

# **“A STUDY OF ECG CHANGES IN HEALTHY YOUNG SMOKERS COMPARED TO NON SMOKERS”**

**By**

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**“A STUDY OF ECG CHANGES IN HEALTHY YOUNG  
SMOKERS COMPARED TO NON SMOKERS”**

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## **LIST OF ABBREVIATIONS USED**

ADH	-	Ant diuretic Hormone
AECG	-	Ambulatory Electrocardiogram
BMI	-	Body mass index
B.P.	-	Blood pressure
C.O.	-	Cardiac Output
CVP	-	Central Venous Pressure
ECG	-	Electrocardiogram
F/H	-	Family history
H/o	-	History of
H.R.	-	Heart rate
I	-	Inverted
ICMR	-	Indian Council of Medical Research
I.E.	-	Isoelectric
MAP	-	Mean arterial pressure
MI	-	Myocardial Infarction
N	-	Normal
NIBP	-	Non-invasive blood pressure
RMP	-	Resting membrane potential
RVLM	-	Rostral ventrolateral medulla
S.D.	-	Standard deviation

## **ABSTRACT**

Nicotine, which is the main component of tobacco, causes sudden coronary death. It also has the propensity to provoke ventricular arrhythmias. 12-lead electrocardiogram (ECG) is a routine, inexpensive tool for assessment of cardiovascular disease and its risk in both clinical and research settings, and ECG changes powerfully predict future cardiovascular events.

The objective of my study is to compare the ECG changes between healthy smokers and non-smokers. This is a prospective case-control study. Cases are taken from apparently healthy smokers between ages 20-40 years, selected from among students and staff of the institute, patients attending inpatient and outpatient departments, and attendants of patients visiting outpatient and inpatient departments at our hospital. Non-smoking controls of the same age group were also selected from the same pool. The sample size is  $125+125 = 250$ . The period of study was from November 2019 to July 2021. Healthy asymptomatic smokers and non-smokers aged between 20-40 yrs of age were included.

ECG results were evaluated for different parameters like Rate, P wave, P.R. interval, QRS interval, S.T. segment, Q.T. interval, Qtc interval, T wave. The results show a statistically significant increase in heart rate, P wave amplitude, P.R. interval, and decrease in Q.T. interval, T wave amplitude, and occurrence of Q waves in smokers compared to non-smokers.

Our study concluded that smoking-induced alterations manifest as significant variation in waveforms in ECG recordings of even asymptomatic smokers compared to non-smokers. ECG can be used as a simple and inexpensive tool to assess smoking-induced damage and counsel and motivate smokers to quit cigarettes.

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## INTRODUCTION

India is the second-largest consumer of tobacco and third-largest tobacco-producing nation and worldwide. In India, The prevalence of Smoking among men is 19 percent, and among women, it was 2 percent. The prevalence of Smoking was 8.3 percent in urban areas and 11.9 percent in rural areas. Every tenth adult (10.7%; 99.5 million) in India currently smokes tobacco.

A daily cigarette smoker spends on an average Rs. 1192.45 per month and daily bidi smokers spend on an average of Rs. 284.12 per month on bidis.<sup>1</sup> Smoking is now justified as a significant cause of respiratory diseases, heart-related ailments, cancer, and a wide variety of other health-related problems.

Future rise in the number of men smoking due to population growth – up from 908 million in 2017 to 913 million in 2030. Mortality due to tobacco in India is estimated at about 1.3 a million yearly.<sup>2,3</sup>

The habit of Smoking is extensively practiced among the world population from time immemorial. There are 1.4 billion tobacco users aged 15 years and above worldwide – 1.07 billion smokers and 367 million smokeless tobacco users. There are 1.12 billion men currently using tobacco and 279 million women<sup>4</sup>.

According to U.S. Surgeon General, “It is safe to say that the Smoking represents the most extensively documented cause of disease ever investigated in the History of biomedical research<sup>5</sup>.

Hence the present study is being undertaken to study the ECG changes in smokers and non-smokers.

# **AIMS AND OBJECTIVES**

## **AIMS AND OBJECTIVES**

To compare the ECG changes between healthy young smokers and non-smokers.

# **REVIEW OF LITERATURE**

## REVIEW OF LITERATURE

### History of Electrocardiogram:

- About 340 B.C. - Electrical phenomena associated with living tissues were observed even by ancient man<sup>6</sup>.
- The History of electrocardiography dates from the end of the eighteenth century<sup>7</sup>.
- In 1787 – Aloysio Luigi Galvani demonstrated that the muscles of the hind limbs of a frog also manifested electromotive phenomena<sup>7</sup>. He accidentally observed that the muscles of a frog exhibited vigorous contractions whenever sparks were drawn from an electrical machine and nerves of the preparation were touched with a knife at the same time<sup>7</sup>. He suspected that this phenomenon was related to the electrical discharge and successfully repeated his experiment by connecting a dissected frog to a lightning conductor during a thunderstorm<sup>7</sup>.
- 1856 – Rudolph Von Koelliker and Heinrich Muller demonstrated electrical currents associated with each heartbeat by applying a galvanometer to the apex and base of the exposed ventricle<sup>8</sup>.
- 1876 – Marey used the electrometer to record the electrical activity of an exposed frog's heart.
- 1878 – The next advance was made when Saunderson and Page recorded the cardiac events in laboratory animals by means of the capillary electrometer<sup>7</sup>.
- In 1887 – Augustus D Waller, using the capillary electrometer, was the first to demonstrate a measurable amount of current in the human body associated with contraction of the heart<sup>8</sup>.

- He showed that currents recorded by the heart muscle could be recorded in intact animals by using surface electrodes and then proceeded to apply this method to human beings. He discovered that the electrical activity of the human heart could be recorded by means of the capillary electrometer without opening the Chest and exposing the heart<sup>7</sup>.
- The capillary electrometer was sensitive but challenging to operate. Therefore, its use was abandoned with the advent of the string galvanometer at the beginning of the 19th century.
- It was not until 1902 that the electric current from the human heart was registered in an accurate, quantitative manner by the application of a new instrument, the string galvanometer, by Willem Einthoven.
- 1903 – String galvanometer was invented by Schwegger but perfected by Willem Einthoven and thus heralded the era of electrocardiography.
- 1924 – Willem Einthoven was awarded the Nobel Prize for his contributions, a landmark in cardiology<sup>9</sup>.
- During the first quarter of the 20th century – considerable progress in electrocardiography was provided by the research of Sir Thomas Lewis<sup>10</sup>.
- Duchosal and Luthi designed the direct writing electrocardiograph in 1932.
- In 1933, Frank N. Wilson and his associates devised the unipolar lead system. The unipolar recording of the heart's electrical activity was first used for experimental purposes but eventually was adopted for clinical medicine<sup>10</sup>.
- In 1942 the American cardiologist Emanuel Goldberger increased the voltage of unipolar limb leads by 50%, leading to the term augmented leads<sup>8</sup>.

- At the end of the Second World War, Goldberger modified Wilson's unipolar technique for augmentation of tracings from the apices of Einthoven's triangle and laid the foundation for the firm establishment of a lead system<sup>10</sup>.

## PHYSIOLOGICAL BASIS OF ELECTROCARDIOGRAPHY

The electrocardiogram (ECG) is a graphic recording of the heart's electrical activity recorded from the body surface by the electrodes positioned to reflect the activity from various spatial perspectives<sup>11</sup>.

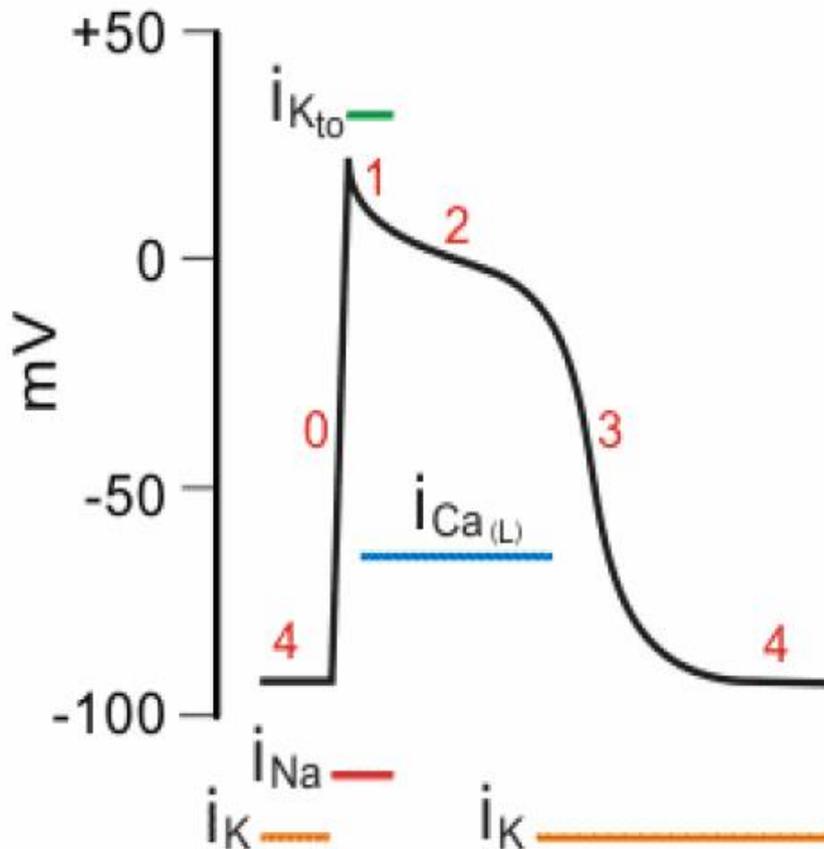
The following are the factors involved in the genesis of the electrocardiogram<sup>7</sup>:

1. Initiation of impulse formation in the primary pacemaker (sinus node).
2. Transmission of impulse through the specialized conduction system of the heart.
3. Activation (depolarization) of the atrial and ventricular myocardium.
4. Recovery (repolarization) of all the above areas.

### Intracellular potentials

Myocardial fibers have a resting membrane potential (RMP) of approximately -90mV. The major factor determining the RMP is the gradient of potassium (K<sup>+</sup>) across the cell membrane. The intracellular concentration of K<sup>+</sup> is 30 to 45 times higher than the extracellular concentration. On the other hand, an opposite gradient exists for sodium (Na<sup>+</sup>). The extracellular concentration of Na<sup>+</sup> is about 10 to 15 times higher than the intracellular concentration. At the onset of depolarization of the cardiac muscle cell, there is the abrupt change in the permeability of cell membrane to sodium ions (and calcium ions to a lesser degree) which enters the cell and result in the sharp rise in the intracellular potential to +20m V. This is designated as phase 0 and represents first inward current<sup>11</sup>. Following depolarization, there is a relatively slow and gradual return of intracellular potential to RMP (phase 4). This is repolarization and is divided into three phases:

**Fig. no. 1- Cardiac action potential**



1. Phase 1: An initial rapid return of intracellular potential to 0 m V. This results mainly due to the abrupt closing of sodium channels. Chloride ions entering the cell may also contribute to this phase.
2. Phase 2: A plateau phase of repolarization owing to the slow entrance of calcium ions into the cell.
3. Phase 3: This represents the slow, gradual return of intracellular potential to RMP. It results from the extrusion of potassium out of cells, which establishes normal negative resting potential.

