

# Summer Internship – 2022

## ECG Signal Processing

## ECG and PPG Module Design

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### ▪ Basic Questions:

- **What information do the leads of a 12-lead ECG provide?**

The 12-lead ECG gives a tracing from 12 different “electrical positions” of the heart. Each lead is meant to pick up electrical activity from a different position on the heart muscle. This allows an experienced interpreter to see the heart from many different angles. ([Link](#))

- **What information does the single-lead ECG provide?**

1-lead ECG (EKG) recorders are normally used primarily for basic heart monitoring, checking for various arrhythmias, or simple educational or research purposes, they can also be used for looking at the effects of exercise on the ECG. One-lead recorders can also be used to accomplish full 12-lead recordings in a sequential manner. ([Link](#))

- **What abnormalities can ECG show?**

1. your heart is beating too fast, too slow, or irregularly
2. you're having a heart attack or you've previously had a heart attack
3. you have heart defects, including an enlarged heart, a lack of blood flow, or birth defects
4. you have problems with your heart's valves
5. you have blocked arteries or coronary artery disease

Abnormal results can signify several issues. These include:

**Defects or abnormalities in the heart's shape and size:** An abnormal ECG can signal that one or more aspects of the heart's walls are larger than another meaning that the heart is working harder than normal to pump blood.

**Electrolyte imbalances:** Electrolytes are electricity-conducting particles in the body that help keep the heart muscle beating in rhythm. If your electrolytes are imbalanced, you may have an abnormal ECG reading.

**Heart attack or ischemia:** During a heart attack, blood flow in the heart is affected and heart tissue can begin to lose oxygen. This tissue will not conduct electricity as well, which can cause an abnormal ECG. Ischemia, or lack of blood flow, may also cause an abnormal ECG.

**Heart rhythm abnormalities:** A heart typically beats in a steady rhythm. An EKG can reveal if the heart is beating out of rhythm or sequence.

**Medication side effects:** Taking certain medications can impact a heart's rate and rhythm. Sometimes, medications given to improve the heart's rhythm can have the reverse effect and cause arrhythmias. Such as beta-blockers, sodium channel blockers, and calcium channel blockers. ([Link](#))

- **What are the applications of heart rate variability estimation?**

The most frequent application of HRV is connected to cardiological issues, most importantly to the monitoring of post-myocardial infarction patients and the prediction of sudden cardiac death. ([Link](#))

The heart is a key component of the human body, acting as a pump that transfers oxygenated and deoxygenated blood around the body. Like all other organs, it is susceptible to diseases and age. Heart rate variability (HRV) is the physiological phenomenon of variation in the time interval between heartbeats. It is measured by the variation in the beat- to-beat interval. Its variation may contain indicators of current disease, or warnings about impending cardiac diseases. The indicators may be present at all times or may occur at random-during certain intervals of the day. It is strenuous and time consuming to study and pinpoint abnormalities in voluminous data collected over several hours. Computer based analytical tools for in-depth study of data over daylong intervals can be very useful in diagnostics. In this paper we have discussed the various applications of HRV and different linear, frequency domain, wavelet domain, nonlinear techniques used for the analysis of the HRV. ([Paper's Link](#))

## ▪ Typical ECG Signal Processing:

- **Extraction of P-QRS-T wave and marking the points**

File name (codes) : **PQRST.m**

File name (results) : **Week03\_Results.mlx**

Example for result: **fig1 – PQRST waves detection**

**P-QRS-T Detection:**

```
[ind_P,ind_Q,ind_R,ind_S,ind_T] = PQRST(x_denoised,fs,file_name);
```

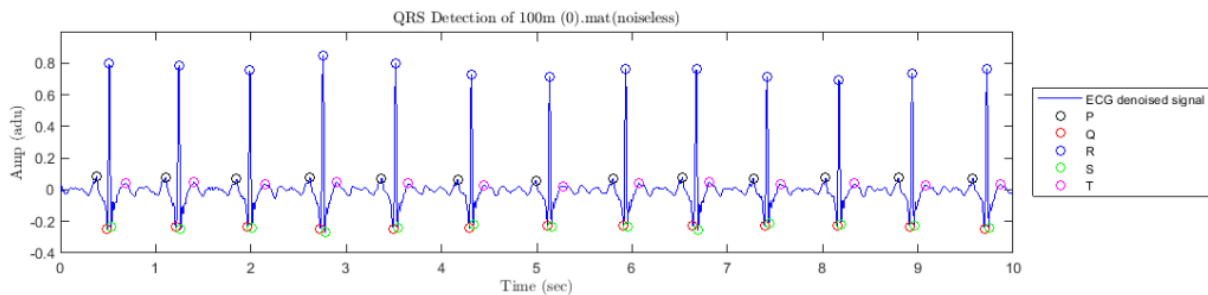


fig1 – PQRST waves detection

- **Detection of R-R and estimation of HRV**

Heart rate variability is the physiological phenomenon of variation in the time interval between heartbeats. It is measured by the variation in the beat-to-beat interval. Other terms used include: "cycle length variability", "R–R variability", and "heart period variability". ([Link 1](#)) , ([Link 2](#))

### 1. Heart-Rate Estimation:

Using average of RR-intervals we can estimate the Heart-Rate:

$$HR = \frac{60}{\overline{NN}}$$

Where  $NN$  is the normal inter-beat interval (the interval between consecutive R waves).

Function for Heart-Rate Estimation: File name (codes) : **HeartBeat.m**

Here is an Example for this estimation:

**R-peaks and Heart-Beat Detection:**

```
HB = HeartBeat(x_denoised,fs)
```

```
HB = 78.1665
```

## 2. RR-interval Scatter Plot:

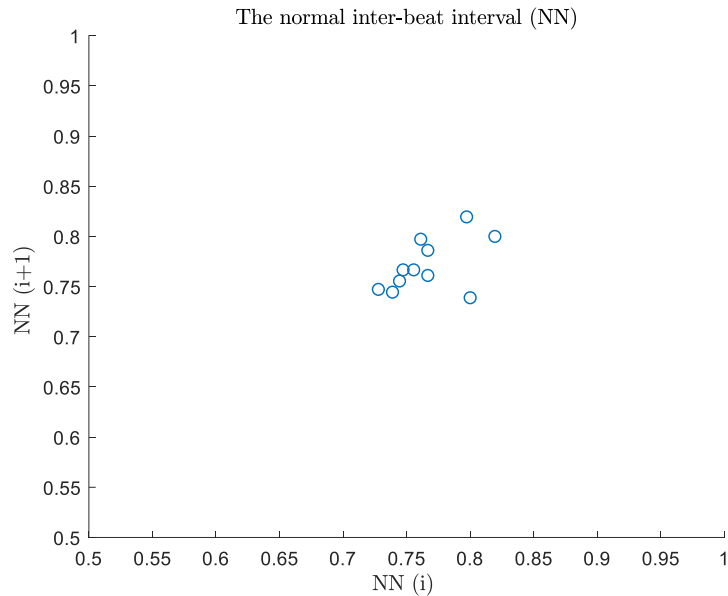


Fig2 – RR-interval Scatter Plot

- **What information can be obtained by applying wavelet transform on ECG?**

R-wave detection of ECG signal by using wavelet transform [\(Link\)](#)

Comparing different wavelet transforms on removing electrocardiogram baseline wanders and special trends [\(Link\)](#)

ECG data compression with wavelet and discrete cosine transforms [\(Link\)](#)

ECG Signal Denoising By Wavelet Transform Thresholding [\(Link\)](#)

ECG Signal Analysis and Arrhythmia Detection using Wavelet Transform [\(Link\)](#)

Application of Wavelet Techniques in ECG Signal Processing: An Overview [\(Link\)](#)

## ■ Pre-Processing of ECG Signal:

- Denoising the ECG signals
- Removing the baseline
- Removing the artifacts
- Omitting the abnormal spikes

File Name (Codes) : **Preprocessing.m**

### **Function 1: Ideal Filter**

```
function [x, x_denoised] = preprocessing(file_name,fs,plt)
    x = load(file_name);
    x = x.val;
    x = x - mean(x);
    x = x / max(x);
    dt = 1/fs;
    t = dt*(1:length(x));
    [x1,~] = highpass(x, 5, fs, 'ImpulseResponse','iir');
    [x2,~] = lowpass(x1, 35, fs, 'ImpulseResponse','iir');
    x_denoised = x2;
    if plt == "show"
        f = figure;
        f.Position = [10 10 1800 800];
        subplot(2,1,1);
        plot(t,x);
        xlabel('Time (sec)','Interpreter','latex');
        ylabel('Amp (adu)','Interpreter','latex');
        title(['ECG signal of ',file_name, '(noisy)'],'Interpreter','latex');
        subplot(2,1,2);
        plot(t,x_denoised);
        xlabel('Time (sec)','Interpreter','latex');
        ylabel('Amp (adu)','Interpreter','latex');
        title(['ECG signal of ',file_name, '(noiseless)'],'Interpreter','latex');
    elseif plt == "do not show"
    end
end
```

### **Function 2: Butterworth filter**

```
function [x, x_denoised] = preprocessing2(file_name,fs,plt)
    x = load(file_name);
    x = x.val;
    x = x - mean(x);
    x = x / max(x);
    dt = 1/fs;
    t = dt*(1:length(x));
    Fn = fs/2;
    Wp = [5 40]/Fn; % Passband
    Ws = [4 60]/Fn; % Stopband
    Rp = 5; % Passband Ripple
```

```

Rs = 20; % Stopband Ripple
[n,Wn] = buttord(Wp,Ws,Rp,Rs); % Butterworth Filter
Order
[b,a] = butter(n,Wn); % Butterworth Filter
Transfer Function Coefficients
[SOS,G] = tf2sos(b,a); % Convert to Second-
Order-Section For Stability
f1 = figure;
freqz(SOS, 4096, fs); % Assess Filter
close(f1)
x_denoised = filtfilt(SOS,G,x);
if plt == "show"
    f = figure;
    f.Position = [10 10 1800 800];
    subplot(2,1,1);
    plot(t,x);
    xlabel('Time (sec)','Interpreter','latex');
    ylabel('Amp (adu)','Interpreter','latex');
    title(['ECG signal of ',file_name, '(noisy)'],'Interpreter','latex');
    subplot(2,1,2);
    plot(t,x_denoised);
    xlabel('Time (sec)','Interpreter','latex');
    ylabel('Amp (adu)','Interpreter','latex');
    title(['ECG signal of ',file_name, '(noiseless)'],'Interpreter','latex');
elseif plt == "do not show"
end
end

```

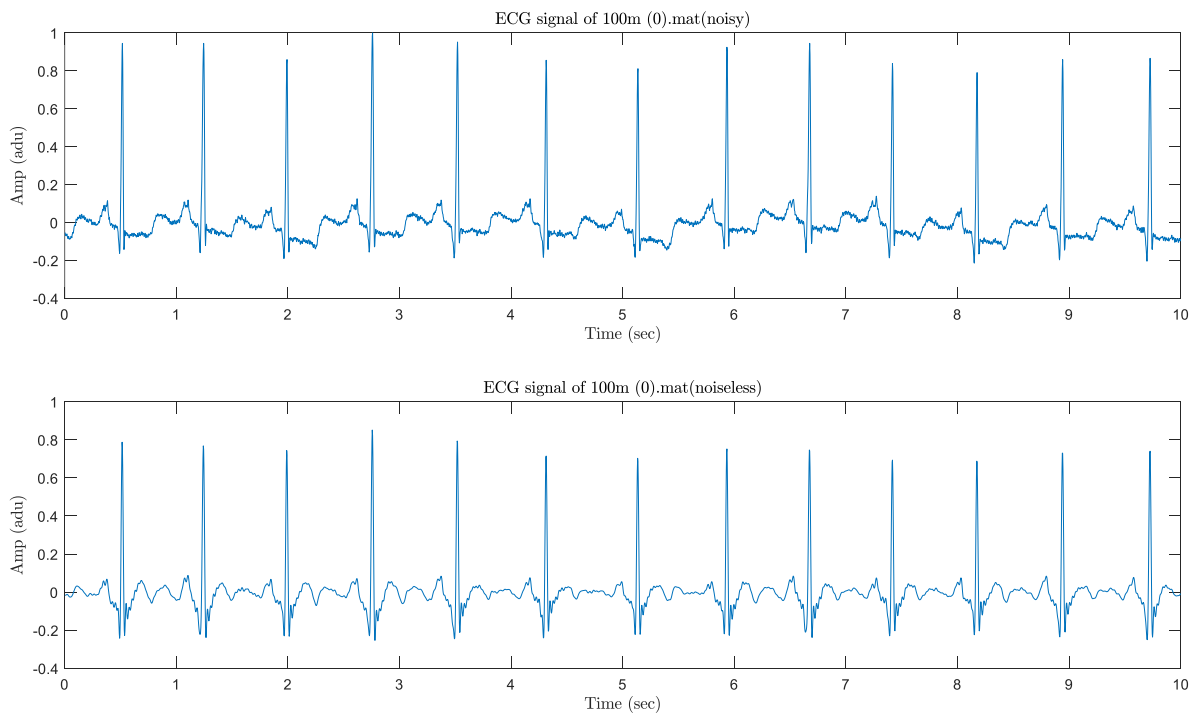
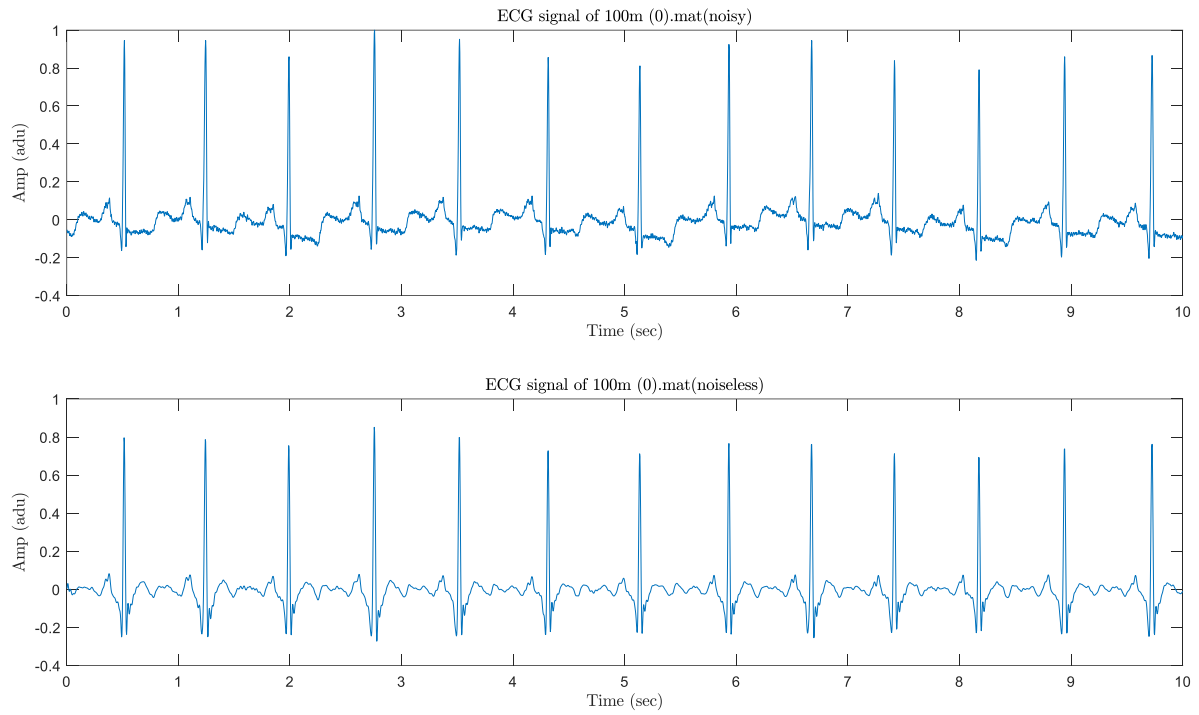


