventricles at the same time. The cardiac conduction system makes the cardiac muscles contract and relax in a very sequential and synchronised way. These contractions are triggered by a signal originated within the heart itself instead of the CNS which is the reason the activity is called myogenic. However, the autonomic nervous system can slow down – or speed up – the heart rate according to the requirements of the body. The autorhythmic cells are very special cardiac cells which do not generate any contractile force, but are responsible for generating and propagating the rhythm for heartbeats. This ability of the heart to generate its own rhythm is called autorhythmicity. There are two types of autorhythmic cells which make the conduction system of the heart:
(i) the cells responsible for action potential generation and beat rhythm are called pacemaker cells; (ii) the other cells are responsible for transmitting the electric signals in highly organized way called conduction fibres. Figure 2.29 shows the conduction system of the heart.

![Conduction system of the human heart][1]

The pacemaker cells are found in concentration at two locations in the myocardium: (i) the sinoatrial node (SA node) is located in the right atrium close to the vena cava; and (ii) the atrioventricular node (AV node) is located in the interatrial septum close to the tricuspid valve. Both nodes generate the action potential at a slightly different rate. The action potential generation is faster at the SA node (70 pulses per minute) than the AV node (50 pulses per minute), so the SA node drives the AV node and defines the heart rate. If somehow the SA node fails to generate the action potential then the AV node will take over the control, and if the AV node fails, then the bundle of His (30-40 pulses per minute) takes the control of generating action potential – which, although having the lowest rate compared with the SA and AV nodes, it is still, nonetheless, able to cope with an emergency situation. The conduction fibres of the heart are bigger in diameter and can conduct action potential at speed of 4 m/s higher than ordinary cells. Once the action potential at the SA node is initiated, it travels rapidly and depolarizes the atria, causing contraction. Then it travels to bundle of His Purkinje fibres to depolarize the ventricles, and causing contraction.

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[1]: Conduction system of the human heart [13]
there. The signal is a little delayed while travelling from the SA node to the AV node. This delay gives the ventricles enough time to fill up. All contractile cells are interconnected through gap junctions which make it possible for ions to flow fast. The intercalated disk keeps the cells attached firmly to withstand mechanical stress.

**Electrical activity of pacemaker cells:** The pacemaker cells are very special and different because they can generate and propagate action potential periodically on their own without any external stimulation. Pacemaker cells do not have any resting potential, and they keep depolarizing slowly after repolarization until they reach the threshold to trigger the action potential. Figure 2.30 shows the action potential generated by pacemaker cells.

![Action potentials fired by pacemaker cells of the human heart](image)

Figure 2.30: Action potentials fired by pacemaker cells of the human heart [13].

The permeability of the cardiac cell membrane depends on sodium, potassium and calcium ions. The cytosol is concentrated with potassium ions but lacks calcium and sodium ions. Sodium is concentrated outside the membrane and tends to move in. The equilibrium potential is negative for potassium ($E_K = -94 \text{ mV}$), and is positive for sodium ($E_{Na} = +60 \text{ mV}$) and calcium ($E_{Ca} = +130 \text{ mV}$). The enhanced value of sodium and calcium tends to make the membrane potential more positive, and an increase in potassium tends to make it more negative. Figure 2.30 depicts the slow depolarization due to the closing of potassium channels (which were opened during the repolarization phase) and the opening of sodium funny channels. They are called funny channels because of their unique operational behaviour. This brings a decrease in potassium outflow and an increase in sodium inflow, which depolarizes the membrane slowly towards the threshold of -55 mV. When the potential is about -60 mV, the transient-type calcium channel are opened for a short time to trigger the action potential, and this happens due to the opening of the voltage-gated long-lasting calcium channels. This increased inflow of calcium leads to rapid depolarization, resulting in an upswing of action potential. The action potential then triggers the potassium channels to be open to bringing in the potassium ion and pulling the membrane potential back down. The rapid fall in potential closes the calcium
channels. The continuous increase in potassium ions inflow and, the decrease in calcium level, repolarizes the membrane fully and ends the action potential.

The whole process of slow depolarization, rapid depolarization and repolarization repeats periodically to generate the action potentials continuously without any neural input. However, the autonomic nervous system controls the rate of action potential to a certain extent. This action potential is then fed to the contractile cells of cardiac tissue present in the myocardium. In the next section the electrical activity in contractile cells is discussed.

**Electrical activity in contractile fibres:** In resting position, the contractile myocardial fibres are polarized by possessing a negative charge inside and a positive charge outside. During depolarization, these charges are inverted with a stimulus, and propagation of depolarization happen from one fibre to other adjacent tissues. Similarly, a repolarization wave again inverts the charges of the fibres by making them negative inside and positive outside. The depolarization wavefront is denoted by a vector with a positive charge at the tip, and, similarly, repolarization wavefront vector has negative charges at its tip [2]. The pacemaker cells and contractile cells of the myocardium are joined together with gap junctions, which transfer the action potential quickly. The action potential in contractile cells is different to the action potential of pacemaker cells. Also the action potential in contractile cells is not the same in different fibres depending upon the size and shape of the fibre, location, and speed of propagation. However, the mechanism of action potential generation is similar in all of them. The action potential in ventricular muscle cells is the most dominant; therefore it is discussed in detail here to explain the electrical activity of contractile fibres. The action potential goes through five phases 0-4 which are depicted in Figure 2.31 [12].

1. **Phase 0** a huge change in action potential from resting potential, going from -90mV to +30mV, is observed. It happens due to the opening of the sodium channels which trigger the opening of more sodium channels, finally bringing the membrane potential closer to its equilibrium potential. This depolarization has an almost vertical trend in the action potential curve.

2. During phase 1 the sodium channels start to close and, hence, this results in a decreased cytosolic sodium level causing a little drop in membrane potential. However, the action potential generated in phase 0 triggers the voltage-gated potassium channels to close and triggers the voltage-gated calcium channels to open.

3. In phase 2 the cytosolic calcium rapidly increases as the cytosolic potassium level rapidly decreases. However they counteract each other and maintain the level of membrane potential in the depolarized state. The increased cytosolic calcium level actively participates in crossbridge-cycle to cause muscular contraction. It keeps the ventricles contracted so that all the blood can flow out of them.

4. In phase 3 the delayed rectifier channels of the sodium, triggered open during phase 1 and 2 due to depolarization, start their influence now because they are
slow to respond. This small rise in the amount of cytosolic potassium pulls the potential towards negative, and also causes the opening of the other potassium channels which were closed due to the depolarization in phase 1. The rapid inflow of potassium causes further repolarization and the closing of calcium channels, resulting in more depolarization close to its equilibrium potential.

5. During phase 4 the resting potential of the membrane, -90 mV, is maintained because the cytosolic potassium level is very high compared to the sodium and calcium levels.

![Diagram of action potential](image)

Figure 2.31: Action potential of cardiac contractile muscle fibres located in the ventricles [12].

2.3.5 ECG wave recording

The electrocardiogram is a representation of electrical activity of the overall heart elements during a cardiac cycle. It is recorded by placing noninvasive electrodes on skin because body fluid behaves like a conductor, in order to prevent synchronized electrical activity from producing a larger amplitude at distance from the source. Because the electrical activity of the heart is more synchronized than others, it can therefore be detected distinctly from the skin surface. Willem Einthoven developed a recording technique by using three electrodes placed on an imaginary equilateral triangle with the heart at its center as discussed in an earlier section of this chapter. Figure 2.32 shows that each pair of electrode form a lead, with one electrode being positive and the other negative. A depolarizing action potential traveling towards the positive electrode results in an upwards deflection in the ECG wave, and a depolarizing action potential traveling towards the negative electrode leads to a downwards deflection in the ECG waveform.
The action potential generated at the SA node travels to the atrium and causes contraction there. While travelling to the AV node, it is delayed a little so that the ventricles can fill up before ventricular contraction. From the AV node it travels through the bundle branches, finally reaching the His Purkinje in the ventricle, causing ventricular contraction. While using Einthoven Triangle method, the ECG waveform is the sum of all these action potentials in time. Figure 2.33 shows a clear representation of ECG wave construction out of all the individual action potentials measured at the SA node, atrial muscles, AV node, common bundle, bundle branches, Purkinje fibre and ventricular muscles [33].