However, the cell is left with an excess of sodium and a deficit of potassium.

To restore the original ion concentration in the cell membrane, the sodium-potassium pump mechanism becomes effective. The energy required for this pump is derived from the conversion of ATP to ADP. This pump removes sodium from the cell and permits potassium influx.

The summation of all 'phase 0' potentials of atrial myocardial cell results in the 'P wave' of ECG. All 'phase 0' potentials of ventricular muscle cells produce the 'QRS complex.' 'Phase 2' correlates with 'S.T. segment' and 'phase 3' with T wave of ECG, respectively<sup>11</sup>.

## **ELECTROCARDIOGRAPHIC LEADS**

Electrocardiographic leads may be physiologically divided into two groups depending upon their orientation to the heart.

1. Frontal plane leads: These are oriented in the frontal plane or coronal plane of the body and consist of standard leads I, II, III and a V.R., a V.L., a V.F.
2. The horizontal plane leads: These are oriented in the horizontal or transverse plane of the body and are formed by the precordial leads V1 to V6<sup>12</sup>.

## **BIPOLAR STANDARD LEADS**

The bipolar standard leads I, II, and III are the original leads selected by Einthoven to record the electrical potential in the frontal plane. Einthoven placed the recording electrodes on the right arm, left arm, and left leg<sup>12</sup>.

The bipolar leads represent a difference in electrical potential between two selected sites. Standard lead I: This lead is derived from the placement of the negative electrode on the right arm and the positive electrode on the left arm.

Standard lead II: This lead is derived from the placement of the negative electrode on the right arm and the positive electrode on the left foot.

Standard lead III: This lead is obtained by placing a negative electrode on the left arm and the positive electrode on the left foot.

The electrocardiographic machine is provided with a right leg electrode which acts as a ground wire and plays no role in producing ECG.

### **AUGMENTED LEADS:**

aVR, aVL and aVF are the three augmented leads that record complexes representing electrical activity at a particular point<sup>10</sup>.

Lead aVR: Augmented unipolar right arm lead. This lead faces the heart from the right shoulder. This lead is usually oriented to the cavity of the heart. Thus all deflections – P, QRS, and T deflections are generally negative in this lead.

Lead aVL: Augmented unipolar left arm lead. This lead faces the heart from the left shoulder. It is oriented to the anterolateral or superior surface of the left ventricle.

Lead aVF: Augmented unipolar left leg lead. This lead is considered to be oriented to the inferior surface of the heart<sup>12</sup>.

### **PRECORDIAL LEADS (CHEST LEADS)**

The Chest leads record the electrical activity from the Chest. There are six Chest leads V1-V6 placed at different places on the Chest.

V1- fourth intercostal space at the right sternal border<sup>13</sup>.

V2- fourth intercostal space at the left sternal border.

V3- Midway between leads V2 and V4 electrode positions.

V4- Fifth intercostal space in the left midclavicular line.

V5- Fifth intercostal space in the left anterior axillary line

V6- Fifth intercostal space in the left midaxillary line<sup>13</sup>.

## NORMAL ELECTROCARDIOGRAPHIC COMPLEXES

**P wave:** This is due to deflection produced by atrial depolarization. It does not exceed 0.11 seconds and 2.5mm in height. **QRS complex:** This is due to ventricular depolarization and measures normally 0.04 to 0.10 seconds.

**P.R. interval:** It is the interval between the beginning of the p wave and the beginning of the QRS complex. It represents the interval between the onset of atrial depolarization and the onset of ventricular depolarization. The normal duration is 0.12 to 0.20 seconds in the adult.

**ST-segment:** Represents a more significant part of ventricular repolarization. It usually is isoelectric.

**T wave:** Represents ventricular repolarization, at least 10% R wave in the same Lead, no upper limit

**QTC interval:** QTC interval measures the duration of electrical activation and recovery of the ventricular myocardium and inversely varies with the heart rate. It is estimated from the beginning of the QRS complex to the end of the T wave. It is rate-dependent and must be corrected for heart rate. QTC interval is determined using modified Bazett's formula by Hodges and co-workers. It corrects more completely for high and low rates<sup>14</sup>.

$QTC = Q.T./\text{square root of R.R. interval}$  The upper limit of the duration of QTC interval is approximately 0.45seconds (450 milliseconds).

**Amplitude of R wave:** It is the upright deflection and is measured from the upper portion of baseline to the peak of the wave. It is best seen in V5-6.

**Amplitude of S wave:** It is the negative deflection and is measured from the lower portion of the baseline to the nadir of the wave. It is best seen in V1-2.

**R.R. interval:** It is the distance between two successive R waves. This interval is used for measuring the heart rate<sup>13</sup>.

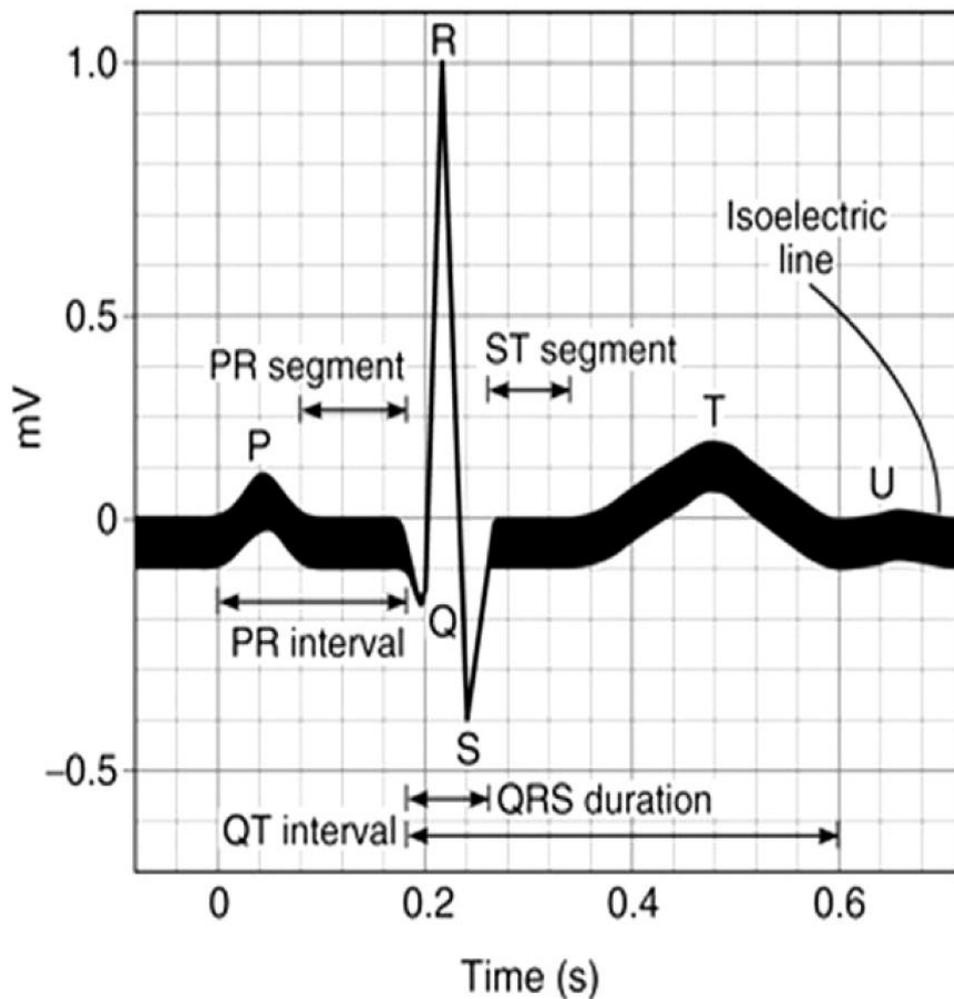


Figure no.2 Electrocardiogram.

## SMOKING

### **Epidemiology and History of Smoking:**

Every year, tobacco causes 3.5 million deaths throughout the world, and this will increase to 10 million annual deaths during the 2020s, with seven million of these deaths occurring in developing countries. Dr. Hiroshi Nakajima, Director-General of WHO, termed the growing tobacco epidemic as 'A fire in the global village.'

The History of tobacco dates to the Portuguese merchants 400 yrs ago who brought tobacco to India. Although new imported varieties from Brazil outclassed some strains of locally grown tobacco in India. The trade boomed, and tobacco quickly established itself as the essential commodity passing through Goa in the 17th century. Later on, the British introduced modern commercially produced cigarettes.

In the era of nineteenth and twentieth centuries, the tobacco industry developed. Later, by developing the movie industry, the first advertisement that presented Smoking as a 'fountain of pleasure' appeared. After certain progress in chemistry, it was possible to find out what kind of substances there are in tobacco leaves. So in 1950, people found out how harmful tobacco was, and then the American medical department published their first report about Smoking. In 1970 medical council decided that there must be a warning about how harmful cigarettes are for our health on every cigarette box.

India is the second-largest consumer of tobacco and third-largest tobacco-producing nation and worldwide. In India, The prevalence of Smoking among men is 19 percent, and among women, it was 2 percent. The prevalence of Smoking was 8.3 percent in urban areas and 11.9 percent in rural areas. Every tenth adult (10.7%; 99.5 million) in India currently smokes tobacco.

A daily cigarette smoker spends on an average Rs. 1192.45 per month and daily bidi smokers spend on an average of Rs. 284.12 per month on bidis<sup>1</sup>. Smoking is now justified as a significant cause of respiratory diseases, heart-related ailments, cancer, and a wide variety of other health-related problems.

### **CIGARETTE AND ITS MAKING<sup>15</sup>:**

Cigarettes look simple, consisting of paper tubes containing chopped-up tobacco leaves, usually a filter at the mouth end. The tobacco is blended from the two main leaf varieties: yellowish 'bright,' also known as Virginia, where it was grown, contains 2.5-3 % nicotine, and 'burley' tobacco with 3.5- 4 % higher nicotine content. In addition to leaf blends, cigarettes contain 'fillers' made from the stems and other bits of tobacco which would otherwise be waste products. High filler content makes a less dense cigarette with lower tar delivery. 'Additives' are used to make tobacco products more acceptable to the consumer. They include 'humectants' (moisturizers) to increase shelf life; sugars to make the smoke seem milder and easier to inhale; and flavorings such as vanilla and chocolate. The nicotine and tar delivery can be modified by the type of paper used in the cigarette; the more porous paper will allow more air into the cigarette, diluting the smoke and reducing the amount of nicotine and tar, reaching the smoker's lungs. Filters are basically made of cellulose acetate and trap some of the tar and smoke particles from the inhaled smoke. Lately, the tar content and nicotine content have been gradually reduced compared to the 1960s. Still, the low tar cigarettes are no safer than higher tar cigarettes because of the likelihood of smokers to compensate for the reduction in tar and nicotine by smoking more or inhaling more deeply.

## **THE RISKS OF SMOKING<sup>16</sup>:**

Smoke is the most vicious component of the cigarette. Smoke contains carbon monoxide and nitrogen oxide, which are harmful gases. When inhaled, it targets the lungs along with the smoke. Tar itself includes 4000 chemicals, some of which are known to cause cancer. Other inhaled chemicals include:

- Cyanide
- Benzene
- Formaldehyde
- Methanol
- Acetylene
- Ammonia, etc.