Fig3 – Pre-Processing with Function 1



**Fig4 – Pre-Processing with Function 2**

## ▪ Spike Sorting:

### Function: ECG\_BD.m

```
function [S_hat,alpha,tau,x_hat] = ECG_BD(x , L , K , file_name ,x_main)
    T = length(x);
    alpha = ones(K,1);
    tau = ((0:K-1)*floor((T-1)/K))'+1;
    idx_z = (0:(L-1)).'+(1:(T-L+1));
    Z = x(idx_z);
    itrMAX=10;
    obj_fun = zeros(1,itrMAX);
    for itr=1:10
        idx_y = tau'+(0:(L-1)).';
        Y = x(idx_y);
        S_hat = Y*(alpha);
        S_hat = S_hat/norm(S_hat,'fro');
        rho = S_hat.*Z;
        for k = 1:K
            [alpha(k),tau(k)] = max(abs(rho));
            rho(max(tau(k)-L+1,1):min(T-L+1,tau(k)+L-1)) = 0;
        end
        x_hat = zeros(1,T);
        idx_x = tau'+(0:(L-1)).';
        x_hat(idx_x) = S_hat*alpha';
        obj_fun(itr) = norm(x - x_hat , 'fro')/norm(x,'fro');
    end
    [tau,I] = sort(tau);
    alpha = alpha(I);
    er=obj_fun(end);
    figure
    subplot(3,1,1)
    plot(S_hat)
    xlim([0 length(x)]);
    title(['Spike of ',file_name, '(noisy)'],'Interpreter','latex');
    subplot(3,1,2)
    stem(tau,alpha,'^')
    xlim([0 length(x)]);
    title('time of spikes','Interpreter','latex');
    subplot(3,1,3)
    plot(x_main);
    xlim([0 length(x)]);
    hold on
    plot(x_hat,'r')
end
```

### • Implementation of BD method:

**Method 1:** We suppose that the number of spikes ( $K$ ) is equal with number of R-peaks. Then We estimate the length of each spike as follows:



$K = \text{Number of } R - \text{peaks}$

$$L = \frac{\text{length of ECG signal (number of samples)}}{K}$$

% Method 1

```
K = length(ind_R);
L = length(x_denoised)/K;
[spike,amp,t_amp,x_hat] = ECG_BD(x_denoised , L , K , file_name , x_denoised);
```

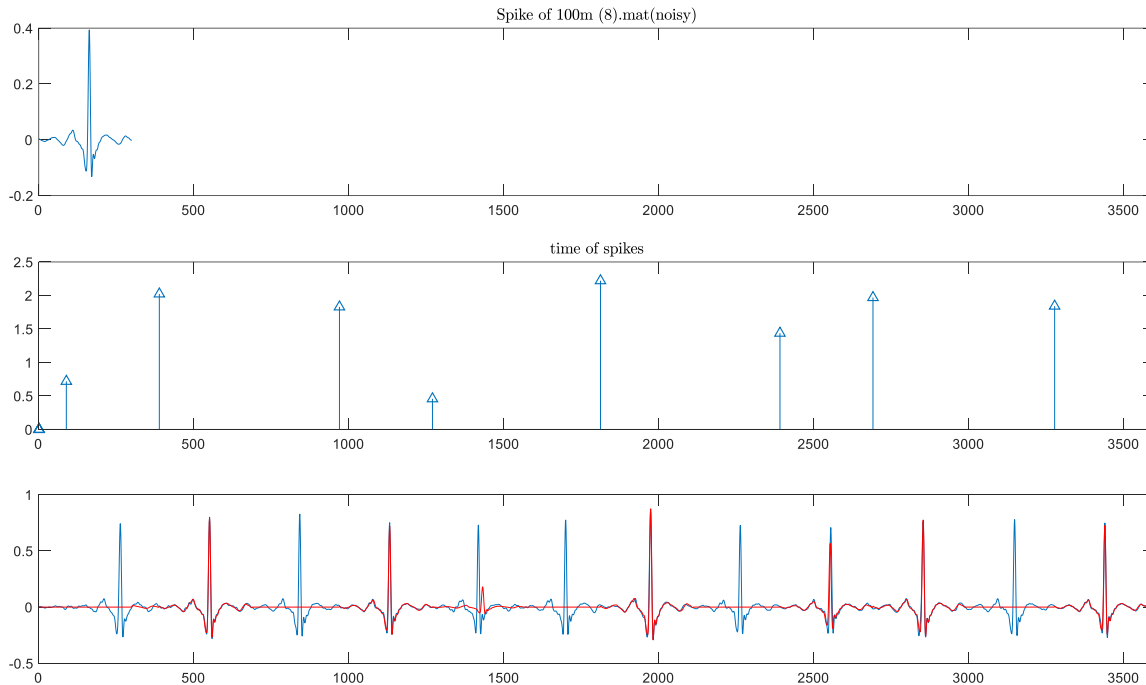


Fig5 – Implementation of BD (method 1)

**Method 2:** We suppose that the number of spikes ( $K$ ) is equal with number of  $R$ -peaks we use  $P$  and  $T$  waves to estimate the length of each spike. Then we call BD function (ECG\_BD) to implement BD methods.

% Method 2

```
K = length(ind_R);
for i = 2:K-1
    if ind_P(i)>ind_R(i-1) && ind_P(i)<ind_R(i)
        if ind_T(i)>ind_R(i) && ind_T(i)<ind_R(i+1)
            L = ind_T(i) - ind_P(i) + floor(5*((ind_P(i+1)-ind_T(i))+(ind_P(i)-ind_T(i-1)))/11);
            break
        end
    end
end
[spike,amp,t_amp,x_hat] = ECG_BD(x_denoised , L , K , file_name , x_denoised);
```

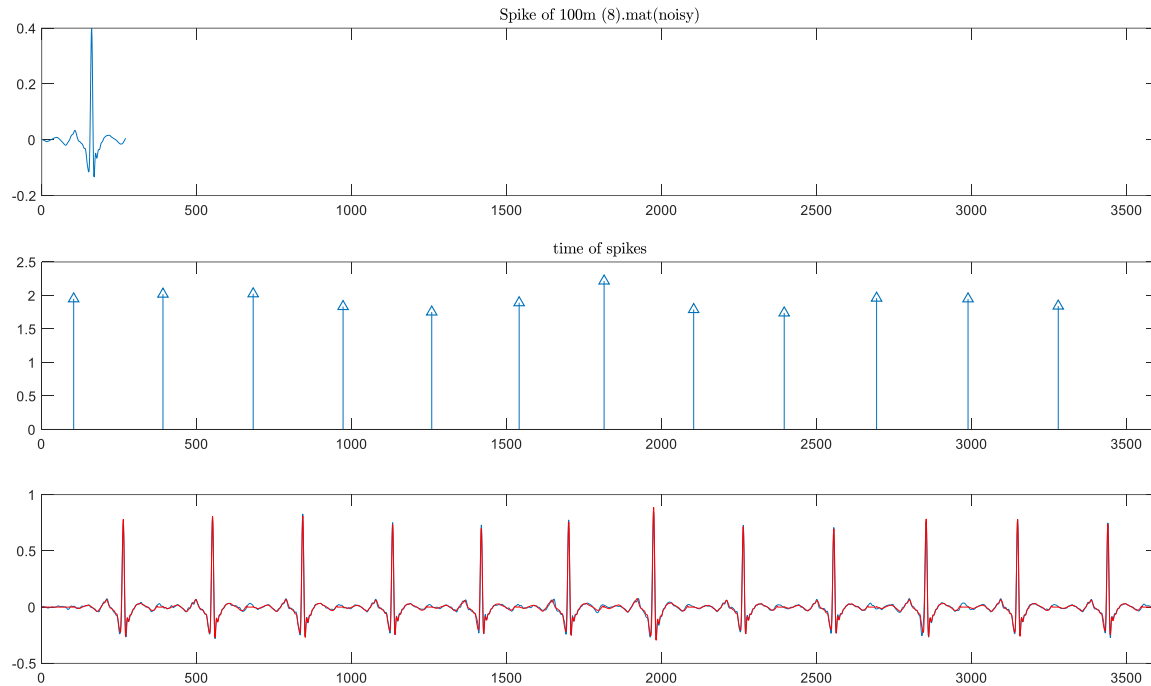


Fig6 – Implementation of BD (method 2)

**Method 3:** In this method after the preprocessing we try to delete the value of signal between last T-wave and next P-wave. Figure 7 shows the ECG signal after removing additions.

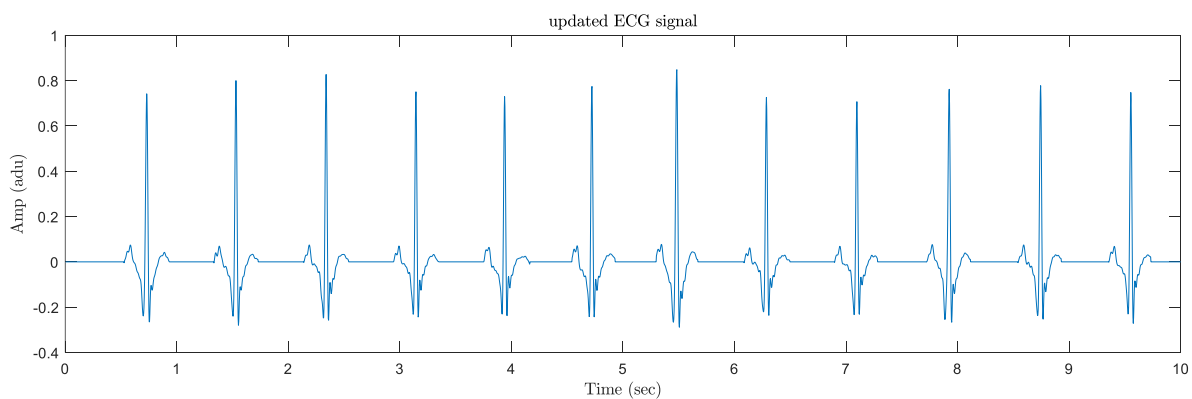


Fig7 – Removing additions of ECG signal

So we use a function to calculate the value of  $L$  and  $K$ . In this function we suppose that the number of spikes ( $K$ ) is equal with number of R-peaks we use  $P$  and  $T$  waves to estimate the length of each spike. Then we call another function (ECG\_BD) to implement BD methods.