![Triangle electrode placement technique of Willem Einthoven](image)

Figure 2.32: Triangle electrode placement technique of Willem Einthoven [13].

The clinical ECG is recorded on paper running at a rate of 25 mm/sec with an amplitude of 1 mV/sec. The ECG waveform has the following characteristics:

1. P wave represents the atrial depolarization.
2. QRS complex is the representation of ventricular contraction.
3. T wave represents the ventricular repolarization.

The baseline of the ECG signal is called the isoelectric line, indicating that there is no electrical activity change going on. Some certain intervals provide vital information about the cardiac functions. Figure 2.34 shows the intervals of the ECG waveform which can reveal important information.
Figure 2.33: ECG construction from individual action potentials of cardiac muscles [33].

1. The P-Q or P-R interval represents the time of conduction through the AV node, and it starts from the onset of P and ends at the onset of the QRS complex.

2. The Q-T interval shows the time of ventricular contraction, also called the ventricular systole. It starts from the onset of the QRS complex and goes until the end of the T wave.

3. The R-R interval is the time span separating two QRS complexes, and defines the heart beat.

4. The T-Q time interval represents the ventricular relaxation, also called the ventricular diastole. It is a segment from the end of a T pulse to the beginning of the QRS complex.
2.3.6 Abnormal activity of heart

If a heart does not behave in a normal way it is called cardiac arrhythmias. If the sinoatrial (SA) node generates action potential abnormally it is called sinus arrhythmias. Sinus arrhythmias can either be tachycardia or bradycardia. In tachycardia, the heart beats abnormally faster (100 beats per minute) than the required rate. Figure 2.35 show an ECG recording of sinus tachycardia in which the T wave can be seen as inverted. The sinus arrhythmias can also be sinus bradycardia, in which the heart beats abnormally slower (50 beats per minute) than the required rate. Figure 2.36 depicts the ECG recording of sinus bradycardia [13].
The conduction through the atrioventricular node (AV), if changed, can cause various degrees of heart block. If the conduction through the AV node is delayed for longer than usual, it causes a first degree heart block which is reflected as an increased P-Q interval in the ECG. If the conduction through the AV node does not always occur, it causes a second degree heart block. If the conduction through the AV node is missed, the ventricles do not depolarize and contract, which is represented as a missing QRS peak in the ECG waveform. In a third degree heart block, the conduction through the AV node does not happen at all, and the association of atrial and ventricular contractions are completely lost. In this case, the ventricular contractions are controlled by the action potential generated by the His fibre which is only 30-40 times per minute. On the other end, the atrial contraction is triggered by the action potential generated from the SA node which is about 100 times per minute. The slow contraction rate of the ventricles is insufficient to meet the oxygen requirements of the body and it can be deadly. Figure 2.37 shows the third degree heart block which contains no QRS peak as evident from the ECG waveform.

![Figure 2.37](image1.png)

**Figure 2.37:** The ECG waveform recorded during third degree heart block [13].

The heart cells are connected by gap junctions, and an external action potential can sometimes trigger the atrial contraction called extrasystole. This premature atrial contraction (PAC) also lead to premature ventricular contraction (PVC). These contractions are not very much important unless they occur at high frequency. Figure 2.38 represents premature atrial contraction.

![Figure 2.38](image2.png)

**Figure 2.38:** The ECG waveform with premature atrial contraction (PAC) [13].

The unsynchronised depolarization of heart muscles is very serious arrhythmia, and is called fibrillation. If the atrial muscles depolarize independently it is known as
atrial fibrillation, and it leads to insufficient blood quantity pumped to the ventricles. Atrial fibrillation causes weakness and headaches, it is not very dangerous as long as the ventricular contractions are occurring regularly happening timely. However the blood churning in the atria can form a blood clot, and clogging the blood vessels leading to pulmonary embolism, stroke and cardiac arrest. Ventricular fibrillation is the most deadly arrhythmia and can cause death in a few minutes. It is recommended to defibrillate the ventricular muscles as quickly as possible by passing a large current through the chest wall of the patient so that an external trigger depolarizes all the muscle cells of the ventricles to save the life. Figure 2.39 shows an ECG waveform of ventricular fibrillation.

Figure 2.39: The ECG waveform representing ventricular fibrillation [13].
3 Electrophysiological signal processing

Electrophysiological signal processing involves the signal acquisition and algorithm development techniques needed to remove the noise and extract the signal parameters. The sources of noise for both EMG and ECG signals will be discussed in the next section, and then the various signal processing techniques that are used will be discussed in later sections.

3.1 Sources of artifact and noise

3.1.1 Artifacts in EMG

There are many types of noises and artifacts that affect the quality of sEMG signal recording. The EMG signal depends on various attributes of the structure of the human body such as skin formation, the velocity of blood flow, skin temperature, structure of tissues, measuring site selection, etc. All these attributes contribute noise to the EMG signal in one way or another [24]. The following lists examples of noise in an EMG signal.

- **Electrode noise:** The impedance of electrodes affect the EMG signal, and electrode noise depends on the size of the electrode as well. However too large electrodes are uncomfortable to wear and technically not feasible. High impedance reduces the signal quality and also degrades the signal-to-noise ratio. This noise can be reduced by using high quality instrumentation and intelligent circuit design.

- **Motion artifacts:** The relative movement and deformation of the skin surface under the recording electrode causes and electrode motion artifact which is low frequency noise at around 20 Hz, and can be eliminated by applying a high pass-filtering technique without compromising the EMG signal itself [21]. Using conductive gel electrodes also reduces the relative movement artifacts of the electrodes [24]. These motion artifacts occur when a patient is in resting state and not moving at all. Ambulatory EMG recordings bring movement artifacts which are very complicated to eliminate.

- **Electromagnetic noise:** The skin surface of the human body acts like an antenna, gathering all kind of ambient noise like the electromagnetic noise of power lines and electrical devices around. This brings a contamination of 50/60 Hz in the recorded signal and can be eliminated by using a good shielding for the electrode wires and high pass filtering [24]. Figure 3.1 shows four harmonics of the 50 Hz noise reference signal used to eliminate the powerline interference (PLI).

\[ PLI_{\text{ref}} = \cos(2\pi 50t) + \cos(2\pi 100t) + \cos(2\pi 200t) + \cos(2\pi 300t) + \cos(2\pi 400t) \tag{8} \]
Figure 3.1: Power line interference cancellation technique [24].

- **Crosstalk:** Crosstalk is caused by the action potentials of other closeby muscles which have an undesired effect on the EMG signal. This can be reduced by decreasing the size of the electrodes and making them more directional to the muscle being recorded. Reducing the inter electrode distance also improves the EMG against crosstalk.

- **Internal noise:** There are several anatomical, biochemical and physiological factors taking place depending upon the depth of the active muscle fibres, number of muscle fibres and number of tissues involved. These all contribute as internal noise added to the EMG signal. The amount of fat in the body also affects the EMG signal by increasing the distance between the active muscle fibres and the electrodes. High-pass spatial filters can be used to reduce these noises [24].

- **Inherent signal instability:** The motor unit firing rate is quasi-random in nature and it affects the EMG signal components between 0 to 20 Hz.