## **ON CARDIOVASCULAR SYSTEM:**

- Other than advanced age, Smoking is the single most important risk factor for coronary artery disease. Ischemic heart disease causes 35-40% of all smoking-related deaths, with an additional 8% attributable to secondhand smoke exposure. Even among non-smokers, inhaled smoke, whether from passive exposure or cigar or pipe consumption, increases coronary risk.
- Landmark studies in the early 1950's first reported a strong positive association between cigarette smoke exposure and coronary heart disease. Over the next 50 years, an exceptionally consistent series of prospective studies have documented the effects of Smoking on coronary risk. These studies suggest that, compared with non-smokers, people who consume or more cigarettes daily have a two-three-fold increase in total coronary heart disease.

- Moreover, the effects depend on dose; consuming as few as one to four cigarettes daily increases CAD risk. Such 'light levels of Smoking have a significantly important role in myocardial infarction. In addition to MI, cigarette consumption directly relates to increased rates of sudden death, aortic aneurysm formation, symptomatic PVD, and ischemic stroke.
- Prospective evidence has linked cigarette consumption to an elevated risk of hemorrhagic stroke, including intracranial hemorrhage and subarachnoid hemorrhage again in a dose-response manner. Continued Smoking is also a significant risk factor for recurrent MI<sup>17</sup>.
- Cigarette use activates platelets, increases circulating fibrinogen, increases heart rate and elevates blood pressure. It appears to promote platelet disruption. Beyond acute unfavorable effects on B.P., sympathetic tone, and reduction in myocardial oxygen supply, Smoking affects atherothrombosis by several other mechanisms<sup>17</sup>.
- Cigarette smoke has been reported to induce platelet adhesion to the vessel wall. Whole smoke, nicotine, and carbon monoxide have been shown to damaged endothelium, resulting in potentially atherogenic injury<sup>18</sup>. In addition to accelerating atherosclerotic progression, long-term Smoking may enhance oxidation of LDL cholesterol and impair endothelium-dependent artery vasodilatation. This late effect is linked to dysfunctional endothelial Nitrous oxide biosynthesis following chronic and acute cigarette consumption.
- In addition, Smoking has adverse hemostatic and inflammatory effects, including increased C-reactive protein, soluble intracellular adhesive molecule-1, fibrinogen, and homocysteine.

- Additionally, Smoking is associated with platelet aggregation, increased monocyte adhesion to endothelial cells, and adverse alterations in endothelial-derived fibrinolytic and antithrombotic factors, including tissue-type plasminogen activator and tissue pathway factor inhibitor. Smokers have an increased prevalence of coronary spasms and reduced thresholds for ventricular arrhythmia.
- Accruing evidence has suggested that insulin resistance represents an additional mechanistic link between smoking and premature atherosclerosis<sup>17</sup>.
- Nicotine, a neurotransmitter at sympathetic ganglia, increases heart rate, blood pressure, and systemic vascular resistance. Thus it enhances the demand of myocardium for oxygen while carbon monoxide decreases the supply. Cigarette smoking, hyperlipidemia, and hypertension are three major risk factors for coronary artery disease.
- Smoking acts both synergistically and independently with these other risk factors. Smoking cessation reduces coronary artery disease mortality by 36% compared to mortality in subjects who continued smoking<sup>18</sup>.

## **HYPERTENSION AND CIGARETTE SMOKING:**

- The nicotine in cigarette smoke transiently increases B.P. by 10-20 mm Hg with every cigarette, thereby elevating the average daytime B.P. in habitual smokers.
- Acutely the surge of nicotine causes an increase in both systolic and diastolic B.P. that may last for 15-30min<sup>19</sup> likely mediated by the release of norepinephrine from adrenergic nerves. Even more prolonged elevations in B.P. follow the use of smokeless tobacco<sup>20</sup>.
- When their pressures are taken more than 30 min after their last cigarette, smokers have generally been found to be no more hypertensive than non-smokers, and many are often less hypertensive because smokers tend to weigh less than non-smokers.
- However, the repeated pressor effects of each cigarette can result in elevated pressure for most of the day in the typical pack-per-day smoker, clearly contributing to increased risk of cardiovascular disease noted among smokers. This pressor effect must be partly responsible for the significant increase in strokes<sup>21</sup> and coronary disease among smokers<sup>22</sup>.

## **ON LUNGS:**

- Cigarette smoke contains so many chemicals, and cancer may develop from-
- Cigarette tar found in the smoker's lung can cause specific DNA damage that is particularly difficult for the cell to repair.

- Cigarette smoke is a source of chemicals called polycyclic aromatic hydrocarbons, leading to specific genetic mutations in the p53 tumor suppressor gene. In a normal state, this gene is protective against cancer.
- Smoke is also a cause for many respiratory diseases like chronic obstructive pulmonary disorders (COPD), emphysema, etc. Including lung cancer, Smoking is related to other cancers like higher rates of leukemia, cancers of the kidney, stomach, pancreas, and bladder.

#### **ON FEMALE FERTILITY AND PREGNANCY:**

- Studies have linked cigarette smoke to many reproductive problems.
- Greater risk for infertility in women.
- Greater risk for ectopic pregnancy and miscarriage.
- Greater risk for prematurity, stillbirth, and low birth weight.
- Smoking reduces folate levels, a vitamin essential for preventing congenital disabilities.
- Women who smoke may pass the genetic mutations that increase cancer risk to their unborn babies.

#### **ON SEXUALITY AND REPRODUCTION:**

Even in male, reproductive and sexual health is not immune from the effects of Smoking.

- Heavy Smoking is frequently quoted as a contributory factor in impotence.
- Smoking also reduces sperm density and thus motility, thereby increasing the risk for infertility.

### **ON BONES AND JOINTS:**

- Smoking impairs the formation of new bone, and women who smoke are at high risk for osteoporosis.
- Smokers are apt to develop degenerative disorders and injuries in the spine.
- In women, Smoking may also cause a small increased risk for developing rheumatoid arthritis.

### **ON GASTROINTESTINAL SYSTEM:**

- Smoking increases the acid secretion in the stomach, reduces the production of compounds that protect the lining, and reduces blood flow in the stomach lining. It is one of the significant risk factors in diverticulitis and its complications, including bleeding and abscess.

### **THE PATHOPHYSIOLOGY OF SMOKING**

- Cigarette smoking is one of the important causes of chronic obstructive pulmonary diseases (COPD). Almost 80% of COPD's are attributable to Smoking.
- Cigarette smoke is less or non-irritant to upper airways- hence enables deep inhalation without apparent discomfort.
- Cigarette smoke is a toxic irritant to lower airways and alveoli. It has a gas phase and a particulate phase<sup>23</sup>.
- The gas-phase which consists of:
  - Irritant gases – phenol, catechol, etc.
  - Metabolic poisons – cyanide, carbon monoxide, etc.

- Oxidant gases and free radicals. About  $10^{17}$  free radical molecules in every puff.

The particulate phase which consists of:

- Respirable particulates – a mixture of fine and ultrafine particles.
- Toxic organic chemicals are adsorbed to particulate surfaces like polycyclic aromatic hydrocarbons, etc.

#### **AIRFLOW LIMITATION<sup>24</sup>:**

- In pulmonary emphysema, the alveolar structures of the lung are destroyed. As a result, the surface area for gas exchange is reduced. This is reflected in reduced diffusion capacity for carbon monoxide, which can be assessed in the pulmonary function laboratory.
- In addition, the destruction of lung tissue leads to loss of lung elasticity. After inhalation, which ordinarily stretches the lung, there is decreased elastic recoil. This results in reduced driving pressure and therefore reduced pressure in the airways.
- The small airways depend on the intraluminal pressure generated by alveolar elastic recoil and the integrity of the peri-bronchiolar alveolar walls, which provide a tethering effect that maintains patency during forced exhalation. As a result of alveolar wall destruction, the walls collapse during forced exhalation, causing effectindependent airflow limitation.
- In other words, trying harder to breathe out does not improve airflow in the subject.

## **INFLAMMATION:**

- Cigarette smokers characteristically have an increased number of inflammatory cells in the lower respiratory tract. Alveolar macrophages, the predominant inflammatory cell in the normal respiratory tract, are markedly increased in the lungs of cigarette smokers.
- Macrophages are typically filled with pigmented debris that is probably derived from smoke and cells injured by the smoke. In addition, neutrophils are present within the airway lumen and in the periglandular areas.
- Cigarette smoke and other insults likely contribute to chronic inflammation of the lungs in several ways.
- Cigarette smoke can activate the epithelial cells that line the airways to release proinflammatory cytokines. Macrophages are similarly activated. In addition, cigarette smoke can activate complement, thus generating proinflammatory mediators in a cell-independent manner.
- These multiple mechanisms probably lead to the recruitment and activation of the diverse inflammatory cells present in the lungs of smokers.
- The inflammatory cells which accumulate in the lung release proteases and oxidants capable of damaging lung structures. In addition, inflammatory cells are themselves rich sources of cytokines that can affect the lung's epithelial cells and mesenchymal cells.
- By damaging and thus altering the architectural structure of the lung, inflammatory cells are believed to be the proximate mediators of tissue disruption in smokers.

## **PROTEASES:**

- Laurell and Eriksson observed that emphysema develops in patients with congenital deficiencies in the serum inhibitor of serine proteases,  $\alpha$ -protease inhibitor.
- The concept that unopposed proteolytic destruction of lung tissue leads to the development of emphysema is proved by an animal model wherein infusion of a proteolytic enzyme into the lungs of an animal has shown similar results.
- Enzymes with elastolytic activity are essential for the pathogenesis, also neutrophil elastase; other serine proteases with elastolytic activity, such as chymotrypsin and proteinase, have a role in it.
- Of the other classes of proteases, including cysteine proteases and metalloproteases with elastolytic activity, particularly metalloprotease, is essential as this enzyme is the major secreted protease contained in macrophages.

## **COLLAGENASES<sup>25</sup>:**

- It is becoming increasingly evident that not elastin degradation alone but alterations in collagen also has a significant role in morphological changes, seen in smoke-induced damage.
- Cigarette smoke can lead to the recruitment of macrophages that are the predominant cells in the airways and alveolar spaces and contain an array of enzymes capable of digesting fibrillar collagen and elastin.
- Macrophages secrete cytokines, which further augment inflammatory response, leading to induction and release of proteolytic enzymes, leading to collagen and elastin degradation.

- The cigarette smoke and macrophages then stimulate the parenchymal lung cells to produce more proteolytic enzymes. This was resulting in a significant imbalance of collagenase, leading to a changed fibril arrangement. The initial damage is followed by a repair process, resulting in increased collagen deposition in the lung.

#### **OXIDANTS<sup>24</sup>:**

- Cigarette smoke contains large quantities of reactive oxygen species capable of causing oxidant-mediated lung damage. In addition, inflammatory cells generate and release reactive oxidants and probably greatly amplify the oxidant stimulus due to cigarette smoke. Oxidants are capable of damaging lung structures in several ways –
- Oxidants can directly damage the extracellular structures of the lung.
- Oxidation of proteins and lipids within the cells can compromise cellular function. Lipid peroxidation products moreover can be potent proinflammatory mediators.
- Oxidants also can deactivate antiproteases through such an effect; oxidant stress could result in an acquired functional equivalent of  $\alpha$ -1-protease inhibitor deficiency.
- Cigarette smoke likely leads to airway injury both by direct effects of smoke and the damage caused by mediators derived from inflammatory cells. In response, airway repair processes are inhibited. Although the result may restore normal airway structure, metaplasia of the airway epithelium may occur.
- The mesenchymal cells that underlie the airway epithelium are also activated following airway injury. Accumulation of excess fibroblasts and

myofibroblasts together with the connective tissue produced by these cells results in airway fibrosis. Contraction of these structures, a characteristic feature of all fibrotic scars, can lead to airway narrowing.