**Function: *Spike\_Extraction.m***

```

function [spike,amp,t_amp,x_hat,PQRST_avg] = Spike_Extraction(x,fs,file_name)
    [ind_P,~,ind_R,~,ind_T] = PQRST(x,fs,file_name);
    dt = 1/fs;
    K = length(ind_R);
    z = x;
    if ind_P(1)<ind_R(1)
        if ind_T(1)>ind_R(1) && ind_T(1)<ind_R(2)
            z(1:floor(9*ind_P(1)/10)) = 0;
        end
    end
    for i = 2:K
        if ind_P(i)>ind_R(i-1) && ind_P(i)<ind_R(i)
            if ind_T(i)>ind_R(i)
                z(floor(ind_T(i-1)+(ind_P(i)-ind_T(i-1))/10):floor(ind_P(i)-(ind_P(i)-
ind_T(i-1))/10)) = 0;
            end
        end
    end
    for i = 2:K-1
        if ind_P(i)>ind_R(i-1) && ind_P(i)<ind_R(i)
            if ind_T(i)>ind_R(i) && ind_T(i)<ind_R(i+1)
                L = ind_T(i) - ind_P(i) + floor(5*((ind_P(i+1)-ind_T(i))+(ind_P(i)-ind_T(i-
1)))/11));
            end
        end
    end
    if ind_T(end)>ind_R(end)
        z(floor(ind_T(end)+(length(z)-ind_T(end))/10):end) = 0;
    end
    [spike,amp,t_amp,x_hat] = ECG_BD(z , L , K , file_name , x);
    [~,j] = max(spike);
    s = spike(1:j);
    ind = find(s == 0);
    [~,i] = max(ind);
    ind = find(s == 0);
    PQRST_avg = spike(i:end);
    t_spike = dt*(1:length(PQRST_avg));
    figure
    plot(t_spike,PQRST_avg);
    xlabel('Time (sec)','Interpreter','latex');
    ylabel('Amp (adu)','Interpreter','latex');
    title('Average spike of ECG signal','Interpreter','latex');
end

```

**% Method 3**

```

[~,x1] = preprocessing('100m (1).mat',fs,"do not show");
Spike_Extraction(x1,fs,'100m (1).mat');

```

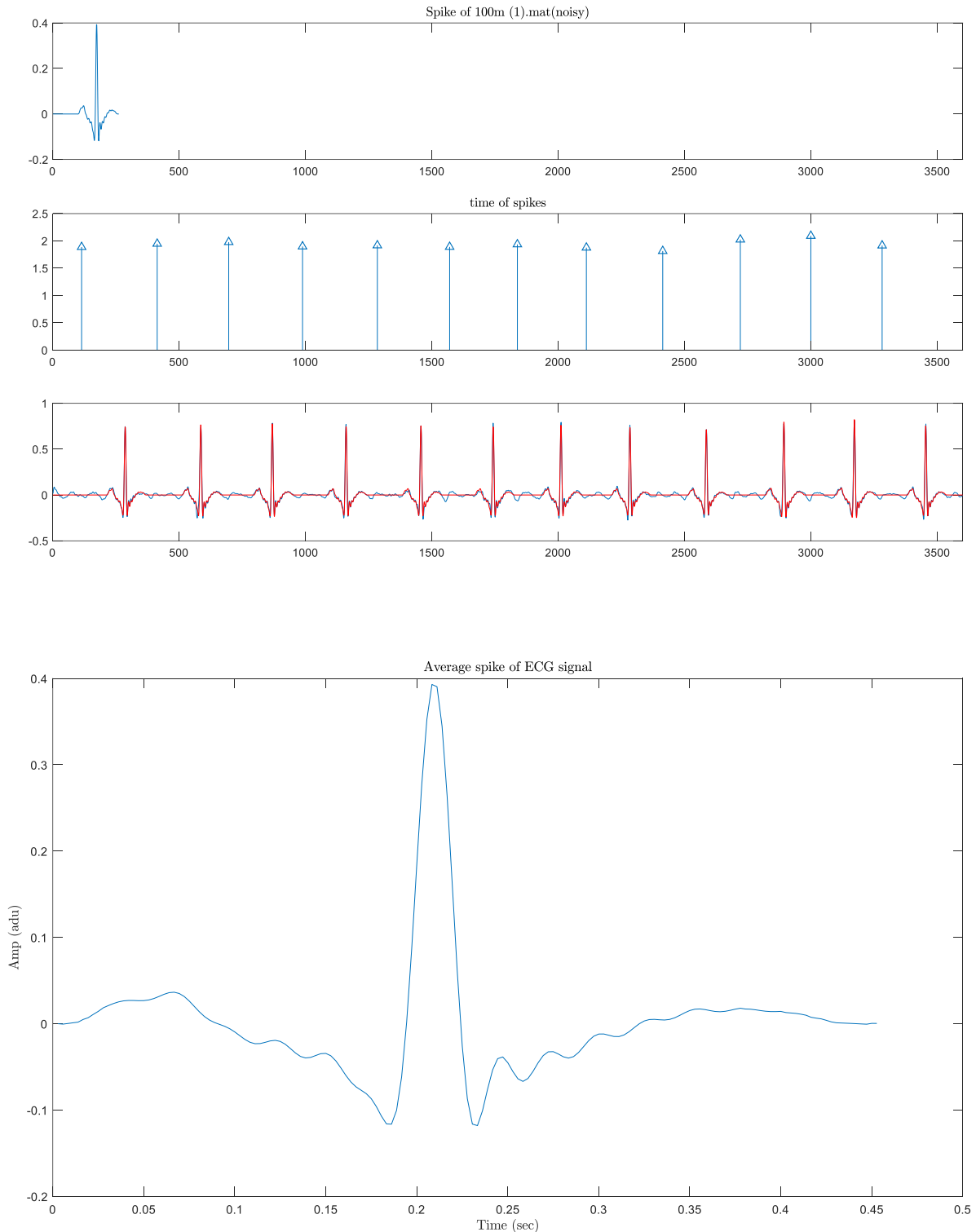
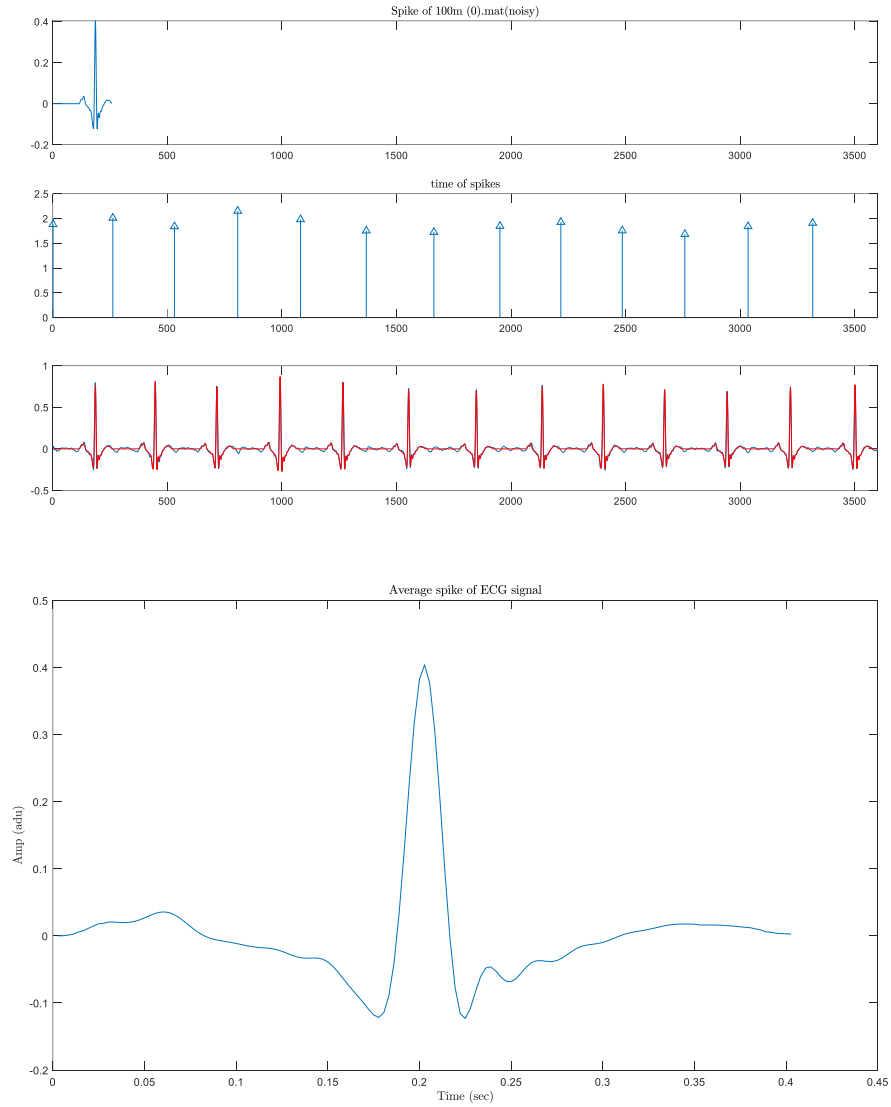


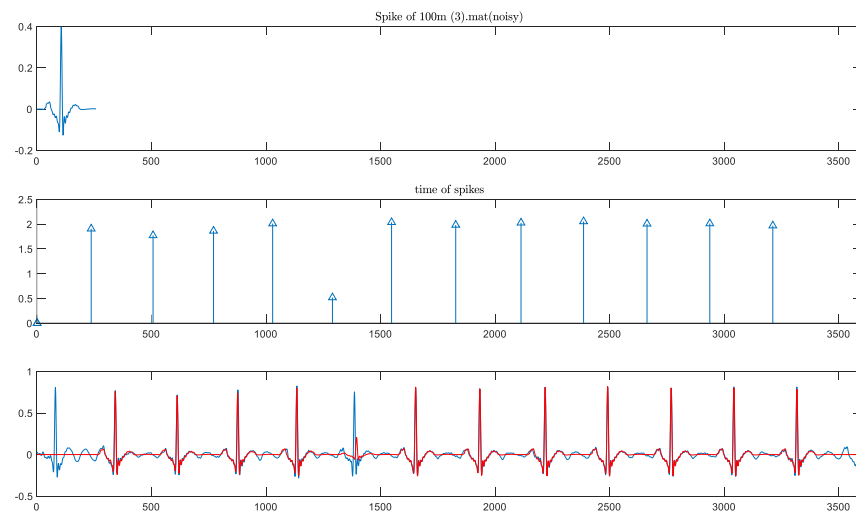
Fig8 – Implementation of BD (method 3)

- **Applying the method on different ECG channels**

Here we apply method 3 on different ECG signals:



**Fig9 – Implementation of BD - method 3 "100m (0).mat"**



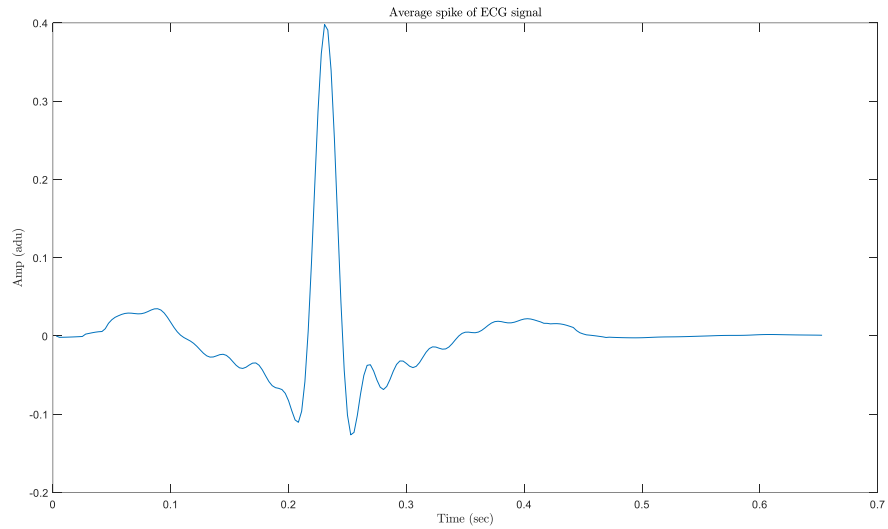


Fig10 – Implementation of BD - method 3 "100m (3).mat"