- **ECG noise:** ECG signal also acts as an artifact for EMG signals, specifically for the EMG signal from trunk muscles. It is eliminated by applying common-mode rejection, and by placing the electrodes along the heart axis [24]. It is most difficult to remove ECG effect because it overlaps the EMG feature. So, there is a need for more advanced noise cancellation techniques [21].

### 3.1.2 Artifacts in ECG

While recording ECG signals, the noise also becomes part of the desired signal. The noise usually has the same temporal distribution but differs in amplitude. The baseline wander is caused by low frequency noise, the medium frequency noise comes from power lines, and high frequency noise comes from the EMG and other electronics appliances nearby. The following are the noise types which affect the ECG recordings [35]:

- **Power-line Interference noise:** As discussed earlier in the EMG section, powerline interference overrides the ECG signal with the frequency of 50/60Hz. The PLI is modeled by:

\[
    n_{50Hz}(t) = Asin(2\pi * 50 + \theta)
\]  

(9)
• **Baseline wander**: It is caused by the electrode movement and changes in the skin-electrode interface. This is less than 1 Hz and is more common in ambulatory monitoring.

• **Channel noise**: It is introduced when the signal passed through poor channels, and is similar to white Gaussian noise containing all ranges of frequency [34].

• **Electrode contact noise** (**Burst noise**): It is introduced due to a poor connection between an electrode and skin.

• **EMG noise**: The action potential generated by motor nerves or muscle fibre during ECG recording also affects the ECG signal. It depends on the intensity of muscular contraction, and can be modeled by a Gaussian distribution function.

• **Motion artifacts**: Because of transient baseline changes, this is caused by electrode-skin impedance change because of motion.

• **Instrumentation noise**: ECG recording equipment components also contribute their noise to the ECG signal. The components in the recording chain such as electrodes, cables, amplifiers, ADCs and processors contribute this noise, and it can be reduced by using good quality instrumentation but unfortunately cannot be entirely eliminated.

### 3.1.3 Artifacts in ECG

The signal processing of an electromyogram involves the development of several techniques and methodologies. Some have been developed with the passage of time for analysis and diagnosis of the recorded data for clinical purposes [21]. Some of the advanced techniques are:

- **Wavelet Analysis (WA)**
- **Higher Order Statistics (HOS)**
- **Empirical Mode Decomposition (EMD)**
- **Artificial Neural Networks (ANN)**
- **Independent Component Analysis (ICA)**

The wavelet analysis technique will be discussed in detail here. Once the signal is segmented and processed, then signal classification is performed, and finally, it is used for control the applications as well – but that is beyond the scope of this thesis.

**Wavelet Analysis (WA)**: In signal analysis the time-frequency plane plays a fundamental role. To analyze the EMG signal the Wigner-ville distribution (WVD) is used. WVD has excellent localization properties which makes it concentrate on the instantaneous frequency and time of the EMG signal. It is a replacement of Fourier transform, and can be divided into discrete and continuous forms. As there is no downsampling, the continuous wavelet transform (CWT) is more consistent. The
Discrete Wavelet Transform (DWT) has been used successfully to analyze surface EMG signals, and it yields a high dimensional feature vector. The basic analytic expression of continuous wavelet transform is shown below [24], Where a is the scale and b is the time location. $\psi(t)$ represents the mother wavelet also taken as a band-pass function. $\sqrt{|a|}$ is an energy preservation factor. The time scale parameters $(a, b)$ can be discretized in different ways.

$$\psi(a, b) = \frac{1}{\sqrt{|a|}} \psi\left(\frac{t - b}{a}\right)$$ (10)

The discrete wavelet transform (DWT) is computed by successive low and high pass filtering in the discrete time domain. A general expression is shown below, where $a = 2k$, $b = 2kl$, and $d(k, l)$ is a sample of $W(a, b)$ at discrete points $k$ and $l$.

$$x(t) = \sum_{k=-\infty}^{\infty} \sum_{l=-\infty}^{\infty} d(k, l) 2^\frac{k}{2} \psi(2^{-k} t - 1)$$ (11)

<table>
<thead>
<tr>
<th>Wavelet Family</th>
<th>Wavelet Subtypes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haar</td>
<td>db1</td>
<td>1</td>
</tr>
<tr>
<td>Daubechies</td>
<td>db2-db45</td>
<td>2-45</td>
</tr>
<tr>
<td>Coiflet</td>
<td>coif1-coif5</td>
<td>46-50</td>
</tr>
<tr>
<td>Morlet</td>
<td>morl</td>
<td>51</td>
</tr>
<tr>
<td>Complex Morlet</td>
<td>cmor</td>
<td>52-147</td>
</tr>
<tr>
<td>Discrete Meyer</td>
<td>dney</td>
<td>148</td>
</tr>
<tr>
<td>Meyer</td>
<td>meyr</td>
<td>149</td>
</tr>
<tr>
<td>Mexican Hat</td>
<td>mexh</td>
<td>150</td>
</tr>
<tr>
<td>Shannon</td>
<td>shan</td>
<td>151-200</td>
</tr>
<tr>
<td>Frequency B-spline</td>
<td>fbsp</td>
<td>201-260</td>
</tr>
<tr>
<td>Gaussian</td>
<td>gaus</td>
<td>261-267</td>
</tr>
<tr>
<td>Complex Gaussian</td>
<td>cgaus</td>
<td>268-275</td>
</tr>
<tr>
<td>Biorthogonal</td>
<td>bior</td>
<td>276-290</td>
</tr>
<tr>
<td>Reverse Biorthogonal</td>
<td>rbio</td>
<td>291-305</td>
</tr>
<tr>
<td>Symlet</td>
<td>sym</td>
<td>306-324</td>
</tr>
</tbody>
</table>

If the MUAP wave shape matches with the shape of Wavelet transform, it shows excellent energy localization in the time-scale plane. So, usually wavelets in the shape of an MUAP wave are chosen. The WT is very useful to detect the MUAP from the signal affected by white noise. The signals are required to be stationary signals for fast and short-term Fourier transform. To overcome this problem, the signals are processed by using continuous WT at different resolution levels. Wavelet denoising algorithms are used to effectively remove White Gaussian noise (WGN) from the EMG signals. To apply this technique, the wavelet base function vtype, the
scale factor, the threshold rule selection, threshold rescaling factor and thresholding function are taken into account.

Wavelet function selection is a very important part in the process of removing noise using the wavelet transform, and it depends on the characteristics of the signal and type of application. There are three hundred and twenty four wavelet functions, and it is crucial to choose a suitable function and decomposition level for proper denoising of the EMG signal. Table 3.1 shows wavelet functions and families. To decompose a continuous function more efficiently, it is good to use a basic wavelet function because it has continuous derivatives.

It is recommended to use the Daubechies function (db2, db4, db6, db44 and db45) by decomposing it by four levels as shown in Figure 3.2 [42]. This leads to better results from surface EMG as used by Daubechies [24]. Figure 3.3-3.7 show the denoising of a raw surface EMG signal recorded from a rectus femoris muscle during maximum walking speed using wavelet functions db2, db4, db6, db44 and db45 respectively.

Figure 3.2: Decomposition tree and level of decomposition [42].
Figure 3.3: The sEMG signal is denoised by using wavelet function db2 [24].

Figure 3.4: The sEMG signal is denoised by using wavelet function db4 [24].

Figure 3.6: The sEMG signal is denoised by using wavelet function db44 [24].
Figure 3.5: The sEMG signal is denoised by using wavelet function db6 [24].

Figure 3.7: The sEMG signal is denoised by using wavelet function db45 [24].