## REVIEW OF OTHER STUDIES

1. **Mallikarjun v, Prashanth Babu et.al.**<sup>26</sup>.in august 2017 did the study which included 100 healthy males, 50 non-smokers, and 50 smokers and, between ages 20-35, and the results were statistically significant increase in heart rate,decrease in P.R. interval,decrease in Q.T. interval,decrease in QT<sub>c</sub>interval,decrease in axis,decrease in T wave amplitude, the occurrence of Q wave in smokers compared to non-smokers.
2. **Swathi K , Garimella S et.al.** <sup>27</sup>.did a study in 2015 on ECG effects in 200 smoking people aged above 20 years selected and the same age as non-alcoholic. The results were P wave amplitude was increased, P-R interval was increased. In addition, QRS complex was reduced. T wave was 100% normal in non-smokers and 91% normal in smokers, T wave was abnormal in 8% of smokers.
3. **Vasant Deokar, Mandade Arjun D, et al.** <sup>28</sup>., included 150 healthy young male Smokers and Non-smokers of the same age group. The study says that Smoking induces significant alteration in cardiac electrophysiology Prolongation of QT<sub>c</sub> interval in apparently healthy young individuals, predisposing to cardiovascular morbidity and mortality.
4. **Dr. Domala Prasad, Dr. AkshayBerad et al.** <sup>29</sup>, included 30 healthy male cigarette smokers of age group between 20-30 years and an equal number of healthy age and sex-matched controls. The study says that short-term Smoking of

2-5 years duration produces depolarization abnormalities represented as ST-segment changes and T-wave changes, a reduction in QTc interval, and reduction in T.P. interval.

5. **Siva subramanian Ramakrishnan, Kinjal Bhatt et al.** <sup>30</sup>, included 31 men with an average age of 49 years, with approximately one-fourth of them being bidi smokers. The study says Heart rate and ectopic beats increase the following Smoking acutely. Ischaemic S.T.-T changes were also detected during Smoking. Spectral parameters of HRV analysis of smokers remained normal limits, but more importantly, the geometrical parameter—HRV index—showed a significant abnormality.
  
6. **Nirmal Kumar Sharma ,Kapil Kumar Jaiswal ,et al.** <sup>31</sup>included 150 smokers and 50 non-smokers between ages 25-40 years, further categorized and compared according to age, sex, and pack-years of Smoking.Heart rate and QTc-interval increased with increase in the number of pack-years. This increase was reflected more in female with a similar number of pack years. P-wave amplitude tended to increase with the increase in the number of pack years more so in males. P-wave duration, PR-interval, QRS-duration and RR-interval tended to decrease with an increase in the number of pack-years, more so in females with a similar number of pack years. QT-interval and ST-segment duration tended to decrease with an increase in the number of pack-years, more so in males.

7. **Chandrasekhar Athikari and Dwarakanath Nallapoola**<sup>32</sup> included 90 subjects, 45 non-smokers and 45 smokers, who smoke 11 - 25 cigarettes per day for the past 15 years. In this case-control study, it was found that abnormalities in ECG parameters were more likely to be prevalent in smokers as compared to non-smokers. QTc-interval increased with increase in pack-years of Smoking. P-wave duration, PR-interval, QRS-duration and RR-interval tended to decrease with an increase in the number of pack.
8. **K Harikrishnan , C Sridhar ,et.al**<sup>33</sup> included 150 young healthy male smokers and 50 nonsmokers. The ECG changes observed were an increased heart rate, increased QTc interval, increased P wave amplitude and decreased S-T segment duration even in apparently young healthy smokers. A statistically significant association was also seen between the prevalence of ECG changes and pack years of 5 or more. ECG changes develop even in healthy smokers, increasing with higher pack years of smoking.
9. **Amol Ghule and Swapnil Shinkar**<sup>34</sup> included 150 healthy young male smokers , non-smokers and 150 drinkers, non-drinkers. PR interval was significantly shortened among smokers & drinkers. No statistically significant difference in QRS interval between the two groups (Smokers & non smokers), QTc interval was slightly higher among smokers than non-smokers.
10. **Ashish Lakhanpal, Malini Kulshrestha,et.al**<sup>35</sup> did prospective case control study w on 100 smokers and 100 non smokers. The heart rate was increased in smokers which was statistically significant. The RR interval, The analysis showed

that QTc interval was shortened and that the QRS complex duration was widened the QT interval and the ST segment were shortened as compared to those in the non smokers which was highly significant statistically. QTc interval was shortened and that the QRS complex duration was widened in the smokers, although the values did not show any statistical significance.

# **MATERIALS AND METHODS**

## **MATERIALS AND METHODS**

### **STUDY DESIGN:**

This is a prospective case-control study.

### **SOURCE OF DATA:**

- This study included outpatients and inpatients of BLDE (Deemed to be University) Shri B.M. Patil Medical College Hospital and Research centre, Vijayapura 586103 and their attenders.
- The patients were informed about the study in all respects, and informed consent was taken.
- Period of study was from November 2019 to August 2021.

### **METHOD OF COLLECTION OF DATA**

#### **Study population.**

- Cases were apparently healthy smokers between ages 20-40 years, selected from among students and staff of the institute, patients attending inpatient and outpatient department and attendants of patients visiting outpatient and inpatient departments at the hospital. Non-smoking controls of the same age group were selected from the same pool. The nature and purpose of the study was described to the subjects and informed consent was obtained from those willing to participate in the study.
- A pre-structured proforma was given to each subject to record personal details and pertinent medical History from both cases and controls. Details of smoking habit, that is duration and quantum of Smoking, was obtained from cases.
- Detailed physical and clinical examination was done in detail.

### **SAMPLE SIZE:**

**125+125 =250.**

With Anticipated Mean Difference of mean heart rate beat between the smokers and non smokers as 5.5 and Anticipated SD as 12.1 the minimum sample size per group is 125 With 90% power and 5% level of significance.<sup>12</sup>

Total sample size  $125*2=250$

By using the formula:

$$n = \frac{(z_{\alpha} + z_{\beta})^2 2 SD^2}{MD^2}$$

Where Z= Z statistic at a level of significance

MD= Anticipated mean difference

SD= Anticipated Standard deviation

### **STATISTICAL ANALYSIS:**

All characteristics were summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation (S.D.) were used. For categorical data, the number and percentage were used in the data summaries and data were analyzed by Chi-square test for association, comparison of means using t-test, ANOVA and diagrammatic presentation.

### **INCLUSION CRITERIA:**

- Healthy asymptomatic smokers and non smokers aged between 20-40 yrs of age.

**EXCLUSION CRITERIA:**

- Patients with diagnosed with Hypertension.
- Any history of cardiovascular illness.
- Any history of Renal disease.

**Materials required for the study:**

- ECG machine
- Height measuring scale
- Weighing scale
- Mercury Sphygmomanometer
- Stethoscope

**ELECTROCARDIOGRAPHIC RECORDING:**

Electrocardiography was done using BPL CARDIART 6108T or VESTA 301i ECG machine. The ECG was recorded and was evaluated for different parameters like heart rate, P wave, P.R. interval, QRS complex, QRS axis, Q.T. interval, QTc interval, STsegment, Q wave and T wave and results were drawn.

# RESULTS

## RESULTS

**Table 1:Age wise distribution of study subjects**

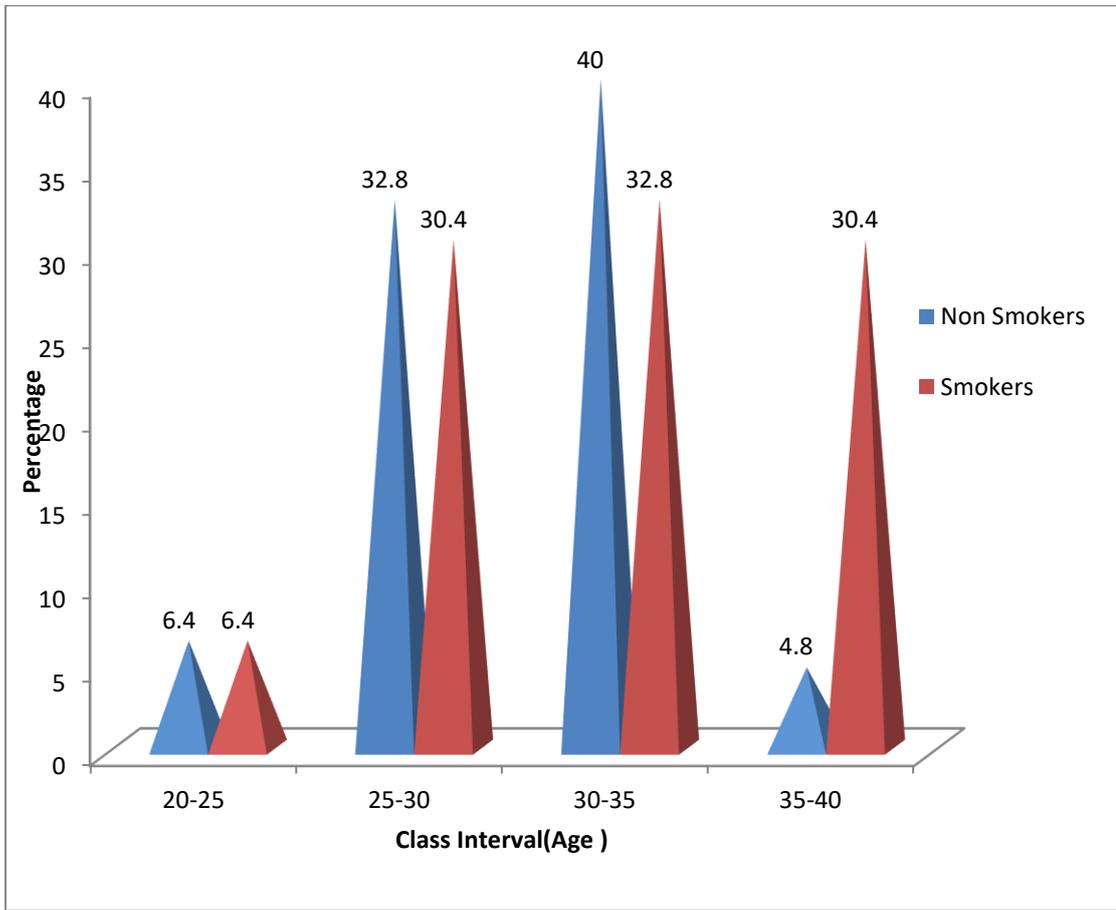
	Class Interval	Groups				Chi- Square value	P- value
		Non Smokers	%	Smokers	%		
AGE(YRS)	20-25	8	6.4	8	6.4	23.903	<b>0.20</b>
	25-30	41	32.8	38	30.4		
	30-35	50	40	41	32.8		
	35-40	6	4.8	38	30.4		
Total		125	100	125	100		

% = FREQUENCY

Total study subjects participated in the study were 250 and out of the 50% were smokers and 50% were non smokers. Among Non smokers, the highest percentage of study subjects were in the age group of 25– 30 years and whereas among smokers , the highest percentage of study subjects were in the age group of 30-35 years.