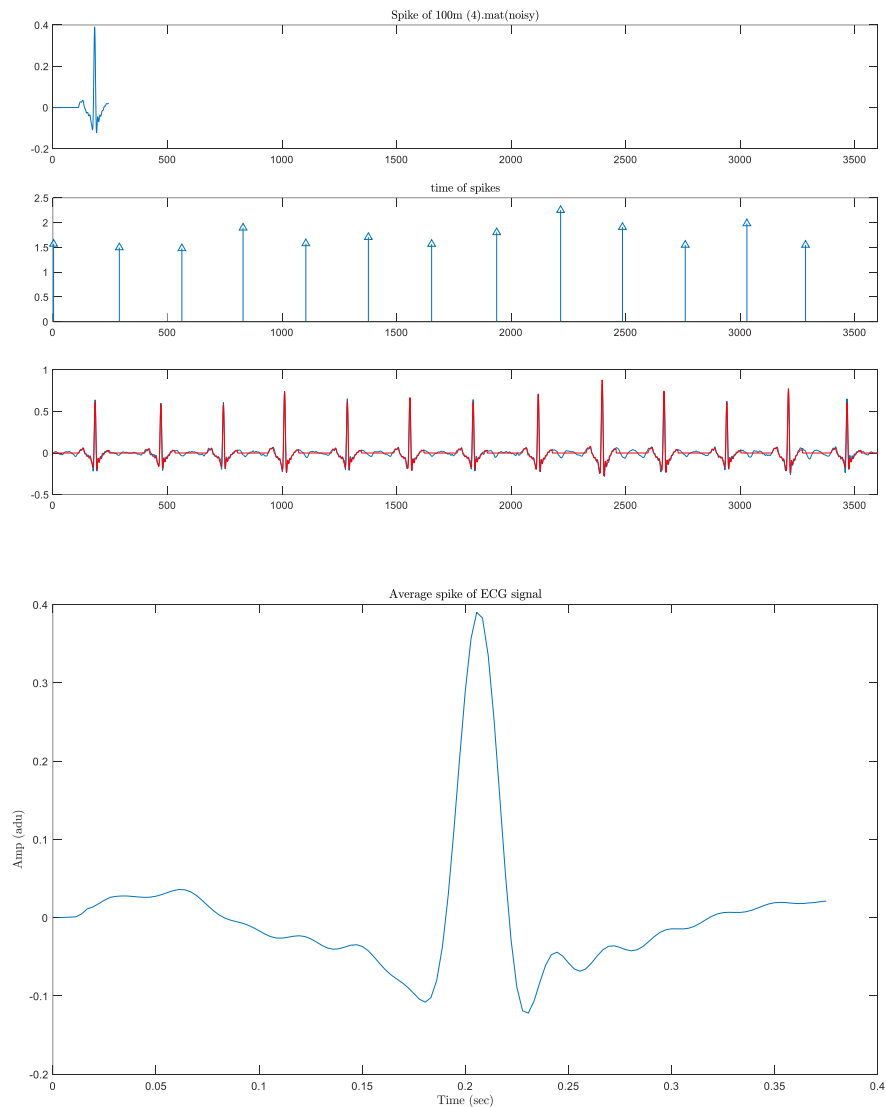
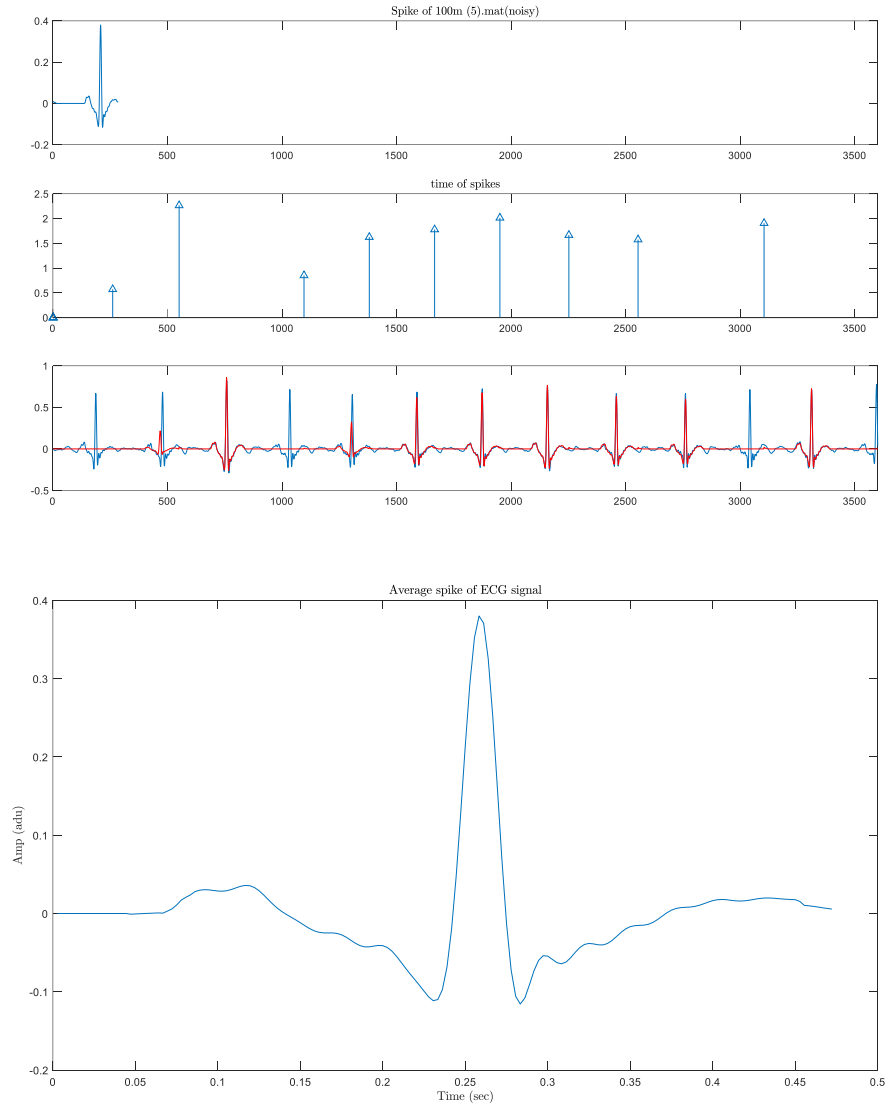
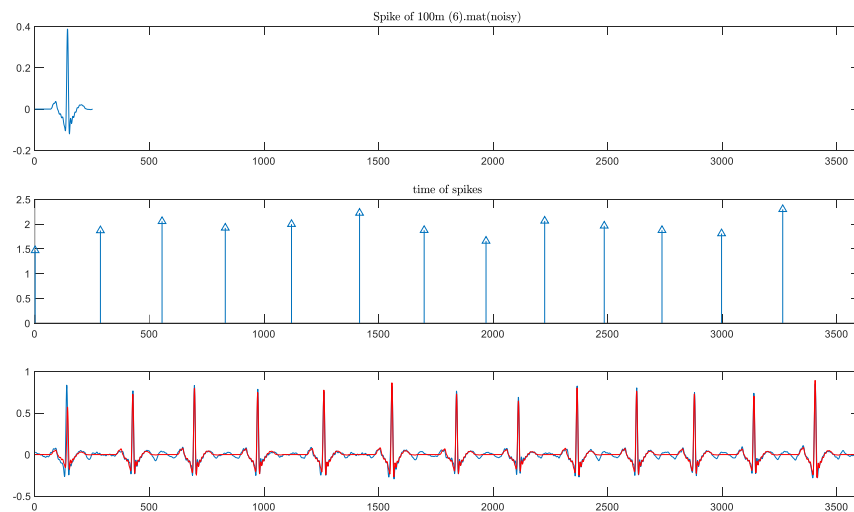
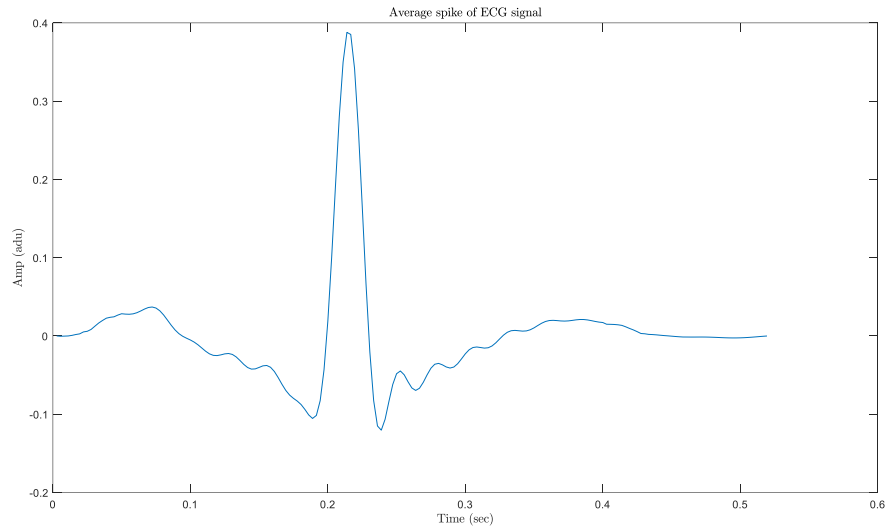


Fig11 – Implementation of BD - method 3 "100m (4).mat"

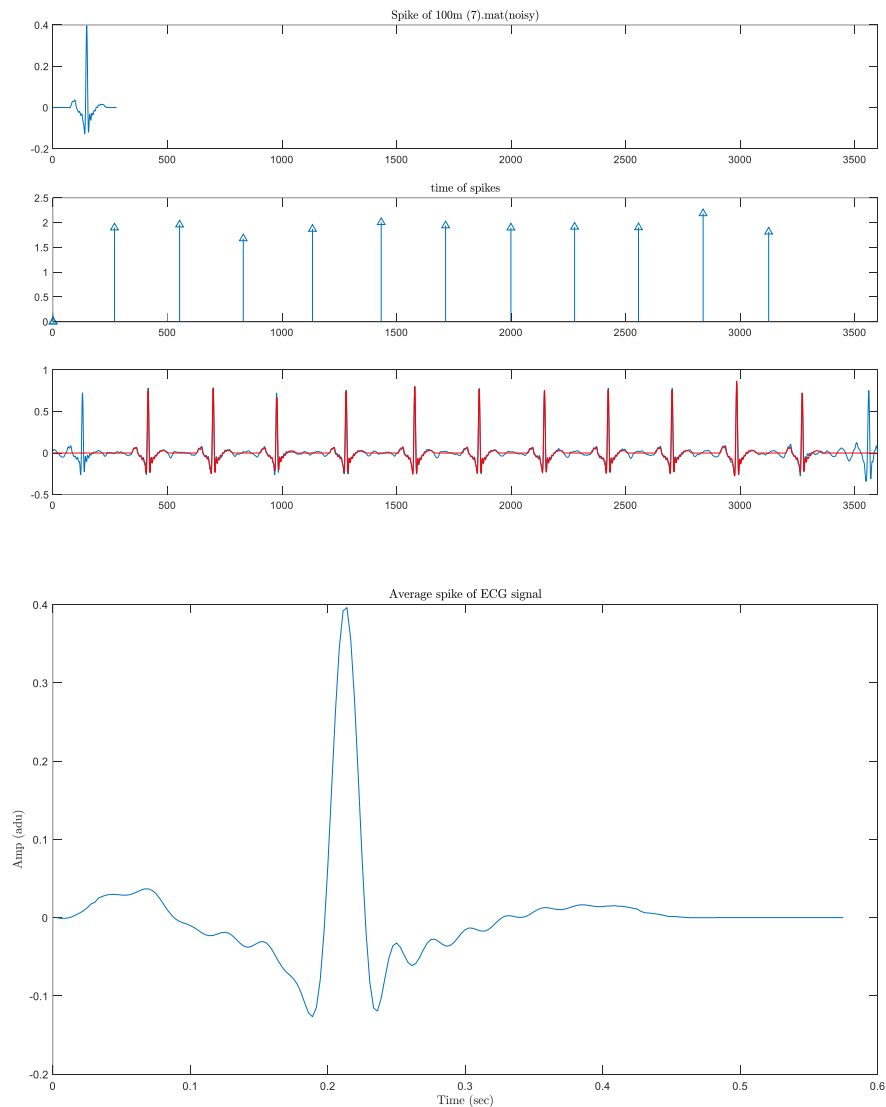


**Fig12 – Implementation of BD - method 3 "100m (5).mat"**





**Fig13 – Implementation of BD - method 3 "100m (6).mat"**



**Fig14 – Implementation of BD - method 3 "100m (7).mat"**



- ***Comparison of the extracted spike in different abnormalities***

## ■ ECG signal processing on a real ECG dataset (MAX86150):

In this part we want to implement above methods on an output signal of MAX86150 evaluation board.

There is a dataset ("log8.csv") that includes about 120 seconds ECG and PPG signals. First we show the raw signal in Figure 15. As we see there are two peaks that are not for ECG signal. So we use the 10-20 seconds part of signal.

```
file_name = 'log8.csv';
data = readtable(file_name);
data = rmmissing(data);
x = data.Var2;
tt = data.Var1;
T = seconds(tt(end)-tt(1));
fs = length(x)/T;
dt = 1/fs;
t = dt * (1:length(x));
f = figure;
f.Position = [10 10 1800 400];
plot(t,x);
xlabel('Time (sec)','Interpreter','latex');
ylabel('Amp (adu)','Interpreter','latex');
title(['ECG signal of ',file_name, '(noisy)'],'Interpreter','latex');
```

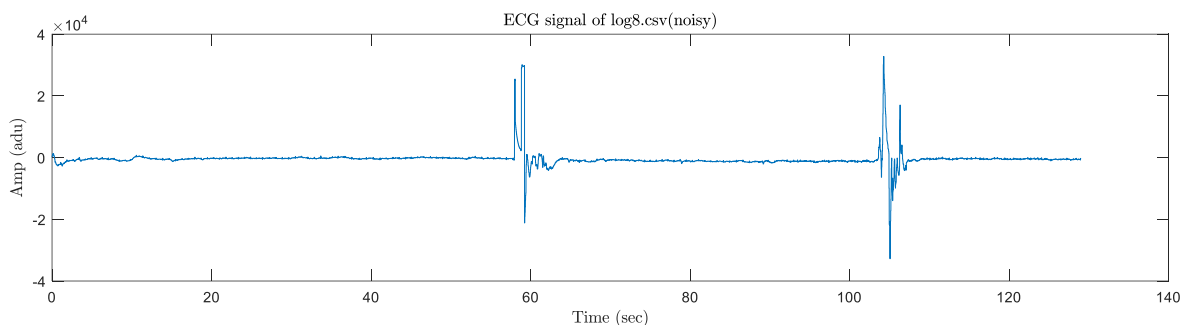


Fig15 – Raw ECG signal "log8.csv"

```
x = x(floor(10*fs):floor(20*fs));
t = t(floor(10*fs):floor(20*fs));
```

Then we preprocess the signal using ideal filter. We change some parts of preprocessing function. Final function is as follows:

```
function x_denoised = preprocessing3(x,fs,plt,file_name)
dt = 1/fs;
t = dt*(1:length(x));
[x1,~] = highpass(x, 5, fs, 'ImpulseResponse','iir');
[x2,~] = lowpass(x1, 35, fs, 'ImpulseResponse','iir');
x_denoised = x2;
```

```

x_denoised = x_denoised - mean(x_denoised);
x_denoised = x_denoised / max(abs(x_denoised));
if plt == "show"
    f = figure;
    f.Position = [10 10 1800 800];
    subplot(2,1,1);
    plot(t,x);
    xlabel('Time (sec)','Interpreter','latex');
    ylabel('Amp (adu)','Interpreter','latex');
    title(['ECG signal of ',file_name, '(noisy)'],'Interpreter','latex');
    subplot(2,1,2);
    plot(t,x_denoised);
    xlabel('Time (sec)','Interpreter','latex');
    ylabel('Amp (adu)','Interpreter','latex');
    title(['ECG signal of ',file_name, '(noiseless)'],'Interpreter','latex');
elseif plt == "do not show"
end
end