3.1.4 ECG signal processing

The ECG signal possess a frequency band of 0.05 to 100 Hz. The noise factors discussed in section 3.1.2 affect the Q and P waves of the ECG signal specifically which may lead to errors in clinical diagnosis [36]. There are many techniques used to eliminate the noise from ECG signals such as FIR filtering, adaptive filtering, discrete wavelet transform (DWT), bionic wavelet transform (BWT), the filtered residue method and empirical mode decomposition [35]:

Kalman filter: The Kalman filter is a recursive estimator that estimates dynamic systems using a series of noisy measurements. It consists of a prediction phase, which means the n-1 state and covariance estimation, and an update phase, which calculates new a new statistic and covariance [36].
Prediction phase:

\[ \bar{x}_k = A \bar{x}_k \]  

(12)

\[ P_k = A \ast P_{k-1} \ast A^T + Q + k \]  

(13)

Update phase:

\[ K_k = P_k \ast H_k^T (H \ast P_k \ast H_k^T + R)^{-1} \]  

(14)

\[ x_k = X_{k-1} \ast K_k (Z_k - H \ast x_{k-1}) \]  

(15)

\[ P_k = P_{k-1} - (K_k \ast H \ast P_k) \]  

(16)

Where \( A = [100 \ 11] \), \( Z \) is the input signal, \( K \) is the gain factor of the Kalman filter, \( \bar{x} \) is the estimated state, \( P \) is estimated error covariance, \( Q \) and \( R \) are noise covariances. The Kalman filter does not require the knowledge of noise or signal frequency, because being an adaptive filter it adapts to the noisy signal.

**Least mean square filter (LMS):** The least mean square filter is also an adaptive filter, which calculates the difference between the actual and desired signals, and finds the desired filter coefficients [36]:

\[ Y_n = W_{n-1}^T \ast u_n \]  

(17)

\[ E_n = d_n - Y_n \]  

(18)

\[ W_n = \alpha W_{n-1} + f(u_n, e_n, \mu) \]  

(19)

where \( Y_n \) is the filter output at step \( n \), \( W_n \) is the vector of the filter weight which estimates at step \( n \), \( u_n \) is the buffed input sample at step \( n \), \( E_n \) is the estimation error at step \( n \) and \( d_n \) is the desired response at step \( n \). The LMS filter always require a reference input in correlation to the noise or in correlation to the desired signal. This factor limits the use of an LMS filter to remove noise from ECG signals.

**Discrete wavelet transform (DWT):** In discrete wavelet transform the signal is passed through two correlated filters. They are called quadrature mirror filters, where the first one is a low pass filter and gives an approximation coefficient at the output, whereas the second one is a high pass filter and gives detail coefficients at the output. This method is repeated \( N \) times to achieve good frequency resolution. The approximation coefficients are divided into a new approximation set and details coefficients. The number of levels \( N \) is calculated by the equation [36]:

\[ N = \log_2(K) \]  

(20)

Where \( K \) is the number of samples. Figure 3.8 shows the multilevel decomposition of discrete wavelet transform. Every level shrinks the original signal and prevents getting full information within the signal. This issue is resolved by using undecimated wavelet transform (UDWV) which is explained in the following section.
**Undecimated Wavelet Transform (UDWV):** Undecimated wavelet transform un-samples the filter coefficients, and it is derived from classical discrete wavelet transform. It is also called stationary wavelet transform. The signal shrinkage problem is resolved by ensuring that all the coefficients have the same length. Thus, it leads to better denoising quality. Figure 3.9 shows a basic algorithm for multilevel decomposition of undecimated wavelet Transform [36].

![Multilevel decomposition of Discrete wavelet transform](image)

Figure 3.8: Multilevel decomposition of Discrete wavelet transform [36].

![Multilevel decomposition of undecimated wavelet transform](image)

Figure 3.9: Multilevel decomposition of undecimated wavelet transform [36].

### 3.2 Electrode types

Biopotential electrodes play vital role in the measurements of biological signals like ECG, EEG and EMG. There are two main types of electrodes polarizable electrodes and non-polarizable electrodes.
3.2.1 Non-polarizable electrodes (Conventional wet method)

Ag/AgCl are categorized as non-polarizable electrodes and are widely used in clinical measurements. Electrolyte gel is used to reduce the electrode-skin interface resistance and to improve the environment for electrochemical reactions [38]. This is also called the resistive ECG method. A path with a resistor and interface potential is used as shown in Figure 3.10. A charge transfer environment is established by the electrolyte between the skin and the electrode [39].

3.2.2 Polarizable electrodes (Dry method)

Modern wireless sensor technologies use these electrodes that have comparatively higher electrode-skin interface resistance. Nonetheless, these electrodes cause skin irritation and allergies due to the metal contact with the body in long-term use.

- **Orbital electrodes**: Orbital electrodes are dry polarizable electrodes made of a mixture of metals like silver/silver chloride, aluminum, gold/gold chloride, nickel and titanium. Such electrodes makes a good connection due to spikes on them, and does not need for any skin preparation to apply them.

- **Stainless steel electrodes**: Stainless steel electrodes are dry and have higher electrode-skin interface resistance.

- **Textile electrodes**: SMARTA project used silver-based conductive yarn to stitch three electrodes. The yarn has resistance of 30 ohm/m. The resistance can be reduced to 1-5 ohms by making ribbon from the electrode to the device. Sweat helps to improve the conductivity and reduce motion artifacts [40].

- **Dry foam electrodes** A comparison between dry foam electrodes and wet gel electrode was made. Dry electrodes were developed from an electrically-conductive polymer made by urethane material with a compression set of 5-10%. A layer of taffeta material made from electrically-conductive fabric was used to cover the foam. The foam was also coated with Ni/Cu to establish the electrical contact. A very thin adhesion layer of Au was used to connect the leads [41].

It is concluded that the signal quality from dry foam is almost identical. For long-term monitoring the skin-electrode resistance is stable in comparison to wet electrodes. During motions conventional electrodes present more artifacts.

3.2.3 Capacitively coupled electrodes

For long-term ECG data collection applications, a noncontact ECG method is used which is also called capacitively coupled ECG (CC-ECG). A thin layer of insulator is placed between the metal electrode and skin. The signal is coupled through the capacitive path in this scenario as shown in Figure 3.10. Jamal, Ibrahim and Tapas designed a two-lead wireless ECG monitoring system based on this method by focusing on convenience, portability and small size. low-power, low-cost integrated circuits
were chosen, and battery-saving techniques were implemented. Three identical PCBs were used as electrodes to acquire the signal and common-mode attenuation[40].

**Pro and cons of CC-ECG method:**

- Removes all the issues of using gel.
- Brings convenience for the patient in long-term use cases.
- It can be implemented unnoticeably in a chair, bed, clothes, etc.
- Brings unbiased data collection for continuous and vital signs monitoring because the user could be unaware of the sensor.
- Signal quality is not good due to high impedance between the ECG source and the sensor.
- Electrode is prone to environmental noise because it behaves as an antenna due to high impedance.
- Due to change in the placement, the coupling capacitance in the electrode changes which affects the ECG signal. Thus it’s more sensitive to motion noise as compared to fixed wet electrodes. The ECG signal is overwhelmed by the displacement current flow.

### 3.3 Applications

#### 3.3.1 EMG applications

Electromyography has a vast range of applications in the clinical and fitness fields of life. Here are some examples of possible applications [21].

- **Clinical diagnosis:** EMG is used to diagnose neuromuscular diseases, specifically the invasive electrodes EMG is used for individual waveform studies of MUAPs, and it helps to detect abnormal activities in case of muscle damage, muscle inflammation, nerve damage, pinched nerve and muscular dystrophy.
• **Kinesiology:** Surface EMG is very useful in body movement studies and its control. It is useful for clinical diagnosis of movement disorders. sEMG is more comfortable to record for longer time spans.

• **Ergonomics:** The amplitudes of sEMG are used for quantitative studies of muscle load during work. For this purpose, sEMG is recorded for light repetitive tasks to study the activity of specific muscles at a particular position during work. This study helps to avoid the fatigue of muscles during specific work tasks.