There is no much difference in age distribution between smokers and non smokers and it was statistically not significant (p-value- 0.20).

**GRAPH- 1**



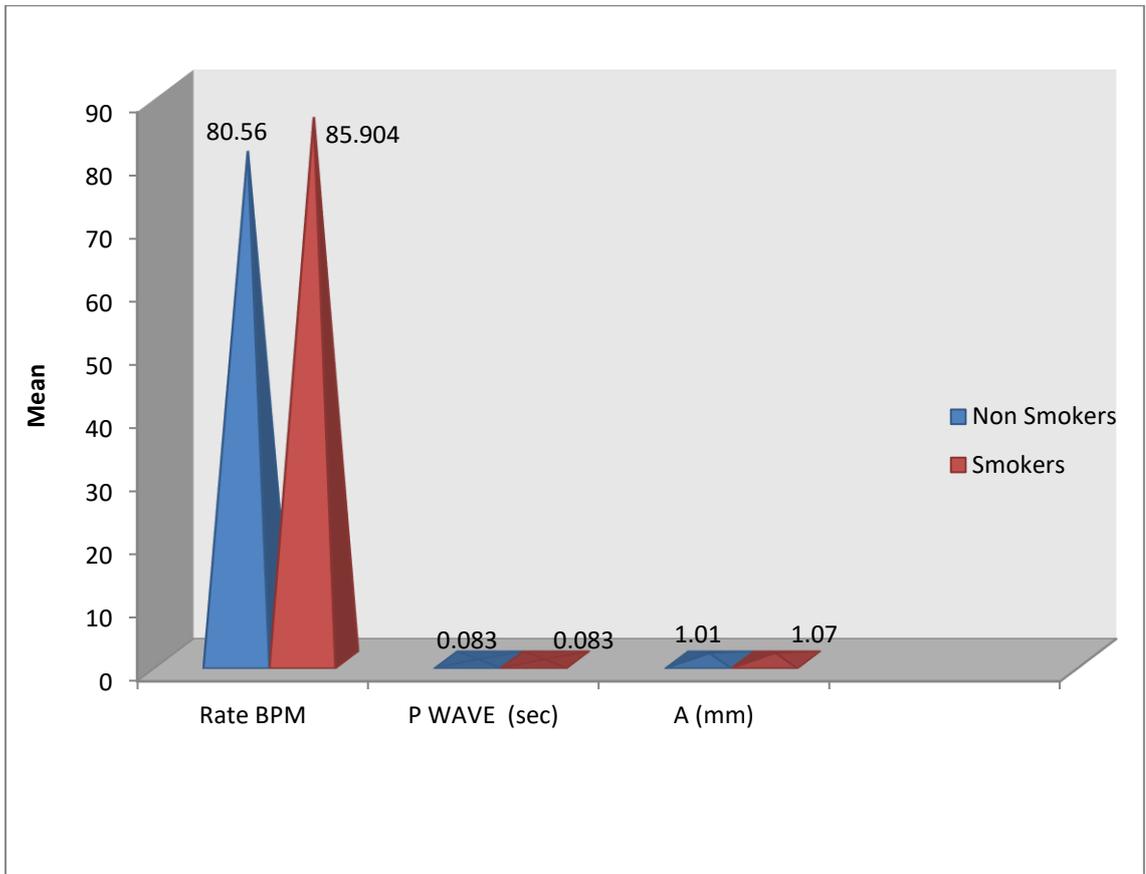
**Table 2 : Comparison of Heart rate and P wave between Nonsmokers and Smokers.**

Variables	Non smokers			Smokers			Mann-Whitney U Value	P-value
	N	Mean	SD	N	Mean	SD		
Rate BPM	125	80.560	7.146	125	85.904	9.326	5221.500	<b>&lt;0.001</b>
P WAVE(sec)	125	0.083	0.007	125	0.083	0.007	7976.00	<b>0.664</b>
A (mm)	125	1.010	0.043	125	1.070	0.184	6717.00	<b>&lt; 0.001</b>

Heart rate (mean) was high among smokers (85.904) compared to Non smokers (80.560) and this difference was found to be statistically significant (P-value – <0.001).

There was no much difference in duration (mean) of P wave between Non smokers (0.083) and Smokers (0.083) and it was not statistically significant (P-value - 0.664). And amplitude (mean) was slightly high among smokers (1.070) compared to Nonsmokers (1.010) which was statistically significant (P-value - <0.001).

**Graph -2**

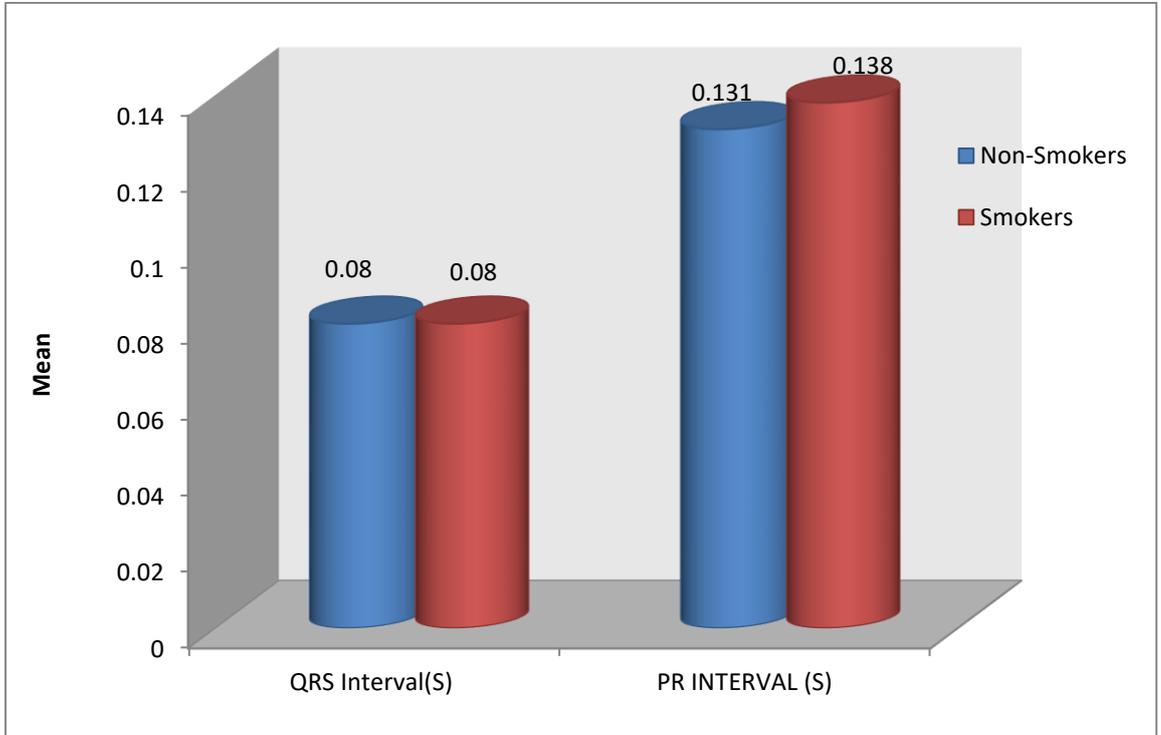


**Table 3: Comparison of P.R. and QRS interval between Non smokers and Smokers.**

Variables	Non smokers			Smokers			Mann-Whitney U Value	P-value
	N	Mean	SD	N	Mean	SD		
PR INTERVAL (S)	125	0.131	0.015	125	0.138	0.020	6376.500	<b>0.006</b>
QRS Interval(S)	125	0.080	0.006	125	0.080	0.008	7697.500	<b>0.730</b>

PR interval (mean) was prolonged among Smokers (0.138) compared to Non smokers (0.131), and it was statistically significant (P-value – 0.006). but there was no statistical significance (P-value – 0.730) in QRS interval (mean) between smokers (0.080) and Non smokers (0.080).

**GRAPH 3**

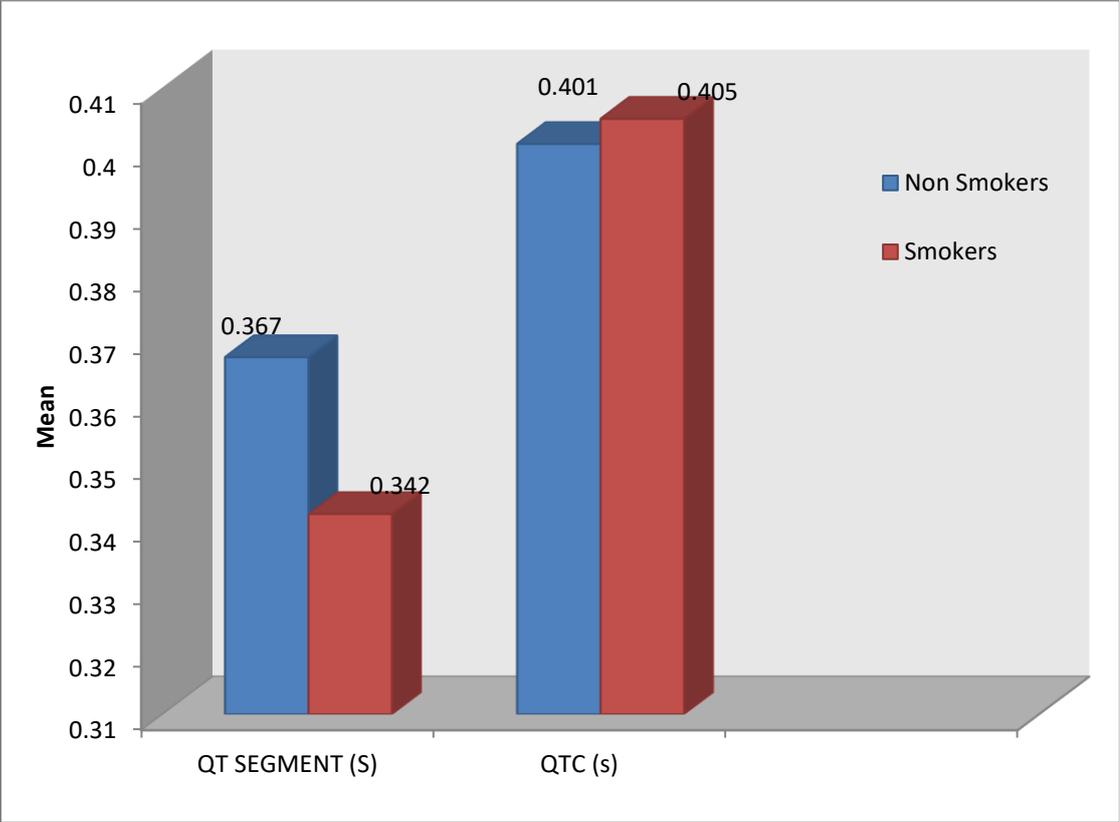


**Table 4: Comparison of Q.T. and QT<sub>c</sub> interval between Nonsmokers and Smokers.**

Variables	Non smokers			Smokers			Mann-Whitney U Value	P-value
	N	Mean	SD	N	Mean	SD		
QT SEGMENT (S)	125	0.367	0.021	125	0.342	0.019	12758.00	< <b>0.001</b>
QTC (s)	125	0.401	0.010	125	0.405	0.048	8442.00	<b>0.267</b>

QT interval (mean) was less among Smokers (0.342) compared to Non Smokers (0.367) and this was statistically significant ( P-value - <0.001). But QT<sub>c</sub> interval (mean) was slightly high among Smokers (0.405) compared to Non smokers (0.401) and this was found to be statistically insignificant (P value- 0.267).