```

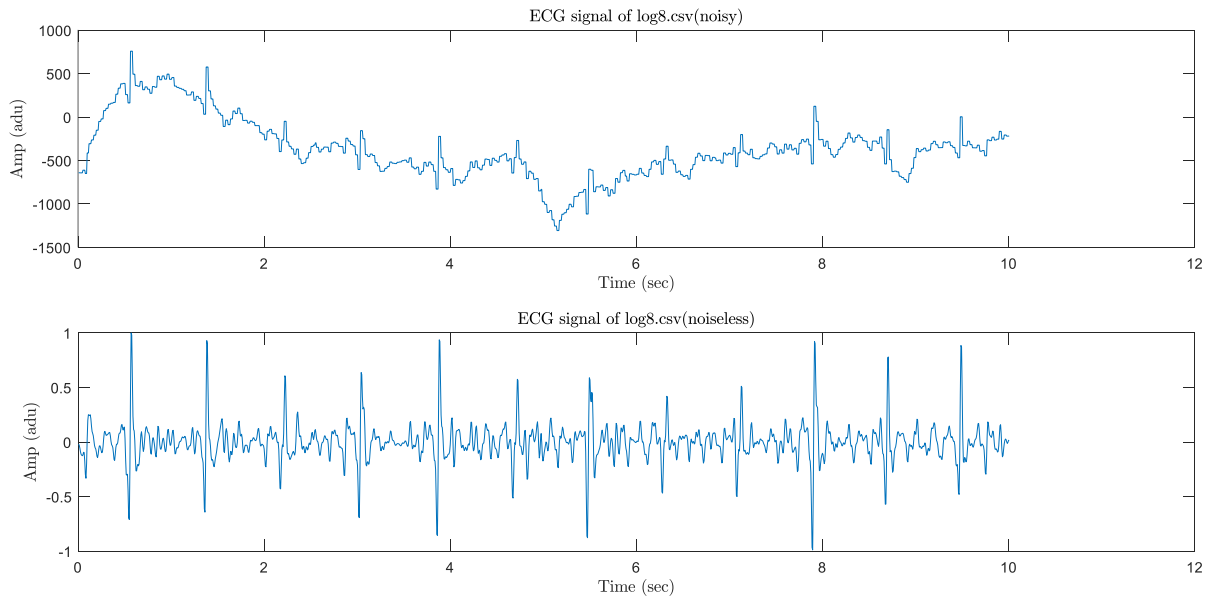


Fig16 – Preprocessed ECG signal "log8.csv"

Now we update the HeartBeat() and PQRST() function as follows:

#### R-peaks and Heart-Beat Detection Function:

```

function [HB] = HeartBeat(x,fs)
    dt = 1/fs;
    t = dt*(1:length(x));
    thr = (max(x) + mean(x)) / 3;
    [~,ind] = findpeaks(x,'MinPeakHeight',thr);
    peak_time = t(ind);
    NN = peak_time(2:end) - peak_time(1:end-1);
    HB = 60 / mean(NN);
end

```

**P-QRS-T Detection Function:**

```

function [ind_P,ind_Q,ind_R,ind_S,ind_T] = PQRST(x,fs,file_name)
    ind_P = []; ind_Q = []; ind_S = []; ind_T = [];
    dt = 1/fs;
    t = dt * (1:length(x));
    %% R detection
    z = x;
    pos = z .* (z>0);
    thr_R = (max(pos) + mean(pos)) / 3;
    [peaks,ind_R] = findpeaks(pos,'MinPeakHeight',thr_R);
    NN = ind_R(2:end) - ind_R(1:end-1);
    ou = find(isoutlier(NN,'mean'));
    if ou
        ind_R = ind_R(peaks ~= min([x(ind_R(ou)),x(ind_R(ou+1))])).';
    end
    %% Q, S detection
    neg = -(z .* (z<0));
    [~,ind1] = max(neg(10:ind_R(1)));
    ind_Q = [ind_Q ,10+ind1-1];
    for i = 1:length(ind_R)-1
        t1 = ind_R(i); t2 = ind_R(i+1); t_diff = t2-t1; t_mean = floor(t_diff/5);
        [~,ind1] = max(neg(t2-t_mean:t2));
        ind_Q = [ind_Q ,t2-t_mean+ind1-1];
        [~,ind2] = max(neg(t1:t1+t_mean));
        ind_S = [ind_S ,t1+ind2-1];
    end
    [~,ind2] = max(neg(ind_R(end):end-10));
    ind_S = [ind_S ,ind_R(end)+ind2-1];

    %% P, T detection
    for i = ind_R
        u = abs(i - ind_Q);
        [~,j1] = min(u);
        for j = ind_Q(j1):-1:1
            if abs(z(j)) <= 0.1
                break
            end
            z(j) = 0;
        end
        v = abs(i - ind_S);
        [~,j2] = min(v);
        for j = ind_S(j2):1:length(z)
            if abs(z(j)) <= 0.1
                break
            end
            z(j) = 0;
        end
        for j = ind_Q(j1):1:ind_S(j2)
            z(j) = 0;
        end
    end
    pos = z .* (z>0);

```

```

[~,ind1] = max(pos(10:ind_R(1)));
ind_P = [ind_P ,10+ind1-1];
for i = 1:length(ind_R)-1
    t1 = ind_R(i); t2 = ind_R(i+1);
    t_mean = floor((2*t1+t2)/3);
    [~,ind1] = max(pos(t1+1:t_mean));
    ind_T = [ind_T ,t1+ind1-1];
    t_mean = floor((2*t2+t1)/3);
    [~,ind2] = max(pos(t_mean:t2));
    ind_P = [ind_P ,t_mean+ind2-1];
end
[~,ind2] = max(pos(ind_R(end):end-10));
ind_T = [ind_T ,ind_R(end)+ind2-1];

% Plot
f = figure;
f.Position = [10 10 2000 400];

plot(t,x,'b',t(ind_P),x(ind_P),'ko',t(ind_Q),x(ind_Q),'ro',t(ind_R),x(ind_R),'bo',t(ind_S),x(
ind_S),'go',t(ind_T),x(ind_T),'mo');
legend('ECG denoised signal','P','Q','R','S','T','Location','EastOutside');
xlabel('Time (sec)','Interpreter','latex');
ylabel('Amp (adu)','Interpreter','latex');
title(['QRS Detection of ',file_name, '(noiseless)'],'Interpreter','latex');
end

```

### R-peaks and Heart-Beat Detection: (Preprocessed using Ideal filter)

```
HB = HeartBeat(x_denoised,fs)
```

```
HB = 80.7373
```

### P-QRS-T Detection: (Preprocessed using Ideal filter)

```
[ind_P,ind_Q,ind_R,ind_S,ind_T] = PQRST(x_denoised,fs,file_name);
```

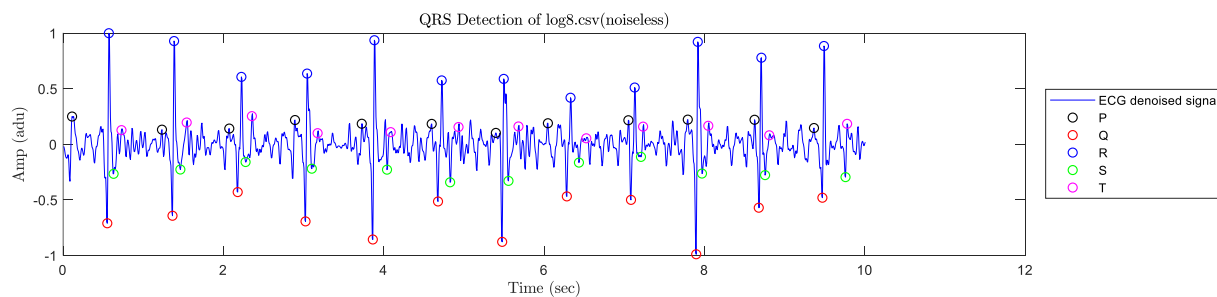


Fig17 – find P-QRS-T waves of ECG signal "log8.csv"

We use another dataset "log9.csv":

```

file_name = 'log9.csv';
data = readtable(file_name);
data = rmmissing(data);
x = data.FilteredECG;
t_min = minute(data.x__Time);

```

```
t_sec = second(data.x__Time);
T = 60 * (t_min(end)-t_min(1)) + (t_sec(end)-t_sec(1));
fs = length(x)/T;
dt = 1/fs;
t = dt * (1:length(x));
f = figure;
f.Position = [10 10 1800 400];
plot(t,x);
xlabel('Time (sec)','Interpreter','latex');
ylabel('Amp (adu)','Interpreter','latex');
title(['ECG signal of ',file_name, '(noisy)','Interpreter','latex');
```

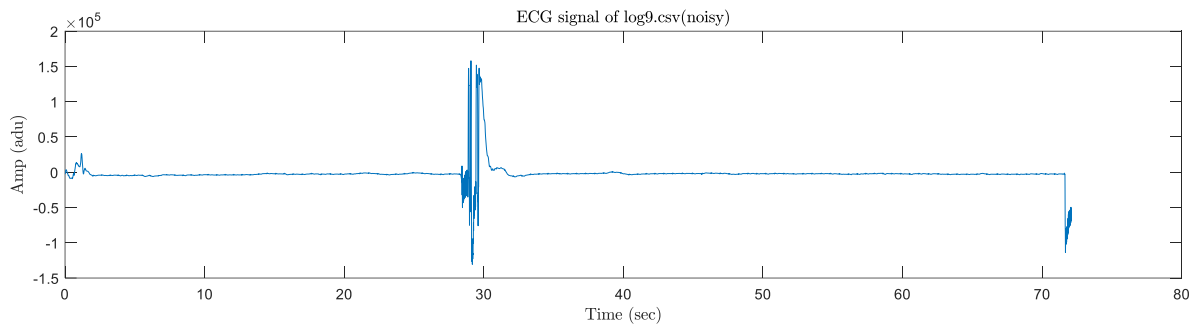


Fig18 – Raw ECG signal "log9.csv"

```
x = x(floor(10*fs):floor(20*fs));
t = t(floor(10*fs):floor(20*fs));
```

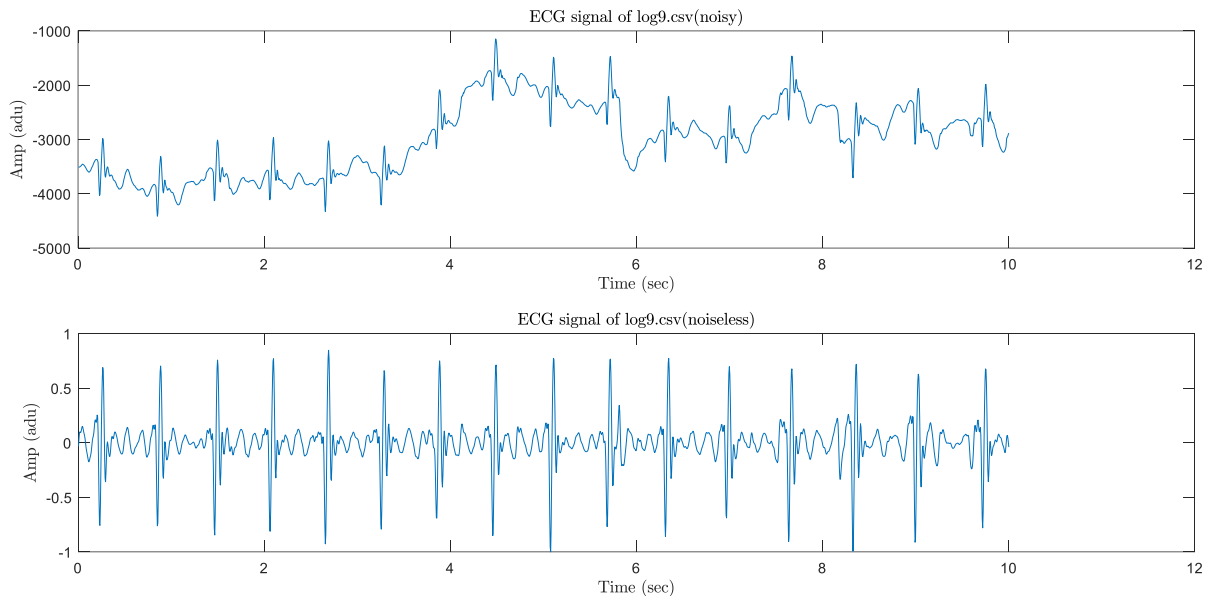


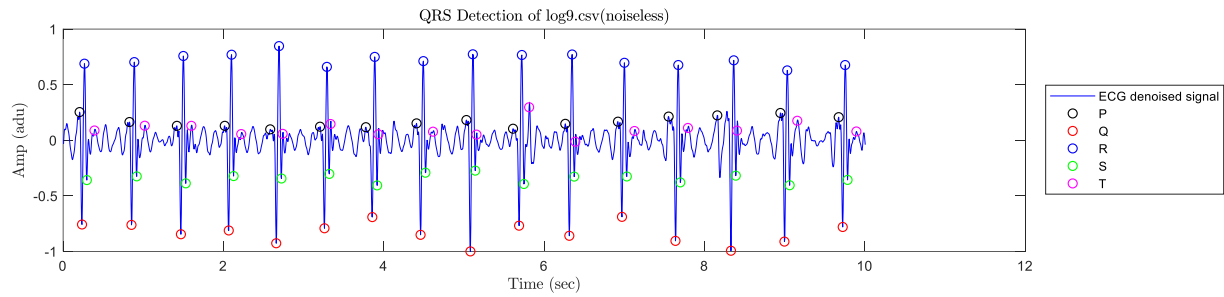
Fig19 – Preprocessed ECG signal "log9.csv"

### R-peaks and Heart-Beat Detection: (Preprocessed using Ideal filter)

```
HB = HeartBeat(x_denoised,fs)
HB = 101.1955
```

### P-QRS-T Detection: (Preprocessed using Ideal filter)

```
[ind_P,ind_Q,ind_R,ind_S,ind_T] = PQRST(x_denoised,fs,file_name);
```



**Fig20 – find P-QRS-T waves of ECG signal "log9.csv"**

## ■ ECG Lead 1:

- **Finding all of the information extracted from Lead 1** ([Link](#))

**12-lead ECG:** This chapter presents an introduction to the 12-lead ECG. The 12-lead ECG gives a tracing from 12 different “electrical positions” of the heart. Each lead is meant to pick up electrical activity from a different position on the heart muscle. This allows an experienced interpreter to see the heart from many different angles. This section is meant only as an introduction to the 12-lead ECG. It will take much practice of you to be able to interpret a 12-lead ECG tracing. This section will give you a basic understanding of how to take a 12-lead EKG, how to place the leads, and how to begin to interpret the tracing.