• **Prosthesis control:** sEMG can also be used to operate the prosthesis of amputated limbs by recording the motor nerve signal generated during voluntary tasks. These EMG signals can be processed and used to control the prosthesis in real time. The same techniques can be used for further gadget developments, for instance wheelchair control [22] and virtual keyboard control [23].

3.3.2 **ECG applications**

An electrocardiogram reveals enormous important parameters of human health. The analysis of patterns and frequency of ECG following important information is extracted [37].

• Heart rate
• Heart rhythm
• Conduction abnormalities
• Heart orientation in the chest cavity
• Hypertrophy (Evidence of increased thickness of heart muscle)
• Evidence of damaged heart muscle
• Acutely impaired blood flow to heart muscle
• Warning signs of abnormal cardiac rhythm disturbances

ECG is also very helpful to diagnose the following health conditions of human healthcare [37].

• Fast or irregular heart rhythms.
• Abnormally slow heart rhythms.
• Abnormal conduction of cardiac electrical impulses, which is very helpful in diagnosing cardiac or metabolic disorders.
• Myocardial infarction (Prior heart attacks).
• Unstable angina (blood flow level decreased during heart attack).
• Cardiac damage from other diseases like high blood pressure and thyroid.

• Cardiac damage from certain lung conditions, such as emphysema and blood clots in lung.

• Cardiac dilatation (Enlarged cardiac chambers).

• Abnormal blood electrolytes - involving calcium, magnesium and potassium.

• Inflammation of the heart (myocarditis) or its lining (pericarditis).

• Cardiomyopathy - a range of conditions in which the heart muscle (myocardium) does not function normally, including several congenital forms of cardiomyopathies.
4 Research materials and methods

The indigenous hardware development of the ECG and EMG platforms, and the methods to collect, filter and process data, will be discussed in this chapter.

4.1 Materials

The development of bioelectrical hardware has always been very critical because it brings threats of electric shocks, and may lead to temporary or permanent disability of human organs. In severe cases it may lead to life threatening situations as well. The electric power sources are miniaturized for two reasons, one to enhance the battery life, and second to avoid electric shocks. The off-the-shelf electronic components and devices used to measure the electrocardiogram and electromyogram. Focus of this thesis is to develop the ambulatory platforms to collect ECG and EMG data from the human body, and transmit it wirelessly to the closest access nodes so that any critical data could be sent further to investigation units for prompt action in emergency situations. The development process was broken into three phases to develop ECG, EMG and IMU platforms independently, and then combine the whole hardware so that all the sensors are powered by the same power source and sampled by the same processing unit. The equipment selection and individual hardware development is discussed in detail in the upcoming sections.

4.1.1 ECG hardware platform development

The ECG hardware platform consists of electrodes to collect electric activity from the body, filters and amplifiers to improve signal quality, and an analog to digital converter (ADC) to convert the collected analog signal into digital form for further processing and data storage. In this thesis the single-lead ECG hardware was developed having three wet-type electrodes.

Figure 4.1 depicts the deployment position of three wet electrodes on the human body where the LA (left-arm) yellow electrode is the positive terminal, the RA (right arm) red electrode is the negative terminal and the RL (right leg) green electrode is a drive terminal. For detection of the ECG signal, the filtering and amplification of the initial radio frequency interference (RFI), an off-the-shelf integrated circuit AD8232 by Analog Devices was selected to develop the ECG acquisition platform for this thesis. It uses 3.3volts and a 170µA current to be very feasible for ambulatory and long-term ECG monitoring [43]. AD8232 has the following circuit blocks for ECG signal filtering and amplification.

RFI Filter: The biopotential from the electrodes also contains radio frequency noise from the environment, appearing as a dc offset voltage in the output. This radio frequency interference is filtered out with a low-pass filter designed using on-chip capacitors. Figure 4.2 depicts the low-pass filters having increased filtering with the addition of series resistors as well. These are placed very close to the instrumentation amplifier inputs and help in overload and patient protection.

Instrumentation amplifier: The biopotential signals collection from single-lead electrodes is fed to an instrumentation amplifier through the RFI filter. The right
arm RA biopotential signal goes to the non-inverting input and the left-arm biopotential signal goes to the inverting input of the instrumentation amplifier. The instrumentation amplifier consists of two well matched transconductance amplifiers (GM1 and GM2), a dc blocking amplifier (HPA) and an op amp integrator [43].

The voltage at the input generates a proportional current through GM1. GM2 gets the feedback from a voltage divider and a dc blocking amplifier which integrates the deviation from the reference voltage. A voltage equal to GM1 also appears across the inputs of GM2 when the feedback is satisfied. The error current is integrated by an op amp integrator to produce the output voltage. A charge pump with a 500 KHz internal oscillator is used to boost the supply voltage for the transconductor amplifiers in order to improve the common voltage range of the instrumentation amplifier. Figure 4.3 shows a simplified schematic of the instrumentation amplifier [43].

**Operational amplifier:** A rail-to-rail general operational amplifier is used at the output of the instrumentation amplifier to achieve addition gain and perform
low-pass filtering. Figure 4.4 shows a simplified version of the output operational amplifier [43].

**Right leg drive (RLD) amplifier:** The output signal of the transconductance amplifier GM1 is applied to the non-inverting input of the RLD amplifier which is configured as an integrator to give a loop gain at frequencies of 50Hz and 60Hz for common-mode line rejection. By tuning the integrator capacitor values, the crossover frequencies can be tuned with the best available gain. The output of the RLD amplifier goes to the right leg RL electrode on the test subject with the maximum 10uA current flow. This current contracts common-mode voltage variations to improve the common-mode rejection ratio. Figure 4.5 shows the RLD amplifier used in SD8232 [43].

The ECG hardware platform was designed using KiCAD software, and the PCB was milled using the inhouse PCB milling machine at TUAS in Aalto university. The component soldering and assembly was performed at the electrical workshop of TUAS. A simple microcontroller based on Arduino was used to collect and visualize the ECG data output during testing of the ECG hardware platform. The author
used himself as a test subject for ECG signal acquisition and recording. Figure 4.6 shows a working version of the ECG platform.

![Indigenously developed ECG platform based on AD8232.](image)

**Figure 4.6** Indigenously developed ECG platform based on AD8232.

### 4.1.2 EMG hardware platform development

Electromyography is used for medical research and also in prosthetic control systems as discussed in the second chapter of this thesis. For EMG hardware platform development, the precision instrumentation amplifier AD8221 by Analog Devices is used to measure the EMG signal from two differential electrodes on the muscle, and one ground electrode away from the muscle, on a bony location. The differential action potential of the single muscle from a short distance (2 cm) is then fed to the precision instrumentation amplifier for measurement. Figure 4.7 shows the placement of the electrodes on the biceps muscle to measure the EMG [44].

The red electrode connected to the +IN input of the precision instrumentation amplifier AD8221 and the yellow electrode is connected to the -IN of the precision instrumentation amplifier. Figure 4.8 shows the simplified schematic version of the instrumentation amplifier AD8221 [44]. It is based on the classic three op amp topology in which the transistors Q1 and Q2 are biased with a constant current so
that any differential input will be fed to A1 and A2 op amps accordingly. Both input setups with transistors and op amps work as precision current feedback amplifiers. The op amp A3 receives the amplified differential signal at the inputs and common-mode signal as well which is cancelled. The common-mode rejection ratio is more than 90 dBs [44].