**GRAPH 4**



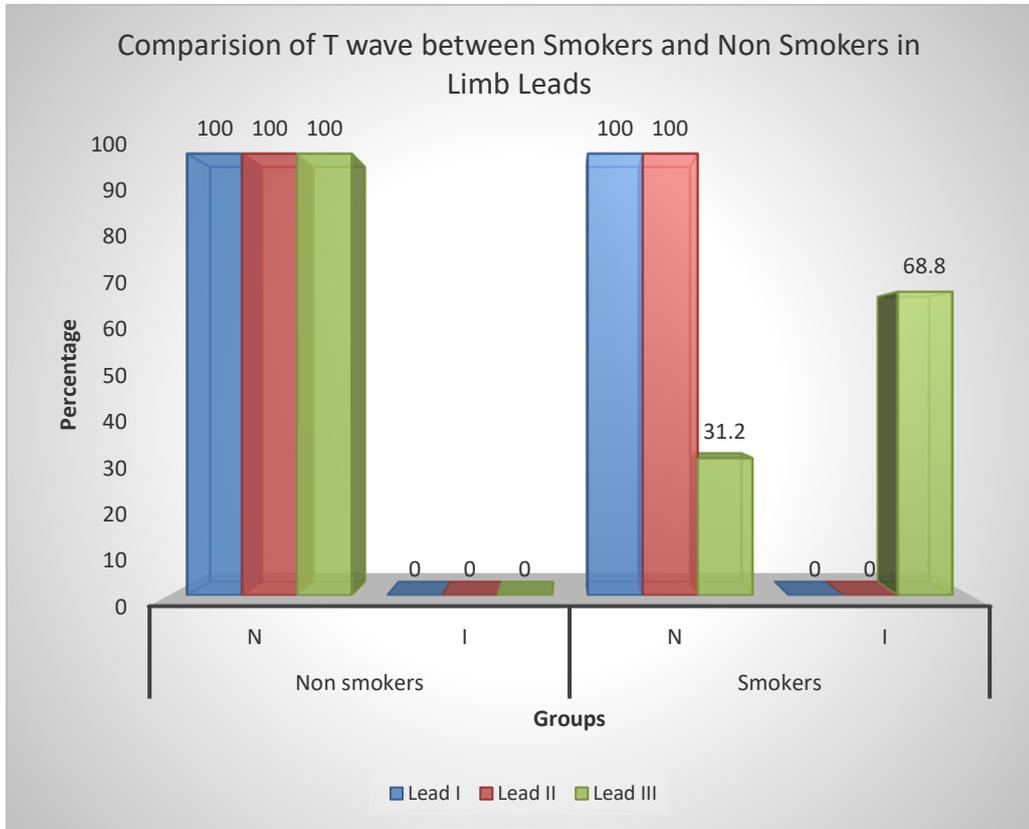
**Table 5: Comparison of T wave in limb leads between Non Smokers and Smokers.**

Limb Leads	Groups								Chi-square Value	P-value
	Non Smokers				Smokers					
	N	%	I	%	N	%	I	%		
Lead I	125	100	0	0	125	100	0	0	-----	-----
Lead II	125	100	0	0	125	100	0	0	-----	-----
Lead III	125	100	0	0	39	31.2	86	68.8	135.802	<b>0.000</b>

N= Normal    I = Inverted

Both Smokers and Non Smokers had normal T wave in Lead I and Lead II Whereas in Lead III, 31.2 % of Smokers had normal wave, 68.8% had inverted wave compared 100 % normal wave among Non Smokers and this difference was found to be statistically significant, thatishighest percentage of inverted waves found in Smokers (P-value – 0.000).

**GRAPH 5**



**Table 6: Comparison of T wave in augmented leads between Non Smokers and Smokers.**

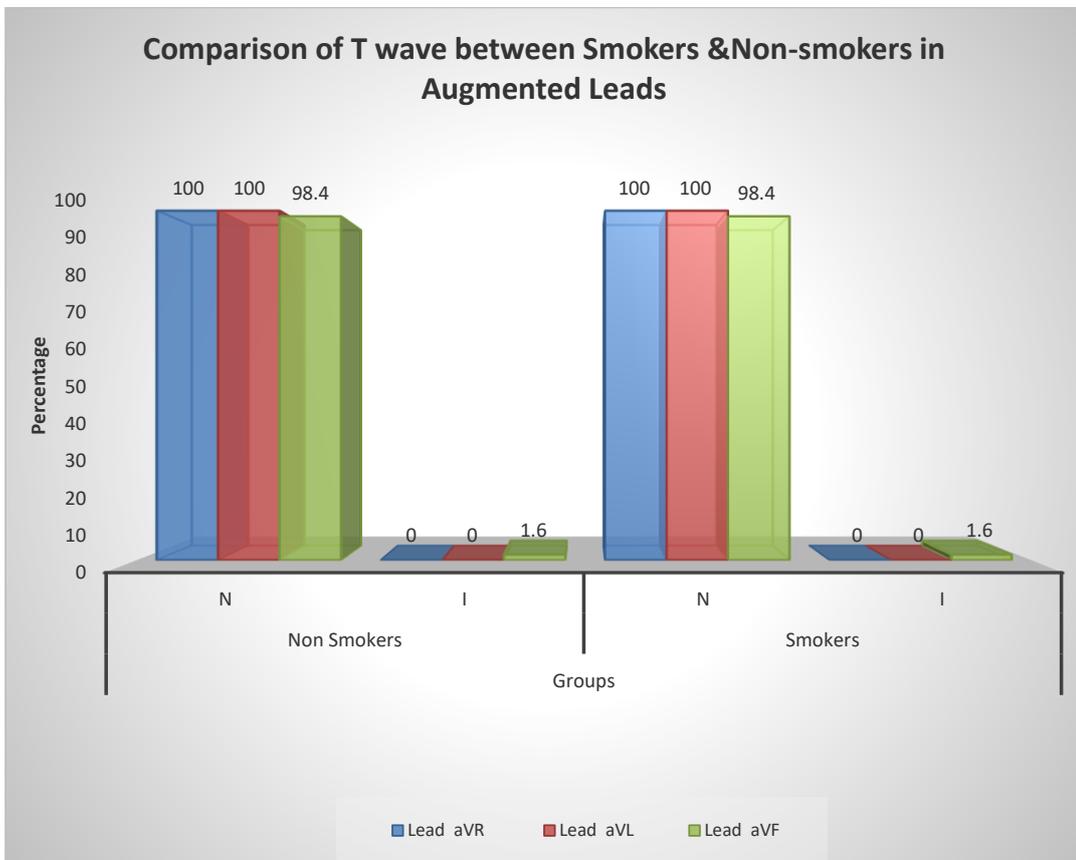
Augmented Leads	Groups								Chi-square Value	P-value
	Non Smokers				Smokers					
	N	%	I	%	N	%	I	%		
Lead aVR	125	100	0	0	125	100	0	0	-----	-----
Lead aVL	125	100	0	0	125	100	0	0	-----	-----
Lead aVF	123	98.4	2	1.6	123	98.4	2	1.6	0.000	<b>1.000</b>

N = Normal

I = Inverted

Both Smokers and Non Smokers had normal T wave in Lead aVR and Lead aVL, whereas in Lead aVF, 98.4% of Smokers had normal wave and 1.6 % had inverted wave compared to 98.4% normal wave and 1.6 % inverted wave among Non Smokers and this difference was found to be statistically insignificant .

**GRAPH 6**



**Table 7: Comparison of T wave in Chest leads between Non Smokers and Smokers.**

Chest Leads	Groups								Chi-square Value	P-value
	Non Smokers				Smokers					
	N	%	I	%	N	%	I	%		
Lead V1	122	97.6	3	2.4	61	49	64	51	75.87	<b>0</b>
Lead V2	121	96.8	4	3.2	105	84	20	16	11.799	<b>0.001</b>
Lead V3	123	98.4	2	1.6	112	90	13	10	8.582	<b>0.003</b>
Lead V4	124	99.2	1	0.8	118	94	7	5.6	4.949	<b>0.176</b>
Lead V5	125	100	0	0	125	100	0	0	-----	-----
Lead V6	125	100	0	0	125	100	0	0	-----	-----

N= Normal

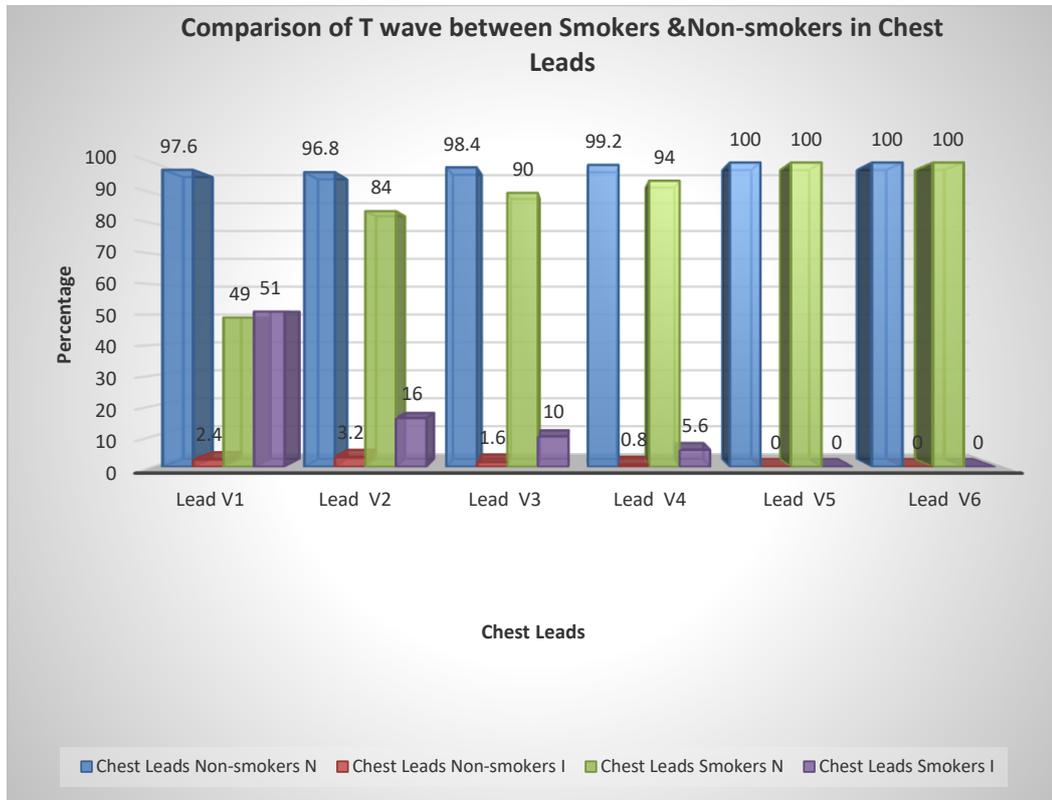
I = Inverted

Non Smokers had 2.4%, 3.2%, 1.6%, 0.8% of inverted T wave in Lead V1,V2,V3,V4 respectively, and 100% of normal wave were present in Lead V5 and Lead V6.