The electrocardiogram is a graphic record of the direction and magnitude of the electrical activity generated by the depolarization and repolarization of the atria and ventricles of the heart. This electrical activity is readily detected by electrodes attached to the skin. However, neither the electrical activity that results from the generation and transmission of electrical impulse, nor the mechanical contractions or relaxations of the atria and ventricles appear in the electrocardiogram.

An EKG lead consists of two surface electrodes of opposite polarity (one positive and one negative) or one positive surface electrode and a reference point. A lead composed of two electrodes of opposite polarity is called bipolar lead. A lead composed of a single positive electrode and a reference point is a unipolar lead.

For a routine analysis of the heart’s electrical activity an ECG recorded from 12 separate leads is used. A 12-lead ECG consists of three bipolar limb leads (I, II, and III), the unipolar limb leads (AVR, AVL, and AVF), and six unipolar chest leads, also called precordial or V leads, (V<sub>1</sub>, V<sub>2</sub>, V<sub>3</sub>, V<sub>4</sub>, V<sub>5</sub>, V<sub>6</sub>).

**Limb leads:** I, II, III, IV, V, and VI

Lead IV also called AVR

Lead V also called AVL

Lead VI also called AVF

**Chest leads:** V<sub>1</sub>, V<sub>2</sub>, V<sub>3</sub>, V<sub>4</sub>, V<sub>5</sub>, and V<sub>6</sub>

Below is a sample of a 12-lead EKG tracing.



### **Location of the Frontal Plane Axis:**

In order to accurately interpret the 12-lead EKG, you must have an understanding of the electrical activity of the heart. The direction in which the impulses flow in the heart is important. It is also important to understand that 12 different leads pick up those impulses as they travel in many different directions through the heart.

### **Definition:**

The frontal plane axis is the orientation of the heart's electrical activity in the frontal plane.

The frontal plane consists of: (Figure 21)

Right-to-Left	/	Left-to-Right Directions
Superior-to-Inferior	/	Inferior-to-Superior Directions

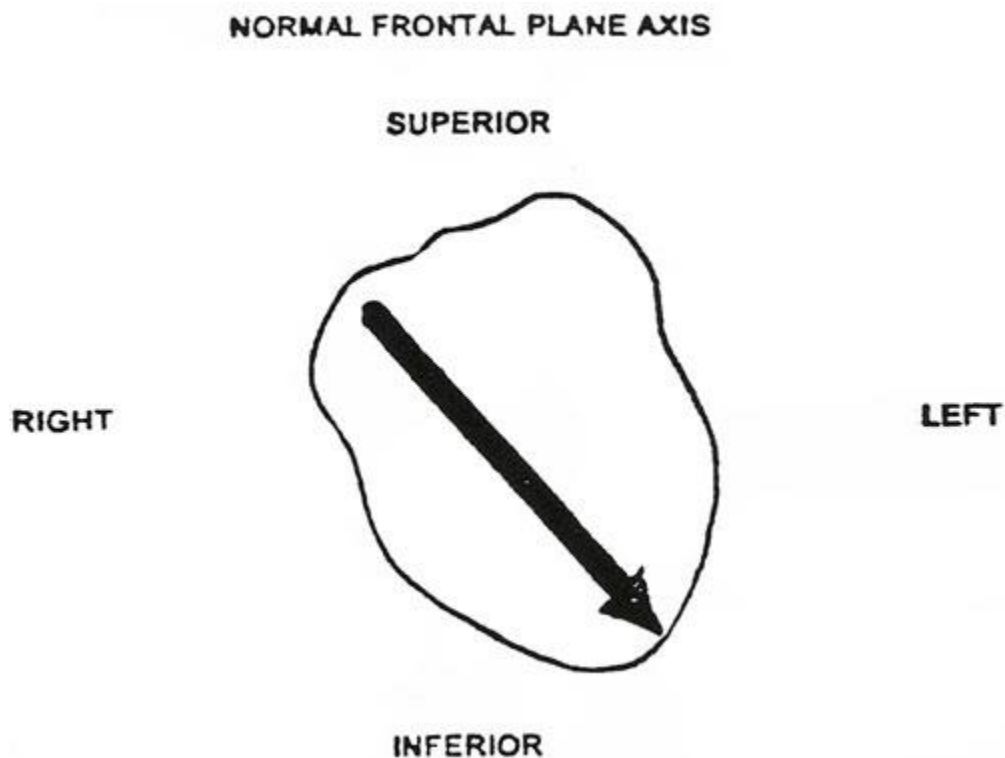


Fig21 – Normal Frontal Plane Axis

### **The Frontal Plane Leads:**

The activity of the heart produces electrical potentials that can be measured on the surface of the skin. Using the galvanometer (EKG machine), differences between electrical potentials at different sites of the body can be recorded. See illustration below: (Figure 22)

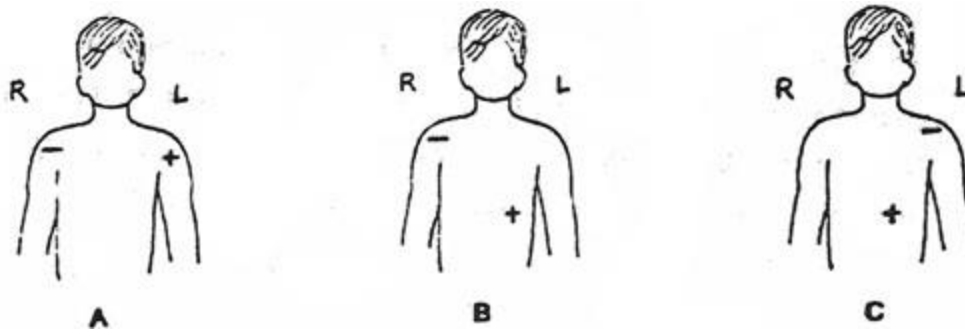


Fig22 – The Frontal Plane Leads

In picture A above, the negative electrode is on the right arm and the positive electrode is on the left arm. This is **lead I**. Lead I records electrical difference between the left and right arm electrodes.

In picture B above, the negative electrode is on the right arm and the positive electrode is on the left leg (left lower chest). This is **lead II**. Lead II records electrical differences between the left leg and right arm electrodes.

In picture C, the negative electrode is on the left arm and the positive electrode is on the left leg (left lower chest). Picture C depicts **lead III**. Lead III records electrical difference between the left leg and the left arm electrodes.

The above illustration shows Leads I, II, and III, their placement and the electrical potential on these three leads.

They are summarized as follows:

**Lead I: Right arm-negative, Left arm-positive**

Records electrical differences between the left and right arm electrodes.

**Lead II: Right arm-negative, Left leg-positive**

Records electrical difference between the left leg and right arm electrodes.

**Lead III: Left arm-negative, Left leg-positive**

Records electrical differences between the left leg and left arm electrodes.

The other three frontal plane limb leads are called the augmented Vector leads. The Galvanometer (EKG machine) records potential differences and, therefore, the technique is Bipolar (potential site A minus potential site B).

However, if the potential of B is zero the recorder records only the potential site A. this means that these next three electrodes, for all practical purposes have a zero potential and do not change during the cardiac cycle. They became known as the V electrodes, and all three leads became known as the V electrodes or UNIPOLAR leads.

As mentioned earlier, unipolar leads measure the electric impulses at only one point, instead of across two points, as the first three leads. With these V leads, the second site is -0- so there is no-need to measure from two pointes, only one point is needed. To obtain the measurements from these V leads, you simply turn the dial on the EKG machine to AVR, AVL, and AVF, respectively. The machine automatically makes the needed connection to measure the voltage from these areas.

The illustrations below show where these measurements take place.

They are summarized here:

- **Lead AVR** Augmented Vector Right, positive electrode right shoulder.
- **Lead AVL** Augmented Vector Left, positive electrode left shoulder.
- **Lead AVF** Augmented Vector Foot, positive electrode on Food.

**AVR** means augmented Vector Right; the positive electrode is on the right shoulder. (Figure 23)

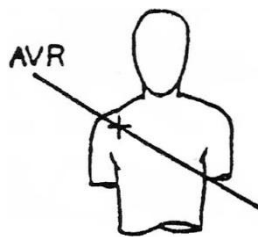


Fig23 – Lead AVR

**AVL** means augmented Vector Left; the positive electrode is on the left shoulder. (Figure 24)

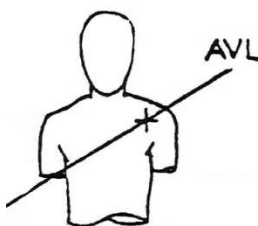


Fig24 – Lead AVL

**AVF** means augmented Vector Foot; the positive electrode is on the foot. (Figure 25)



Fig25 – Lead AVL

Now combine the three limb leads I, II, III and the three augmented Vector leads AVR, AVL, AVF: (Figure 26)

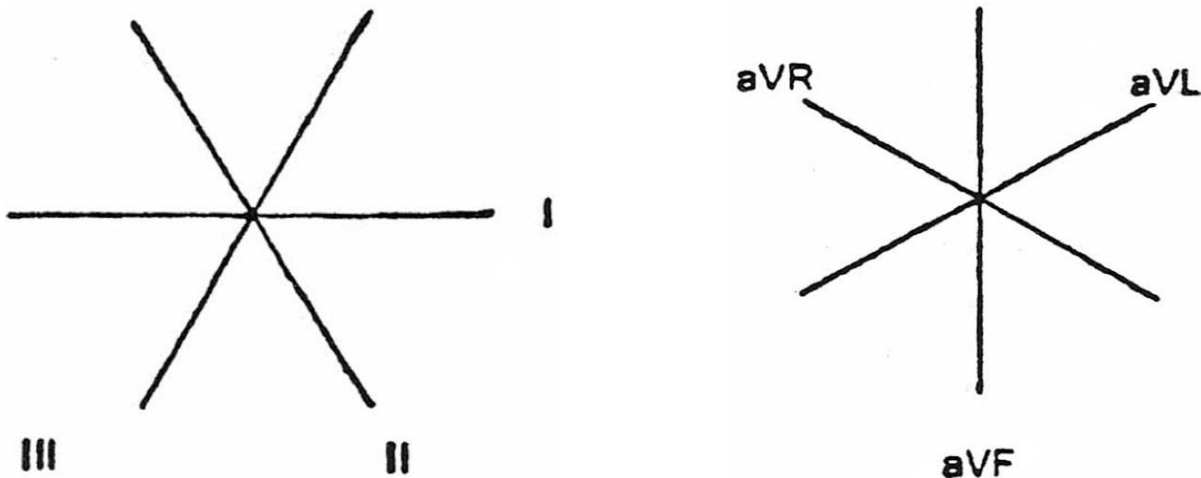


Fig26 – The three limb leads and the three augmented Vector leads

and this combination creates the **Hexaxial Reference System**: (Figure 27)

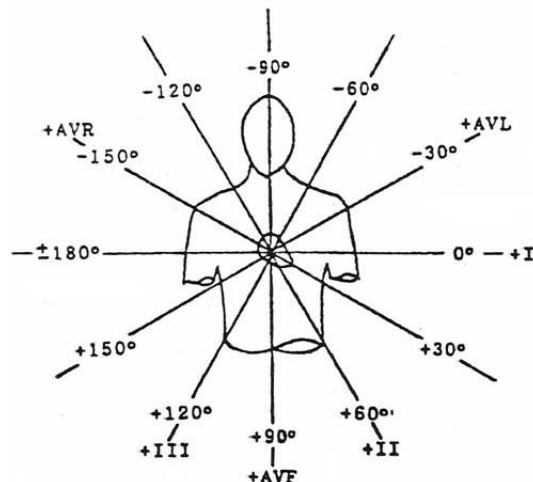


Fig27 – The Hexaxial Reference System

The angles are as follows:

**LEAD I** is located at 0 degrees and (+) (-) 180 degrees.

**LEAD II** is located at +60 degrees and -120 degrees.

**LEAD III** is located at +120 degrees and -60 degrees.

**LEAD AVR** is located at +30 degrees and -150 degrees.

**LEAD AVL** is located at -30 degrees and +150 degrees.

**LEAD AVF** is located at +90 degrees and -90 degrees.

### **The Chest Leads (or Precordial Leads):**

We will now discuss the remaining six leads of the 12-lead EKG. These next six leads are called the **Chest leads or the Precordial leads**. This part may also become confusing because these precordial leads are six additional leads that use the same V technique we used with the 3 limb leads. Therefore, do not confuse these Precordial **V leads** with the three V limb leads (AVR, AVL, AVF).

The precordial (chest leads) leads each consist of a positive electrode strategically placed on the chest of the patient. The positions of the positive electrode for the six precordial leads are very important for a valid tracing to be made on the EKG machine.

These positions are: (INSERT NUMBER with V) (Figure 28)

Lead	Positioned
V <sub>1</sub>	Fourth intercostals space, right sterna border.
V <sub>2</sub>	Fourth intercostals space, left sterna border.
V <sub>3</sub>	One-half way between V and V in straight line with them.
V <sub>4</sub>	Fifth intercostals space, left midclavicular line.
V <sub>5</sub>	Fifth intercostals space, left anterior auxiliary line.
V <sub>6</sub>	Fifth intercostals space, left midauxiliary line.