Figure 4.8: Simplified schematic of instrumentation amplifier by Analog Devices [44].
The gain bandwidth product is enhanced by increasing the gain because of the current feedback amplifiers at the input. The gain of the AD8221 is given below which is dependent on the external resistor $R_G$ [44]:

$$G = 1 + \frac{49.4K\Omega}{R_G}$$  \hspace{1cm} (21)

Once the EMG input signal is measured by the precision instrumentation amplifier AD8221, it is then fed to the precision full-wave rectifier. A general purpose JFET operational amplifier TL084 by STMicroelectronics was used for full-wave rectification. Figure 4.9 shows the schematic of the full-wave precision rectifier by using two op amps of the TL084.

![Figure 4.9: Schematic of full-wave precision rectifier [44].](image)

The full rectified signal is then fed to an integrator for high-frequency noise filtering and then finally amplified by an operational amplifier. A variable resistor VR controls the gain of the operational amplifier. Figure 4.10 shows the schematic of the integrator and amplifier. The amplified signal is then fed to the ADC for analog to digital conversion. Both of these op amps were used from the same package of the TL084 by STMicroelectronics.

![Figure 4.10: shows a schematic diagram of the integrator and amplifier [44].](image)

The EMG platform was designed using KiCAD and developed using the resources present at TUAS. The dual power supply was made using two 9V alkaline batteries.
Figure 4.11 shows the hardware of the EMG sensor which is used with the Arduino development board to collect and visualize the recorded data.

Figure 4.11: EMG hardware platform running with arduino development board.

4.1.3 IMU hardware platform development

The inertial measurement unit (IMU) hardware platform was designed and developed using the MPU-9250 by InvenSense. It is a multichip module having a 3-axis gyroscope, 3-axis accelerometer and 3-axis magnetometer which provide nine degree of freedom (9DOF). It also has a digital motion processor on-chip for the sensor fusion facility on the chip. The MPU-9250 was initially used with the Arduino board to test the functionality. The I2C bus communication interface of 400 KHz was established between the sensors and the CPU. Figure 4.12 shows the basic IMU hardware platform running with the Arduino development board.

Figure 4.12: IMU hardware platform with Arduino development board.
4.1.4 Combined platform development

The ECG hardware platform, the EMG hardware platform and the sensor IMU hardware platform were brought together on a single unit to use the same power and processing resources. A 24-bit sigma delta analog to digital converter (ADC) ADS1220 by Texas Instruments was used to digitize the ECG and EMG waveforms. Figure 4.13 shows a functional block diagram of the AD1220 which has four channels and the SPI interface was established between the ADC and the processing unit [45].

The combined hardware is developed by using an Atmel microcontroller ATmega328 to collect, manipulate and send all the data from the ECG, EMG and IMU. the Arduino bootloader was uploaded to the microcontroller to program it by using IDE. An off-the-shelf bluetooth RN-42 modem by Sparkfun was used to establish a communication protocol between the CPU and other data-receiving units such as laptop, smartphone and desktop computers. It has a class 2 radio and works up to a distance of 60 feet at 2.4 GHz ISM band with a baud rate up to 921 Kbps. A second communication serial interface is developed for controller code burning to the microcontroller and also for the wired high data rate connection. The IMU was placed on the main hardware board, and the board was tied to the chest of the test subject to collect the motion data. An off-the-shelf 5V portable power bank was used to power up the combined hardware platform. Figure 4.14 shows the block diagram of the combined hardware platform with the placement of electrodes and IMU position on the test subject.

The combined hardware platform schematic and the PCB were designed using KiCAD software. The PCB was then milled using the milling machine available in the measurement lab at TUAS. The component soldering, assembly, and hardware debugging was performed in the electric workshop at TUAS. Figure 4.15 shows the running version of the combined hardware platform which is tied on the chest of the test subject.
4.2 Methods

Once the hardware platform was developed and tested, the methods for data collection, data recording and processing were devised. The CPU on-board was used only for data sampling, organizing and transmitting through bluetooth. The bluetooth interface at a baud rate of 115200 was established with a laptop device for data reception wirelessly. An open source data recording application was used to record the data in text files continuously for prescribed test events. The signal processing, filtration, data analysis and algorithm implementation was done on a laptop using MATLAB® R2017b version 9.3 by MathWorks Inc.

4.2.1 ECG data collection

The ECG data is digitized and then sampled by the processor on-board and finally sent to the laptop by the bluetooth interface along with the other data. For ECG
motion artifact analysis, the following events were planned in such a way that each event was recorded for one-minute time span:

- ECG recording while laying still.
- ECG recording while sitting still.
- ECG and IMU data recording while sitting and breathing heavily.
- ECG and IMU data recording during transition from sitting position to standing position and vice versa.
- ECG and IMU data recording while walking with normal speed.

4.2.2 EMG data collection

The EMG for the biceps muscle is focused in this thesis and the events related to the left-arm biceps muscle movement were planned. The EMG data is also digitized and sampled by the on-board CPU before sending it along with the data from other sensors. The following events were planned and executed for EMG data collection with a time span of 30 seconds:

- EMG and IMU recording while repeated folding arm on elbow pivot with empty hands.
- EMG and IMU recording while repeated folding arm on elbow pivot by carrying weight in hand.
- EMG and IMU recording while stretched arm and still making 90 degree with subject body and holding weight in hand.

4.2.3 Data recording

The on-board CPU is used to organize the sampled data of all sensors in one line separated by commas. All the samples were made at the frequency of 100 Hz. An open source application was used on a laptop to record the received data in text files. Later on, MATLAB® R2017b version 9.3 was used to import and delimit the data of all the sensors from the text files for further processing.

4.2.4 Filters and algorithms

The ECG and EMG signals are very noisy when recorded as explained in chapter 2. These signals are then analyzed to find out the noise components. A fast fourier transform is performed to analyze the frequency components in the frequency domain. Then different denoising algorithms are applied to remove the noise elements and extract the sensor signals. The finite impulse response filter (FIR), Savitzky-Golay filtering technique, The heart-beat detection algorithm and the Pan-Tompkins QRS detection algorithm was applied to detect the QRS peaks in the ECG signal.
MATLAB® R2017b version 9.3 by MathWorks Inc. was used for the implementation of all the above signal processing techniques. The results of the signal recording and analysis are discussed in chapter 4.
5 Results

The results of the signal processing techniques applied to the recorded ECG and EMG data are discussed in this chapter. *MATLAB®* R2017b version 9.3 is used to apply the data processing techniques and to visualize the data graphically.

5.1 ECG data processing

The ECG data was preprocessed to make it ready for analysis and further processing. The data was organized in chunks of one-minute, and then it was analyzed and processed using various data processing techniques in *MATLAB®* R2017b version 9.3. For better visualization, 30-seconds duration graphs are represented in the following sections.

5.1.1 ECG data detrending

The raw version of the ECG recorded data is preprocessed to remove the dc components from the data. A data detrending technique is applied to remove the nonlinear trends from the ECG data. To detrend the data, the mean of the whole recorded data set of 60-seconds is calculated and then removed from each of the data points, hence removing the dc offset trends from the actual signal. Figure 5.1 shows the detrending process performed on the ECG data recorded during the event – ECG recording while laying still.

![ECG data detrending](image)

Figure 5.1: ECG data detrending.
5.1.2 High frequency noise components removal

The detrended signal is then filtered by using Savitzky-Golay filter to smooth the digital data and improve the signal-to-noise ratio. The data smoothing is based on local least-squares polynomial approximation by forming low pass filter which is equivalent to discrete convolution with a fixed impulse response [46]. The sgolayfilt function of MATLAB® R2017b version 9.3 is used to filter the ECG data by using the built in function. Figure 5.2 shows a 5 second plot of ECG data detrended and ECG data through S-G filter for the event – ECG recording while laying still.