51%,16%,10%, 5.6% of Smokers inverted T wave in Lead V1,V2,V3,V4 respectively ,and 100% of the normal wave were present in Lead V5 and Lead V6.

The difference in the presence of inverted T wave among Smokers and Non Smokers in Lead V1 to V4 was found to be statistically significant.

**Graph7**

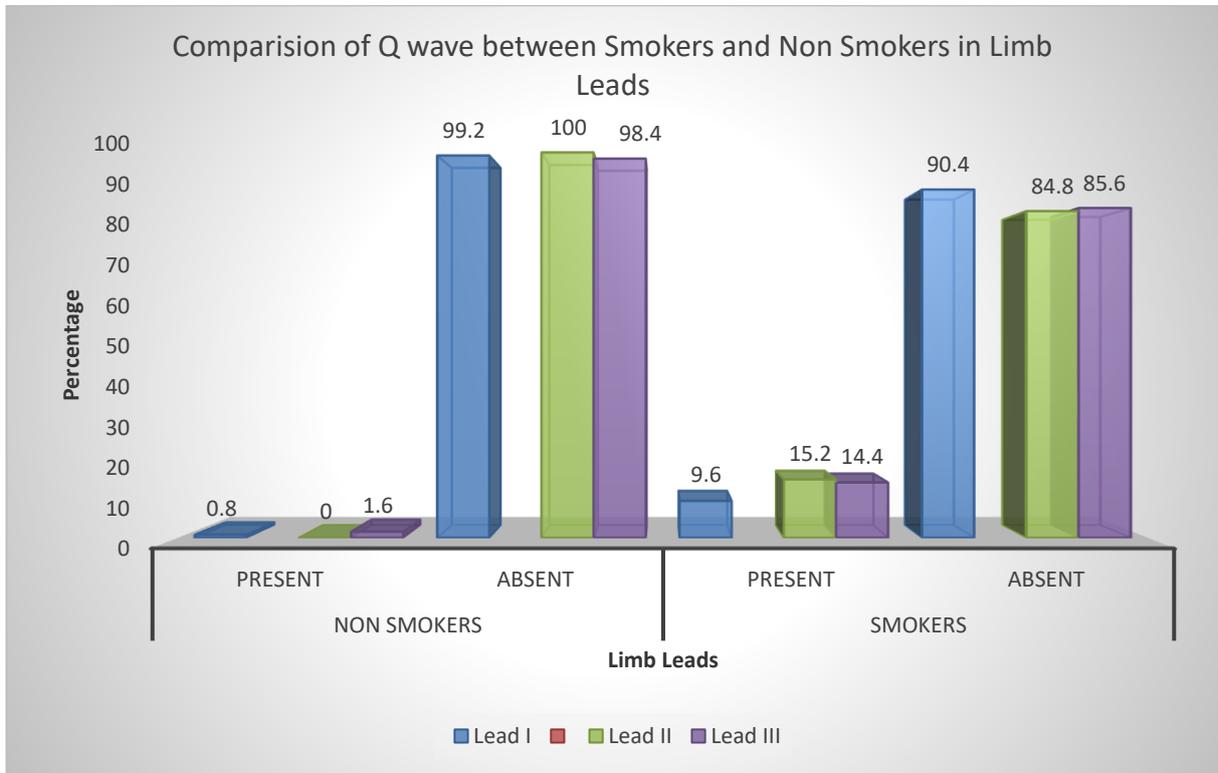


**Table 8: Comparison of Q wave in limb leads between Non-Smokers and Smokers.**

Limb Leads	Groups								Chi-square Value	P-value
	Non Smokers				Smokers					
	Present	%	Absent	%	Present	%	Absent	%		
Lead I	1	0.8	124	99.2	12	9.6	113	90.4	9.818	<b>0.002</b>
Lead II	0	0	125	100	19	15.2	106	84.8	20.563	<b>0.000</b>
Lead III	2	1.6	123	98.4	18	14.4	107	85.6	13.913	<b>0.000</b>

Q waves were present among 9.6%, 15.2%, 14.4% of Smokers compared to 0.8%, 0%, 1.6% among Non Smokers in Lead I, II, III, respectively . This difference is statistically significant.

**Graph 8**

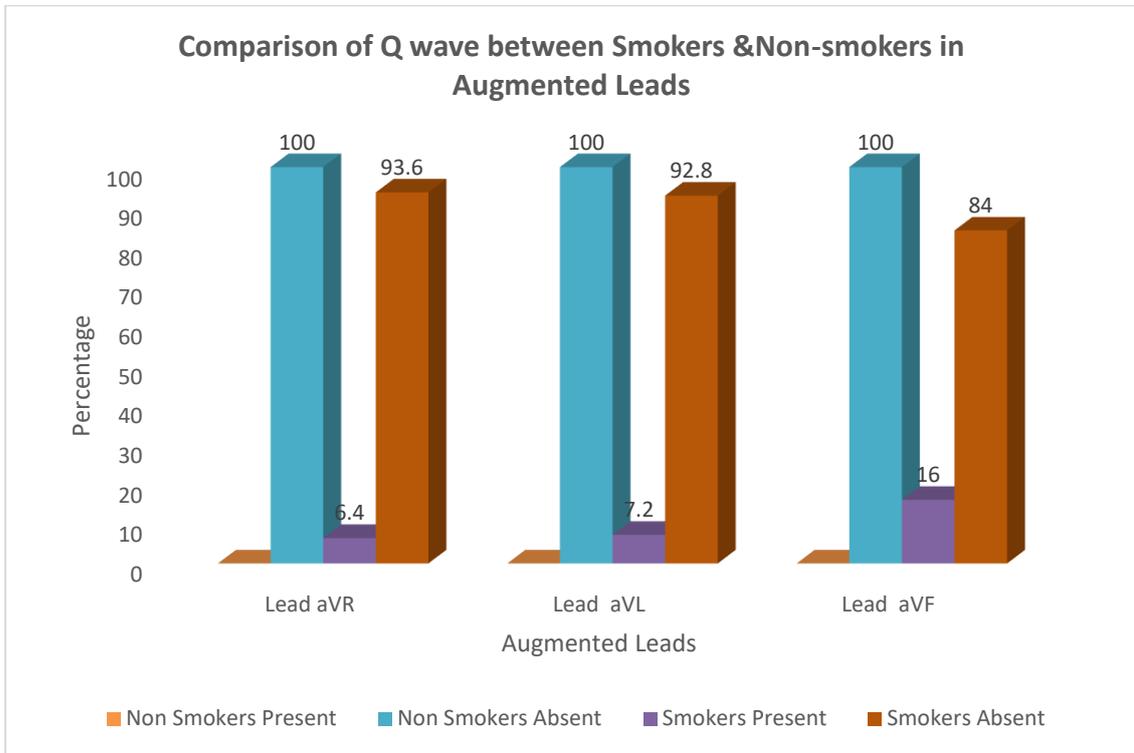


**Table 9: Comparison of Q wave in augmented leads between Non Smokers and Smokers.**

Augmented Leads	Groups								Chi-square Value	P-value
	Non Smokers				Smokers					
	Present	%	Absent	%	Present	%	Absent	%		
Lead aVR	0	0	125	100	8	6.4	117	93.6	250.00	<b>0.000</b>
Lead aVL	0	0	125	100	9	7.2	116	92.8	9.336	<b>0.002</b>
Lead aVF	0	0	125	100	20	16	105	84	21.739	<b>0.000</b>

Q waves are absent among Non-smokers in all three augmented leads, whereas among smokers, 6.4%, 7.2%, and 16% of Q waves are present in Lead aVR, aVL, and aVF, respectively and it is statistically significant.

**Graph 9**



**Table 10: Comparison of Q wave in Chest leads between Non-Smokers and Smokers.**

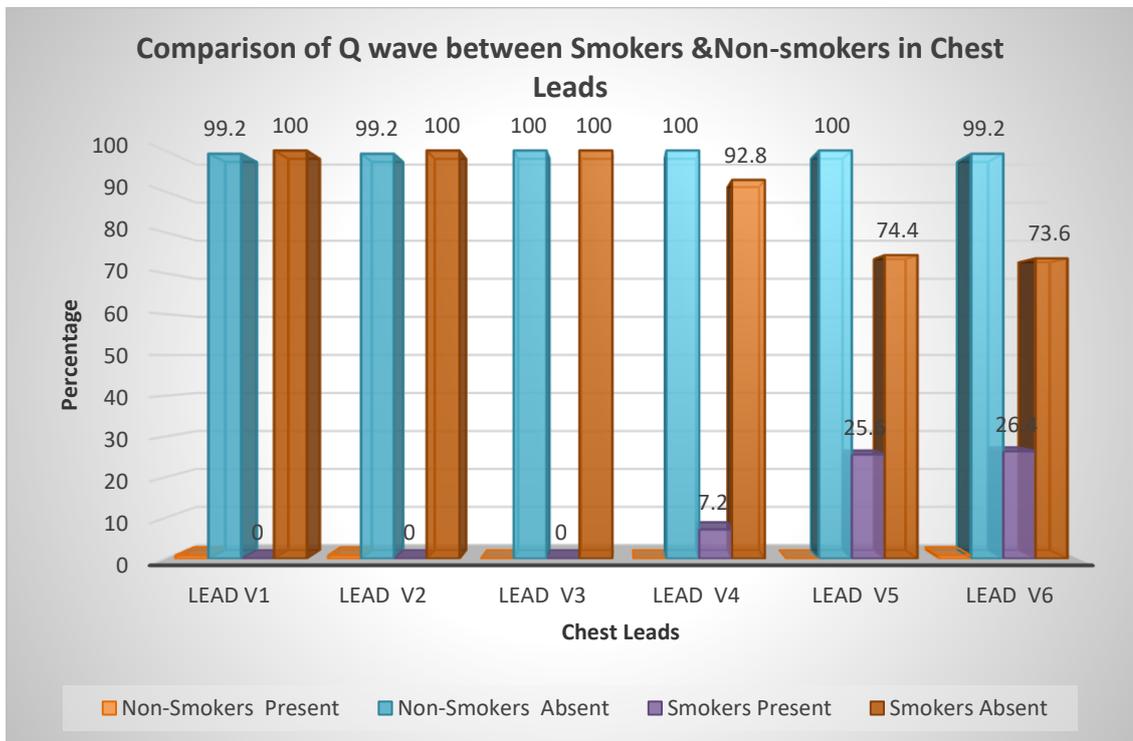
Chest Leads	Groups								Chi-square Value	P-value
	Non Smokers				Smokers					
	Present	%	Absent	%	Present	%	Absent	%		
Lead V1	1	0.8	124	99.2	0	0	125	100	1.004	<b>0.316</b>
Lead V2	1	0.8	124	99.2	0	0	125	100	1.004	<b>0.316</b>
Lead V3	0	0	125	100	0	0	125	100	-----	-----
Lead V4	0	0	125	100	9	7.2	116	92.8	9.336	<b>0.002</b>
Lead V5	0	0	125	100	32	25.6	93	74.4	250	<b>0</b>
Lead V6	1	0.8	124	99.2	33	26.4	92	73.6	34.87	<b>0</b>

There was no statistically significant difference in the occurrence of Q wave between smokers and Non Smokers in Lead V1 and V2.