These positions are critical for interpretation of the EKG. At fort it may seem complicated to position these electrodes. After some practice it is relatively easy to place the leads properly. The below illustration will show the correct positions across the chest. One of the main reasons that the precordial leads are important is that these leads show the R Wave Progression. From V (NUMBER) through V

(NUMBER) the R Wave becomes progressively larger. The experienced interpreter of these leads will be able to rule out many different cardiac disorders by attaching the R wave and other configurations in the pericardial leads. (Figure 29)

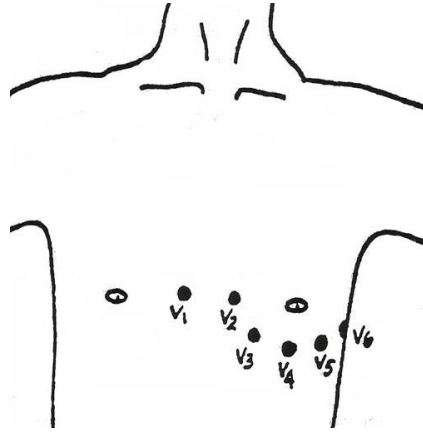


Fig28 – V - Lead (Precordial Leads) Placement on the chest

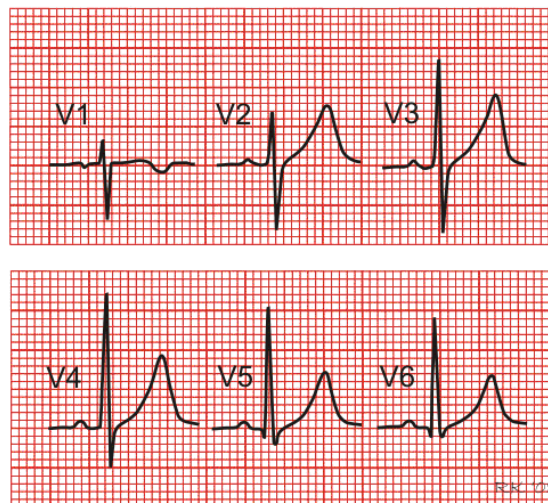


Fig29 – The normal ECG morphology of the complex in the V leads

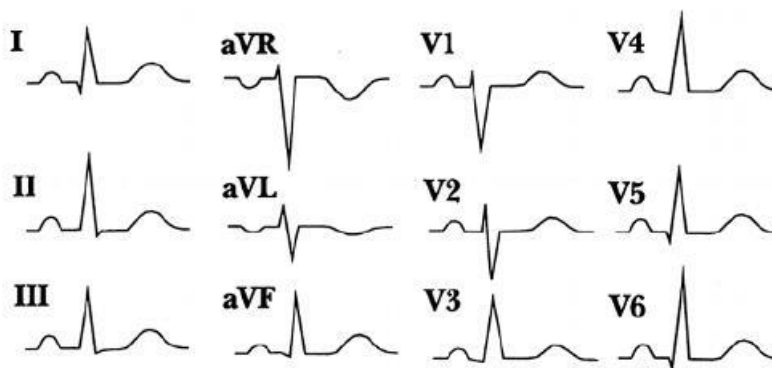


Fig30 – 12-Lead ECG



### ■ Normal and Abnormal ECG leads:

(a) Normal ECG sample and (b) abnormal ECG sample in preliminary dataset.

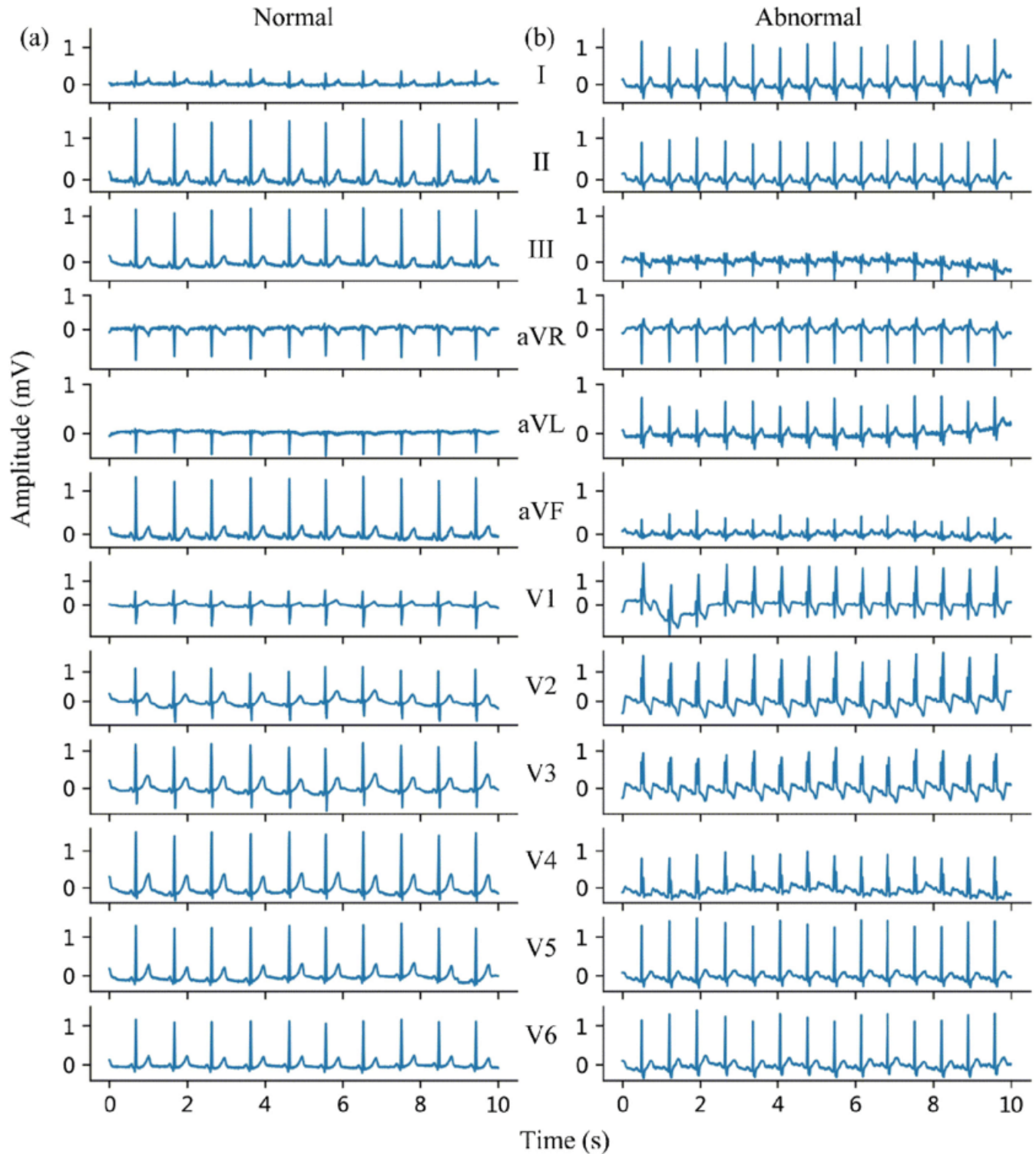


Fig31 – Normal and Abnormal ECG leads

## ■ **Abnormal ECGs:**

- **common arrhythmias**

- **common cardiac arrhythmias:** atrial fibrillation, atrial flutter (AF), supraventricular tachycardia (SVT) and ventricular tachycardia (VT)
- acute coronary syndromes (ACS), including angina and myocardial infarction
- pericarditis

- **Tachycardia:** ‘Tachycardia’ is an all-encompassing term that refers to a fast heartrate, usually in excess of 100 bpm. Tachycardia may be classified as ‘regular’ or ‘irregular’, and may have a ‘narrow’ or ‘wide’ complex.

- **Regular narrow complex tachycardia:** A regular narrow complex tachycardia is usually one of three rhythms: [\(Figure 32\)](#)

1. sinus tachycardia (ST)
2. supraventricular tachycardia (SVT)
3. atrial flutter

**Sinus tachycardia:** Sinus tachycardia is common, but usually reflects systemic illness or injury (such as sepsis, trauma or dehydration), rather than a primary cardiac condition. The electrical conduction through the heart is normal, albeit accelerated. The ECG appearances of ST include:

- regular rate, usually less than 120 bpm in an otherwise healthy person
- normal appearance of P, QRS and T waves
- QRS may be widened if an associated conduction defect is present—bundle branch block (BBB).



**Diagnosis:** Sinus tachycardia is usually apparent on an ECG, but if the heart rate is above 140 bpm the P wave may be difficult to distinguish from the previous T wave and one may confuse it with a paroxysmal supraventricular tachycardia or atrial flutter with a 2:1 block.

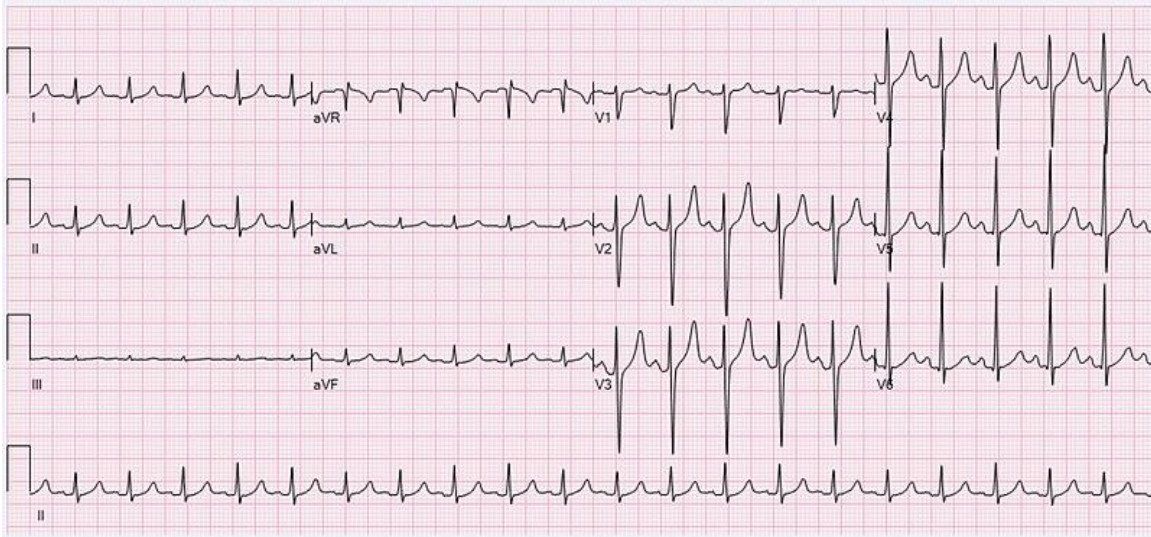


Figure 32 - ECG Sinus Tachycardia

**Supraventricular tachycardia (SVT):** SVT includes several tachycardias arising from the AV node or above. The two most common forms are AV node re-entry tachycardia and tachycardia associated with accessory conduction pathways, such as Wolf-Parkinson-White syndrome (WPW)<sup>3</sup>. In both forms, an alternative electrical pathway is present which allows repeated rapid conduction of atrial impulses to the ventricles and subsequent ventricular stimulation. (Figure 33)

The ECG appearances of SVT are characterized by:

- hidden or absent P waves
- regular QRS complexes (ventricular rate)—typically 120–180 bpm
- normal QRS complexes in the absence of associated BBB (QRS may be widened in WPW with conduction along the accessory pathway)

- normal T waves

**Diagnosis:** Subtypes of SVT can often be distinguished by their electrocardiogram (ECG) characteristics. Most have a narrow QRS complex, although, occasionally, electrical conduction abnormalities may produce a wide QRS complex that may mimic ventricular tachycardia (VT). In the clinical setting, the distinction between narrow and wide complex tachycardia (supraventricular vs. ventricular) is fundamental since they are treated differently.



Figure 33 - **ECG Supraventricular Tachycardia** : Lead II electrocardiogram strip showing PSVT with a heart rate of about 180.

**Atrial flutter:** Atrial flutter is a common regular tachycardia more prevalent in older patients. Atrial flutter is often misdiagnosed as SVT, but a rate of 150 bpm with narrow QRS complexes should alert one to the diagnosis. It may be present in association with atrial fibrillation. (Figure 34)

The ECG appearances of atrial flutter are characterized by:

- P waves (flutter waves) at a rate of 300 bpm (atrial rate). These are often described classically as ‘sawtooth’ in appearance.

- Regular QRS complexes (ventricular rate). However, the ventricular rate depends on conduction through the atrio-ventricular (AV) node. If every second P wave (atrial beat) is conducted, the QRS (ventricular) rate will be 150 (2:1 AV block). If every third P wave is conducted, the QRS rate will be 100 (3:1 AV block).
- Normal QRS complexes in the absence of associated BBB.
- Normal T waves

**Diagnosis:** Typical atrial flutter is recognized on an electrocardiogram by presence of characteristic "flutter waves" at a regular rate of 200 to 300 beats per minute. Flutter waves may not be evident on an ECG in atypical forms of atrial flutter. Individual flutter waves may be symmetrical, resembling p-waves, or maybe asymmetrical with a "sawtooth" shape, rising gradually and falling abruptly or vice versa.

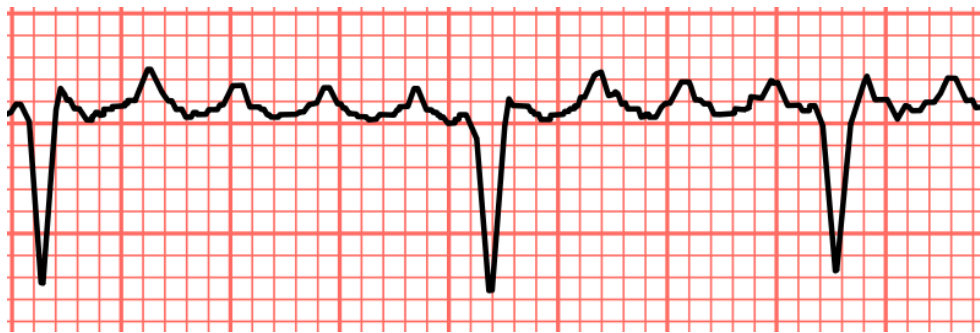


Figure 34 – Atrial flutter : Lead V1 electrocardiogram

- **Irregular narrow complex tachycardia:**

**Atrial fibrillation:** An irregular tachycardia in a conscious patient is almost always atrial fibrillation (AF). (Figure 35)

AF is the result of chaotic electrical current flow in the atria resulting in independent muscle fibre contraction (fibrillation), rather than a synchronous

contraction of all muscle fibres. The sino-atrial (SA) node no longer acts as a pacemaker for the heart.