![ECG data filtered using an S-G smoothing filter for event – ECG recording while laying still.](image)

5.1.3 Breathing motion artifact removal

The S-G filter has removed the high frequency components of noise from the ECG signal to smooth it out. The low-frequency components of the breathing motion artifact and chest muscle action potential artifacts are still part of the ECG signal. A fast fourier transform is applied to the smoothed ECG signal, and lower frequency components were removed. Figure 5.3 shows a 30-second plot of the S-G filtered signal, and then an FFT of the signal, and, finally, a plot of the ECG signal with breathing artifact removed for the event – ECG recording while laying still. The same procedure was performed for the event – ECG recording while sitting still, because it also presented the same breathing artifacts and chest muscle action potential artifacts. Figure 5.4 shows a 35-second plot of the event – ECG recording while sitting still.
The IMU data was recorded for the event – sitting and breathing heavily. The fast breathing brought motion artifacts into the ECG signal which were also detected by all axis of the accelerometer of the IMU. Figure 5.5 shows 60-second plot of the detrended ECG signal and the 3-axis of accelerometer to depict the heavy breathing motion artifact. Figure 5.6 shows the 30-second plot of the motion artifact removal from the ECG signal recorded during the breathing heavily event using detrending, S-G filtering of high frequency noise and signal smoothing, FFT and the filtering of low-frequency noise components respectively.

5.1.4 Sit-stand motion artifact removal

The IMU data was also recorded for the event – ECG and IMU data recording during transition from sitting to standing position and vice versa. These repetitive and almost symmetric movements introduced motion artifacts into the ECG recording. All 9 axis of the IMU reflected the event movements: Figure 5.7 depicts the ECG and the 3-axis of the accelerometer recordings, Figure 5.8 represents the ECG and the 3-axis gyroscope recordings, and Figure 5.9 shows the ECG and 3-axis of the magnetometer recordings. Finally, Figure 5.10 elucidates the sit-stand motion artifact removal from the ECG signal using data detrending, S-G filtering and filtering of the low-frequency noise components.
5.1.5 Walking motion artifact removal

The ECG data was recorded for 60-seconds of normal walking while the IMU on board was tied to the chest. The walking movement introduced artifacts in the ECG data. The same motion was also recorded by the 9-axis of the IMU on the board. The ECG signal and IMU data were detrended for further processing. Figure 5.11 shows a 30-seconds plot of the detrended ECG signal containing the walking motion artifacts and the 3-axis accelerometer data. Similarly, Figure 5.12 represents a 30-second plot of the detrended ECG signal and the 3-axis data of the gyroscope.

The ECG signal was then passed through the S-G filter for smoothing and filtering of the high frequency noise components. Then FFT was performed and all low-frequency noise components were removed. Figure 5.13 depicts a 30-seconds plot of the ECG signal processed by high and low pass filters.

5.1.6 QRS peaks threshold filter

The QRS peaks of the ECG data after motion artifact removal were still uneven in amplitude. A QRS threshold filter was implemented to remove this effect from the ECG signal. A sliding window filter was used to detect the QRS peaks, and then a threshold factor was used to remove the uneven motion effect from the QRS peaks. Figure 5.14 shows the output results of threshold filtering for all the events.
5.1.7 QRS peaks detection

The QRS peaks are the highest points in the ECG signal which represent the ventricular contraction, and it repeat once in the ECG waveform of a single heart beat. The QRS peaks is the recording of the action potential running through the contractile cells of the ventricle to squeeze the blood out of the ventricle, and it is very prominent because of its highest potential in the ECG waveform. A famous Pan-Tompkins algorithm was applied in MATLAB® R2017b version 9.3 to detect the QRS peaks from the ECG recorded signal for all events. For the QRS detection implementation, the ECG data was first detrended to cancel the dc drift. Then the signal was passed through the S-G low pass filter for signal smoothing and high frequency noise filtering, followed by a high pass filter to remove the low-frequency noise components.

A moving window algorithm with a window size, half of an approximated single ECG cycle, was used to detect the highest peak in the window. The magnitude of the data points were compared to identify the local maxima in the sliding window, and every QRS peak was flagged using the timestamp. The flagged timestamp was then used to calculate the minimum distance between two peaks. This minimum distance helped to optimize the window size. A second pass through the optimized sliding window was implemented to find the QRS peaks. Figure 5.15 shows a 30-second plot of the ECG signal with QRS peaks detected for the event – laying still.

Figure 5.5:  ECG signal and accelerometer recordings during event – breathing heavily.
The QRS peak-detection process was repeated for the event – sitting still. Figure 5.16 represents a 30-second plot of the ECG signal with QRS peaks detected for the event – sitting still. The top plot shows the result of the detrending algorithm used to remove the dc offset from the ECG signal. The breathing artifact is still visible as a very low-frequency component. The breathing creates two type of artifacts in the ECG signal. One, the motion of the chest and skin stretching which alters the skin-electrode impedance, and hence affecting the ECG recording. The other is the chest muscle action potential, which is also recorded by the same electrodes used for the ECG recording. The middle plot represents the flagged timestamps at the positions of the detected QRS peaks. The bottom plot in blue represents the unprocessed ECG signal, with all the noise and artifacts superimposed by the red plot of the detected QRS peaks and their magnitudes.

An ECG signal plot of 30-seconds for QRS peaks detection in the event – breathing heavily is depicted in Figure 5.17. The breathing artifact in the form of low-frequency components is clearly visible. However, in comparison to normal breathing in the event – sitting still, it is at a higher rate and not very consistent due to the random pace of fast breathing. It is still possible to detect the QRS peaks from the breathing heavily event using the Pan-Tompkins algorithm because the variation in skin-electrode impedance and chest muscle action potentials are not high enough to override the QRS peaks.

During the sit-stand event, the movement of arms and shoulders was also part of the exercise, which greatly influenced the ECG recording because the ECG electrodes
were placed very close to the shoulders. The action potential of the shoulder muscles was high enough to override some of the QRS peaks, and the ECG data there was totally lost. The sit-stand motion also brought mechanical pulls on the electrode wire during transition that is another artifact which affected the ECG waveform. The sliding window of the QRS peaks detection algorithm was not capable to finding the local maxima on the estimated QRS peak destination, and thus some QRS peaks did not appear in the ECG data plot. Figure 5.18 shows a 30-seconds plot of the ECG signal with QRS peaks detected for the event – sit-stand, where the missing peaks can be visualized because of the motion artifacts.

A plot of a 30-second ECG signal in Figure 5.19 elucidates the QRS peaks detection for the event – walking. During this event, there were no big contractions happening in the arm and, specifically the shoulder muscles. Therefore, the ECG electrodes did not record any large muscle action potential in their vicinity which could override the QRS peaks of the ECG signal. The sliding window algorithm was sufficient enough to detect almost all the QRS peaks as represented on the middle plot of Figure 5.19. There was, however, a low-level consistent walking motion artifact recorded with the ECG signal, which represent the footsteps, and these are visible in the IMU data as shown in Figure 5.11 and Figure 5.13.
5.2 EMG data processing

The EMG data preprocessing was done before proceeding to further processing operations. *MATLAB®* R2017b version 9.3 was used for implementation of various processing techniques on organized data in chunks of 10-60-seconds for better visualization.

5.2.1 EMG data detrending

The EMG data recorded in raw version had dc drifts in the signal. It was preprocessed to remove the dc components from the EMG action potential peaks. A data detrending technique is applied to remove the nonlinear trends from the EMG data. Figure 5.20 shows the detrending process performed on the EMG data recorded during the event – repetitive elbow bending with empty hands.

5.2.2 Removal of high frequency noise components

The Savitzky-Golay filter was used to smooth the EMG digital data and improve the signal-to-noise ratio. The local least-squares polynomial approximation was used to smooth the data by forming a low-pass filter which is equivalent to discrete convolution with a fixed impulse response.

The sgolayfilt function of *MATLAB®* R2017b version 9.3 was used to filter the EMG data with the frame size of 17 and zero degree arguments. Figure 5.21 shows a
5-second plot of the EMG data detrended and the EMG data through the S-G filter for the event – EMG recording while repetitively elbow bending with empty hands. While recording the EMG data, the IMU on board was tied to the chest, and the left arm was used for the EMG signal collection for all events described in section 4.2.2.

The elbow bending movements were also reflected by the IMU tied on the chest of the test subject. Figure 5.22 represents a 10-seconds plot of the EMG data with 3-axis accelerometer deflection. The EMG data is filtered through the S-G filter to remove the high frequency noise. Figure 5.23 shows a 10-second plot of the same EMG data with the 3-axis of the gyroscope representation. However the magnetometer did not contribute here because the test subject was sitting all the time facing only one direction during the EMG recording for all events.

**5.2.3 EMG signal processing for event – weight lifting**

The EMG data was recorded with the 9-axis IMU tied on the chest of the test subject while repetitively lifting a weight of 5Kg for 60-seconds. The EMG signal was filtered for high frequency noise components and smoothed by using the S-G filter. Only the gyroscope reflected the muscle movements during this event for two reasons: first, the IMU was tied on chest and, second, the frequency of the biceps muscle contract was reduced due to weight lifting. Figure 5.24 shows a 30-second plot of the S-G filtered EMG with the 3-axis of the gyroscope.
5.2.4 EMG signal processing for event – heavy weight lifting

The EMG of the left arm biceps muscle was recorded during this event and an object that was too heavy to lift was chosen. A comparatively less frequent failed lifting attempt was made for a 60-seconds period of time. The action potential peak was prolonged over the time span of each lift attempt. There was a vibration in the biceps muscle when maximum force was applied, and this vibration was recorded in the action potential peaks – and also deflected by all 3-axis of the accelerometer and 3-axis of the gyroscope of the IMU data. Figure 5.25 depicts a 45-second plot of the S-G filtered EMG signal and the 3-axis data of the accelerometer. Similarly, the same EMG is shown in Figure 5.26 with the 3-axis gyroscope data.

The frequency of the action potential peaks in the EMG signal reflects the frequency of the lift attempts made during the EMG event data recording. The amplitude of the EMG wave represents the amplitude of the action potential. The biceps muscle vibration during this event represents the variation in the action potential of the afferent branch for the biceps muscle. There is a gradual rise noticed in the beginning of each peak of the EMG which represents the recruitment of additional motor units to support the lifting event. A flat trend in the peak of the EMG signal depicts that all the motor units of the biceps muscle are engaged in the heavy weight lifting event.
Figure 5.11: ECG signal and 3-axis accelerometer recording during walking motion.

Figure 5.12: ECG signal and 3-axis gyroscope recording during walking motion.
Figure 5.13: Walking movement artifacts removal from the ECG signal using an S-G filter and high pass filters.

Figure 5.14: The QRS peaks in ECG using threshold filter for all events.
Figure 5.15: Laying still ECG signal processed to detect QRS peaks.

Figure 5.16: Sitting still ECG signal processed to detect the QRS peaks.
Figure 5.17: Breathing heavily ECG signal processed for QRS peak detection.

Figure 5.18: The ECG signal with sit-stand motion is processed to detect the QRS peaks.
Figure 5.19: The ECG signal processed to detect the QRS peaks for the walking event.

Figure 5.20: EMG data detrending.
Figure 5.21: EMG smoothing using S-G filter for elbow bending with empty hands.

Figure 5.22: The EMG recording with 3-axis accelerometer for elbow bending with weight.
Figure 5.23: The EMG recording with the 3-axis gyroscope for elbow bending with no weight.

Figure 5.24: The EMG signal with 3-axis gyroscope for elbow bending with weight.
Figure 5.25: EMG with 3-axis accelerometer for elbow bending with heavy weight.

Figure 5.26: EMG with 3-axis gyroscope for elbow bending with heavy weight.
6 Summary

The human body has an enormous capacity to sensing, control and maintain routine daily activities through the nervous system by using an electrochemical communication network spread out on a cellular level. Most of these electrochemical activities are measured from the skin surface using different types of noninvasive electrodes. The measured electrical signals are used to monitor healthcare and diagnose various diseases. This thesis focused on the electrocardiogram (ECG), the electrical activity of the heart, and the electromyogram (EMG) – the electrical activity of muscular contractions. Both are used for diagnosis of various critical health abnormalities, and the former is also used for prosthesis development.

The ECG comprises all the action potentials happening in the human heart during a single beat, and is represented as a standard P-QRS-T waveform. The deviation from the standard structure of the waveform leads to the diagnosis of various heart abnormalities depending upon the type and level of deformation. Similarly, the EMG comprises the action potentials of all motor units recruited to perform a muscular contraction. The amplitude of the EMG pulse represents the number of motor units – the larger the number of motor units the higher the peak amplitude. The duration of the peak reflects the span of muscular contraction.

The measurement of ECG and EMG signals from the human skin surface involves electrodes, leads, instrumentation amplifier hardware, analog to digital conversion, data communication interface hardware and preprocessors. All this hardware, the environment and other electrical activities happening at the same time in the body, and various body movements, introduce noise and artifacts in the ECG and EMG signals – specifically when the test subject is in ambulatory state for long-term healthcare monitoring. These challenges are answered by designing the measurement equipment intelligently, and by developing advanced signal computational and processing techniques smartly.

This thesis is based on the development of data acquisition hardware that utilizes readily available off-the-shelf discrete and IC components for a single-lead ECG measurement setup and a muscular differential potential reader for EMG measurement setup. An additional sensor inertial measurement unit (IMU) comprising a 3-axis accelerometer, 3-axis gyroscope and 3-axis magnetometer was also brought onto same measurement board to record the test subject movement during ECG and EMG recording. A bluetooth-based wireless data communication interface was developed to transfer the data to a laptop in a real-time environment for recording and further processing. Various ambulatory events were designed and executed to collect data from the test subject for both ECG and EMG measurements.

The recorded data was then prepared and processed using MATLAB® R2017b version 9.3 to filter out the noisy components and extract the required parameters of the ECG and EMG waveforms. Both ECG and EMG signals were detrended to remove dc components, filtered with the S-G filter to remove high-frequency noise components and smooth out the waveforms and, finally, the analyzed motion artifacts with the IMU data. The ECG signal was further processed to remove the breathing, sit-stand and walking motion artifacts, and the Pan-Tompkins algorithm
was implemented to extract the R-peaks. The detected R-peaks were then filtered through a threshold filter and analyzed on raw collection of the ECG data. All the computational and algorithmic process results were presented in chapter 4 of this thesis.
7 Recommendations

The thesis work is a basic contribution towards the development of wearable biomedical electronics for wirelessly long-term ambulatory biomedical parameters monitoring, analysis, recording and processing in a real-time environment for digital healthcare. There is a huge potential for future development and improvements in this area of healthcare. One improvement is to develop three-leads ECG instrumentation for better demonstration of the heart’s electrical activity by covering the angles which are impossible in a single-lead measurement setup. It is recommended to use independent IMU on top of each electrode to develop a 3D model of the test subject in order to better visualize the local motion artifact and to develop a strategy to counteract this challenge.

The use of dry electrodes is another area of development to bring the comfort for the test subject for long-term monitoring, making it easier to bring biomedical devices to ordinary wearable garments. The miniaturization of the electronic equipment by pushing all the circuit onto a single chip, and the optimization of power consumption, is also important for long-term battery life, as is also the use of various recharging techniques that are implemented by harvesting energy from body movements. The flexible wiring and use of textile conductive material opens a whole new world of development in wearable biomedical instrumentation development. One provision could be faster data communication protocols for real-time data transmission using IoT networks and processing using centralized powerful processing units.
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