The ECG appearances of AF are characterized by:

- variable ventricular rate (QRS complexes)
- absence of P waves
- erratic baseline between QRS complexes
- narrow QRS complexes in most cases, but these may be wide if an associated BBB is present (aberrant conduction)
- normal T waves.

**Diagnosis:** The evaluation of atrial fibrillation involves a determination of the cause of the arrhythmia, and classification of the arrhythmia. Diagnostic investigation of AF typically includes a complete history and physical examination, ECG, transthoracic echocardiogram, complete blood count, and serum thyroid stimulating hormone level.

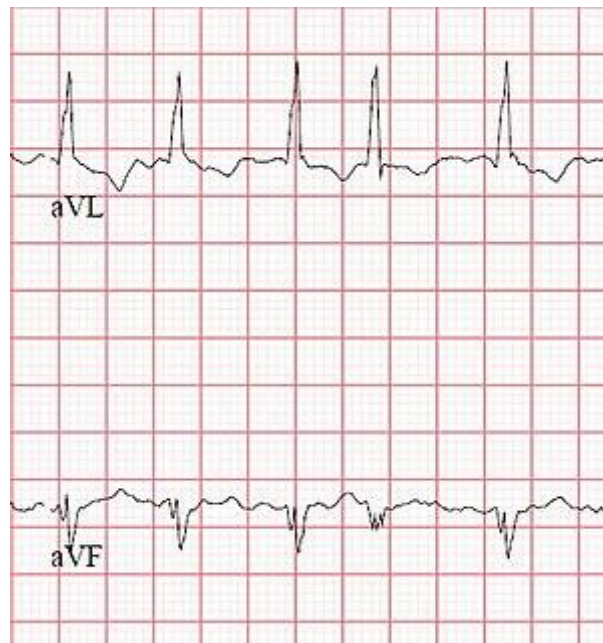


Figure 35 – **Atrial fibrillation:** Leads aVL and aVF of an electrocardiogram showing atrial fibrillation. There are irregular intervals between heart beats. No P waves are seen and there is an erratic baseline between QRS complexes. The heart rate is about 125 beats per minute.



- **Regular wide complex tachycardia:**

Wide complex tachycardia should always be regarded as being ventricular tachycardia (VT) until proven otherwise.

The differential diagnosis includes any of the narrow complex regular tachycardias (ST, SVT, atrial flutter) with an associated interventricular conduction defect (BBB). If these rhythms are confirmed, they can be managed as above. However, treatment of VT with calcium channel or beta blocking drugs may cause death of the patient.

**Ventricular tachycardia:** VT consists of at least three consecutive ventricular complexes with a rate of more than 100 bpm. (Figure 36)

Patients may or may not be conscious as a result of their VT. The ECG appearances of VT are characterized by:

- no P waves
- regular QRS complexes 120–200 bpm
- wide QRS complexes > 120 msec
- no identifiable T waves.



Figure 36 – **Ventricular tachycardia:** Lead II electrocardiogram

**Ventricular fibrillation:** VF is an asynchronous chaotic ventricular rhythm that follows no regular pattern and produces no cardiac output. The patient will be unconscious. (Figure 37)

- The ECG appearances of VF are characterized by:
- irregular undulations
- no clear-cut ventricular complexes
- varying ECG amplitude



Figure 37 – Ventricular fibrillation

- **Atrio-ventricular block:**

AV block is the term used to describe delayed conduction or non-conduction of an atrial impulse through the AV node. It may be intermittent or persistent, and is traditionally divided into three categories: first- second- and third-degree block.

**First-degree heart block:** In first-degree heart block, conduction through the AV node is delayed, but all impulses are conducted. The ECG in first-degree heart block is characterized by: (Figure 38)

- normal P waves
- prolonged PR interval  $> 20$  msec (5 mm)
- normal QRS complexes and T waves.

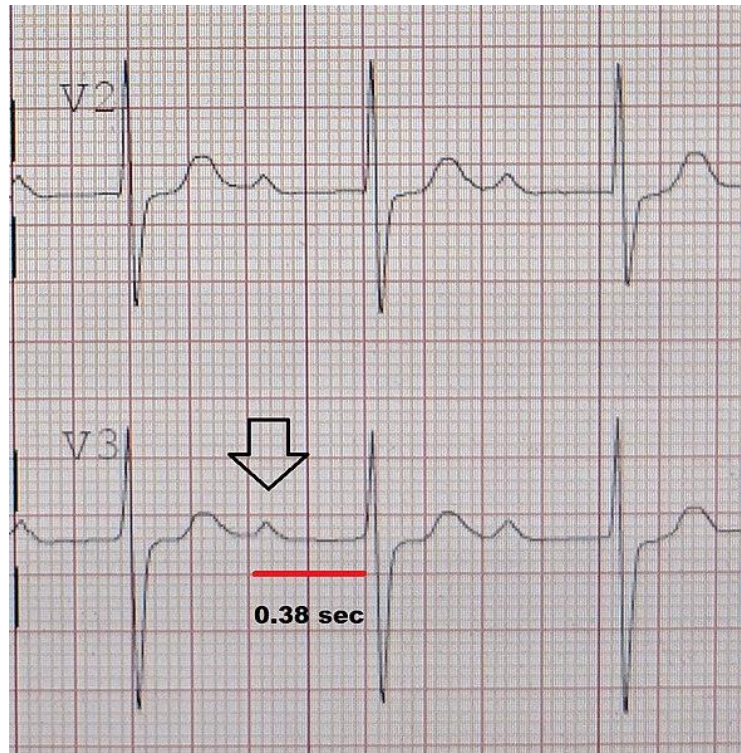


Figure 38 – First-degree heart block

**Second-degree heart block:** In second-degree heart block, some—but not all—impulses are conducted through the AV node. Second-degree heart block may be either Mobitz Type 1 (Wenckebach) or Mobitz Type 2. (Figure 39)

**1. Mobitz Type 1 (Wenckebach):** The ECG in Type 1 second-degree heart block is characterized by:

- normal P waves
- progressive lengthening of the PR interval until one P wave is no longer conducted and the sequence begins again
- normal QRS complexes and T waves

**2. Mobitz Type 2:** The ECG in Type 2 second-degree heart block is characterized by:

- normal P waves
- fixed, identical PR intervals before a long pause with no conduction

▪ *variable QRS response:*

- if the block is 2:1, ventricular rate (QRS) will be half the atrial rate (P)
- if the block is 3:1, the ventricular (QRS) will be one-third the atrial rate (P)

Mobitz I or Wenckebach



Mobitz II



2:1 block



Figure 39 – Second-degree heart block

**Third-degree (complete) heart block:** Third-degree heart block is characterized by complete failure of atrial impulses to be conducted through the AV node to the ventricles. The ECG in complete heart block is characterized by: (Figure 40)

- normal regular P waves present, unless AF present
- lack of association between P waves and QRS complexes (AV dissociation)
- regular ventricular rate (QRS) 30–50 bpm
- QRS complexes may be narrow or broad, depending on the level of block and origin of ventricular pacemaker focus



Figure 40 – Third-degree heart block



- **ST segment/T wave abnormalities:** ST segment and T wave abnormalities on the ECG may be caused by several processes (ACS, pericarditis, ventricular aneurysm).

The most important of these abnormalities is acute coronary syndrome (ACS) (angina and myocardial infarction). Myocardial tissue damaged by ischaemia has a lower negative membrane potential compared with normal myocardium. This results in flow of current from normal to abnormal myocardium and produces characteristic features on the ECG.

The ECG features of ACS are variable and depend on the extent and acuity of the ischaemia. Common ECG patterns of ACS include **ST segment elevation**. This is the classical feature of acute myocardial infarction (AMI) or ST elevation myocardial infarction (STEMI). ST segment elevation occurs early in transmural infarction.

**ST segment depression:** this is the classical feature of acute angina and usually resolves with the patient's symptoms. It may also be present acutely in non-ST elevation myocardial infarction (non-STEMI).

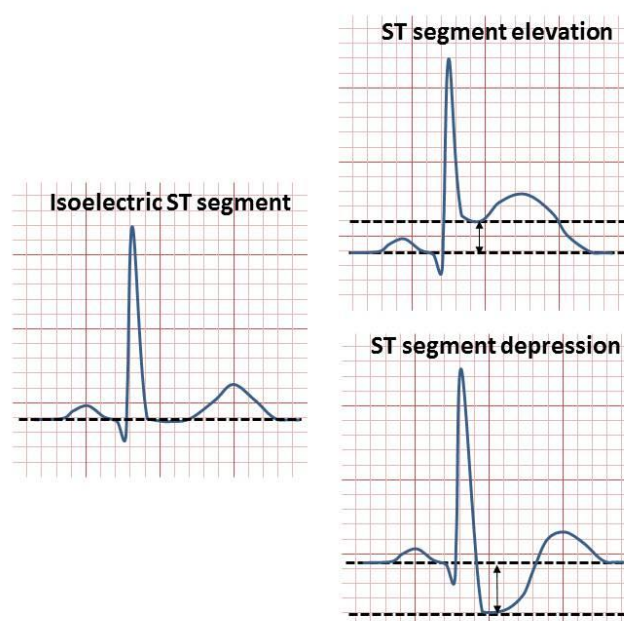


Figure 41 – ST segment abnormalities

**T wave inversion:** this feature may be seen after the initial phases of an STEMI, as the only feature in a non-STEMI, or as an indicator of ischaemia without infarction (angina).

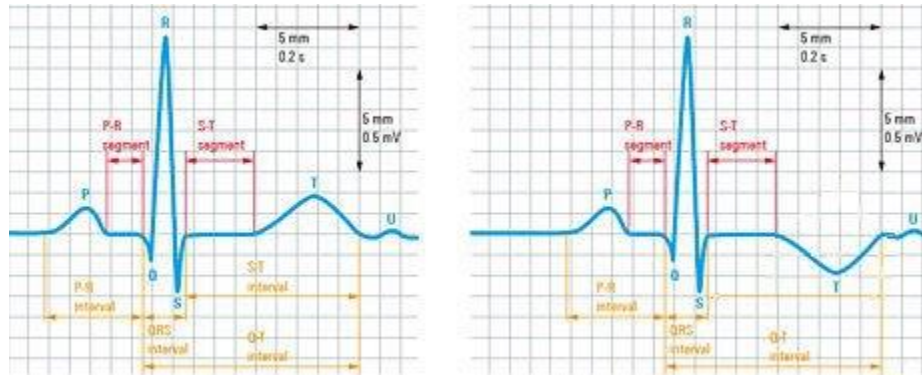


Figure 42 – T wave inversion

The ECG appearances of pericarditis can be very similar to those of an STEMI. In both conditions there is ST elevation. Along with clinical assessment, the ST elevation of pericarditis usually differs from STEMI in the following ways:

- the ST elevation is very widespread, sometimes involving all ECG leads
  - the ST elevation is classically concave, rather than the convex appearance in STEMI
  - there may be associated depression of the PR interval below the isoelectric line
- **QRS abnormalities:** The normal QRS has four characteristics:
    1. its duration is < 120 msec (3 mm)
    2. in V1, the S wave is greater than the R wave
    3. in V5 or 6, the R wave is < 2.5 mV (25 mm)
    4. left ventricle leads (V5 or 6) may show Q waves, but these are < 1 mm across and < 2 mm deep.

The QRS is abnormally wide when electrical conduction and depolarization of the ventricles do not follow the normal conduction pathway. Circumstances when this occurs include:

1. left bundle branch block (LBBB) or right bundle branch block (RBBB)

2. artificial pacemaker-initiated ventricular contraction
3. abnormal ventricular focus generating ventricular escape beats, extra systoles or tachycardia.

**Bundle branch block (BBB):** When LBBB is present, the ventricular complex in the ECG classically shows an RSR pattern in the lateral leads (V5 and 6). In RBBB, there is an RSR pattern in the right-sided leads (V1 and 2).

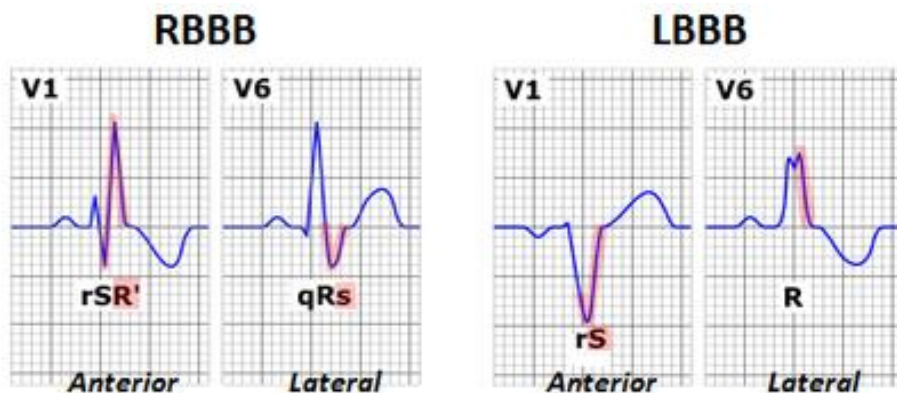


Figure 43 – QRS abnormalities

▪ **Machine Learning and ECG:**

- *Literature review*
- *Finding the features describing the ECG signals as best as possible*

▪ **PCB Design:**

- *Learning the basics of PCB*
- *Designing PCB for simpler circuits*
- *Creating the final PCB for our product*