No Q wave was seen among both Smokers and Non smokers in Lead V3.

Q waves among smokers were 7.2%, 25.6%, 26.4% present in Lead V4, V5,V6 respectively whereas no Q waves is present in Lead V4 and V5, and 0.8% Q waves are present in V6, and this difference is statistically significant in Lead V4,V5, V6.

**Graph 10**



# **DISCUSSION**

## DISCUSSION

In this study ECG changes are compared between Smokers and Non Smokers and results analysed.

### **Heart Rate:**

In this study Heart rate (mean) was high among smokers (85.904) compared to Non smokers (80.560) and this difference was found to be statistically significant (P-value – <0.001).

Mallikarjun v et.al<sup>26</sup>, Nirmal Kumar Sharma et.al<sup>31</sup> and K Harikrishnan et.al<sup>33</sup>, in both studies, found statistically significant increased heart rate among Smokers.

The increase in heart rate could be due to stimulation of sympathetic ganglia and discharge of catecholamines from adrenal medulla<sup>32</sup>.

Heart Rate(bpm) (mean)	Nirmal Kumar Sharma et.al	K Harikrishnan et.al	Our study
Smokers	Increased in Smokers	86.11	85.904
Non Smokers		77.38	80.560

### **P – wave:**

In this study there was no much difference in duration (mean) of P wave between Non smokers (0.083) and Smokers (0.083), and it was not statistically significant (P-value - 0.664). And amplitude (mean) was slightly high among smokers (1.070) compared to Nonsmokers (1.010), which was statistically significant (P-value - <0.001). Swathi k et al. <sup>27</sup>, Dr.Domala Prasad et al. <sup>29</sup>, Chandrasekhar Athikari et al.<sup>32</sup> and K Harikrishnan et.al<sup>33</sup> also found amplitude was slightly high among smokers compared to Non-Smokers.

An increase in P wave amplitude might be due to the reduced right ventricular compliance, subsequently producing right atria hypertrophy as a result of chronic smoking<sup>32</sup>.

P wave Amplitude (mean)	Swathi k et al	Dr.Domala Prasad et al	K Harikrishnan et.al	Our study
Smokers	1.220	1.067	1.3080	1.070
Non smokers	1.060	1.030	1.165	1.010

### **P.R. interval:**

In this study, the P.R. interval (mean) was prolonged among Smokers (0.138) compared to Non-smokers (0.131) and it was statistically significant (P-value – 0.006). Swathi k et al.<sup>27</sup> shows prolongation of P.R. interval among smokers whereas Mallikarjun v and et.al<sup>26</sup>, Dr.Domala Prasad et al.<sup>29</sup>, Chandrasekhar Athikari et al.<sup>32</sup>, AmolGhule et.al<sup>34</sup> and AshishLakhanpal et.al<sup>35</sup> studies show decrease in P.R. interval among Smokers.

PR interval (S) (mean)	Swathi k et al.	Chandrasekhar Athikari et al	Amol Ghule et.al	Ashish Lakhanpal et.al	Our study
Smokers	0.1560	0.131	0.159	0.160	0.138
Non Smokers	0.1470	0.142	0.167	0.180	0.131

### **QRS Complex:**

In this study but there was no statistical significance (P-value – 0.730) in QRS interval (mean) between smokers (0.080) and Non smokers (0.080). In Swathi k, et.al.<sup>27</sup>, Chandrasekhar

Athikari et.al.<sup>32</sup> , and Ashish Lakhanpal et.al<sup>35</sup> shows reduced QRS interval among Smokers, which not statistically significant.

**Q.T. interval:**

In this study, Q.T. interval (mean) was reduced among Smokers (0.342) compared to Non-Smokers (0.367), and this was statistically significant ( P-value - <0.001).Mallikarjun v et.al<sup>26</sup> Chandrasekhar Athikari et al.<sup>32</sup> and AshishLakhanpal et.al<sup>35</sup> also show decreased Q.T. interval, but it was not statistically significant.

**QTc interval:**

In this study, QTc interval (mean) was slightly high among Smokers (0.405) compared to Non smokers (0.401) and this was found to be statistically insignificant (P value- 0.267). Mallikarjun v et.al<sup>26</sup>, Vasant Deokar et al.<sup>28</sup>, K Harikrishnan et.al<sup>33</sup> and Amol Ghule et.al<sup>34</sup> also shown statistically significant QTc prolongation.

The ventricular repolarization is altered in young male cigarette smokers. The differences in the heterogeneity of ventricular repolarization between smokers and non-smokers are mainly due to heart rate differences between the 2 study groups<sup>28</sup>.

QTc interval (s) (mean)	Mallikarjun v et.al	Vasant Deokar et al.	K Harikrishnan et.al	Amol Ghule et.al	Our study
Smokers	0.390	0.384	0.364	0.480	0.405
Nonsmokers	0.378	0.378	0.361	0.420	0.401

**T wave:**

In this study both Smokers and Non Smokers had normal T wave in Lead I and Lead II, Whereas in Lead III, 31.2 % of Smokers had normal wave, 68.8% had inverted waves compared to 100 % normal wave among Non Smokers and this difference was found to be statistically significant, that is highest percentage of inverted waves found in Smokers (P-value – 0.000).

Both Smokers and Non Smokers had normal T wave in Lead aVR and Lead aVL, whereas in Lead aVF, 98.4% of Smokers had normal wave and 1.6 % had inverted wave compared to 98.4% normal wave and 1.6 % inverted wave among Non Smokers and this difference was found to be statistically insignificant .

Non Smokers had 2.4%, 3.2%, 1.6%, 0.8% of inverted T wave in Lead V1,V2,V3,V4 respectively, and 100% of normal wave were present in Lead V5 and Lead V6. 51%,16%,10%, 5.6% of Smokers inverted T wave in Lead V1,V2,V3,V4 respectively ,and 100% of normal wave were present in Lead V5 and Lead V6. Difference in presence of inverted T wave among Smokers and Non Smokers in Lead V1 to V4 found to be statistically significant. Swathi k et.al.<sup>27</sup> shows 18% abnormal T wave among Smokers.

T wave inversions may be due to prolonged repolarization leading to ischemia<sup>29</sup>.

### **Q wave:**

Q waves was present among 9.6%, 15.2%, 14.4% of Smokers compared to 0.8%, 0%,1.6% among Non Smokers in Lead I,II,III respectively . This difference is statistically significant.

Q waves are absent among Non-smokers in all three augmented leads , whereas among smokers 6.4%, 7.2% and 16% of Q waves are present in Lead aVR, aVL and aVF respectively and it is statistically significant.

There was no statistically significant difference in occurrence of Q wave between smokers and Non Smokers in Lead V1 and V2.

No Q wave was seen among both Smokers and Non smokers in Lead V3.

Q waves among smokers is 7.2%, 25.6%, 26.4% present in Lead V4,V5,V6 respectively whereas no Q waves is present in Lead V4 and V5, and 0.8% Q waves are present in V6, and this difference is statistically significant in Lead V4,V5, V6.

# CONCLUSION

## CONCLUSION

- The following conclusions can be drawn from the results of this study.
- There was statistically significant increase in heart rate in smokers when compared to non-smokers.
- There was a statistically significant increase in P wave amplitude in smokers when compared to non-smokers.
- There was statistically significant prolongation in P.R. interval in smokers when compared to non-smokers.
- There was a statistically significant decrease in Q.T. interval in smokers when compared to non-smokers.
- There was a statistically significant decrease in T wave amplitude in smokers when compared to non-smokers.
- There was the statistically significant occurrence of Q wave in smokers when compared to non-smokers.
- Though our study is by no means exhaustive, it does provide a glimpse into the variety of ECG changes in the absence of any cardiac disease in smokers. Although we understand to some extent these changes and also since few studies have been done on this aspect, further research is needed to study the effect of Smoking on electrocardiogram.

# **SUMMARY**

## **SUMMARY**

The study entitled “A study of ECG changes in healthy smokers compared to non-smokers” was conducted in the department of medicine, BLDE(Deemed to be University) Shri B.M. Patil medical college, hospital and research centre, vijayapura during the period from 2019-2021. The aim of the study was to know the electrocardiographic changes in smokers when compared to Non Smokers.

The study included 125 smokers and 125 Non-smokers, each between a 20 - 40years, selected from patients and attendants of patient outpatient and inpatient department from Shri B.M. Patil medical college, hospital and research centre, Vijayapura.

After taking consent and a detailed history from the subjects, electrocardiogram was recorded during resting state in supine position. The ECG results were evaluated for different parameters like heart rate, P wave, PR interval, QRS complex, QT interval, QTc interval, ST segment, Q wave and T wave.

### **The results were as follows:**

There was statistically significant increase in heart rate, increase in P wave amplitude, increase in PR interval, decrease in QT interval, decrease in T wave amplitude, occurrence of Q wave in smokers when compared to non-smokers.

Though our study is by no means exhaustive, it does provide a glimpse into the variety of ECG changes in the absence of any cardiac disease in smokers. Although we understand to some extent these changes and also since few studies have been done on this aspect, further research is needed to study the effect of Smoking on electrocardiogram.

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## **ANNEXURE – I**

# ETHICAL CLEARANCE CERTIFICATE



IEC/NO-131/2019  
22/11/19

**B.L.D.E. (DEEMED TO BE UNIVERSITY)**

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act. 1956)  
The Constituent College

**SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE**

## **INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE**

The ethical committee of this college met on 13-11-2019 at 3-15 pm to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

**Title:** A study of ECG changes in healthy smokers compared to non smokers.

**Name of PG student:** Dr Chethan R Rajpurohit, Department of General Medicine

**Name of Guide/Co-investigator:** Dr Vijaykumar G Warad, Professor, Department of General Medicine

**DR RAGHVENDRA KULKARNI**  
**CHAIRMAN**  
Institutional Ethical Committee  
B.L.D.E.'s Shri B.M. Patil  
Medical College, Dahanu. Dist-586103

**Following documents were placed before Ethical Committee for Scrutinization:**

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

## ANNEXURE – II

### INFORMED CONSENT FORM

**TITLE OF RESEARCH:** A STUDY OF ECG CHANGES IN HEALTHY YOUNG SMOKERS COMPARED TO NON SMOKERS

GUIDE : DR VIJAYKUMAR G WARAD,<sup>M.D</sup>

P.G.STUDENT : DR CHETHAN R RAJPUROHIT

**All aspects of this consent form are explained to the patient in the language understood by him or her.**

#### **PURPOSE OF STUDY:**

I have been informed that the purpose of this study is to study diagnostic importance of platelet volume indices in patients with acute chest pain suggesting acute coronary syndrome.

#### **PROCEDURE:**

I understand that I will undergo detailed history and clinical examination and investigations

#### **BENEFITS:**

I understand that my participation in this study will have no direct benefit to me other than the potential benefit of treatment which is planned to prevent further morbidity and mortality in me.

#### **CONFIDENTIALITY:**

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulation of hospital. If the data is used for publication the identity will not be revealed.

**REQUEST FOR MORE INFORMATION:**

I understand that I may ask for more information about the study at any time.

**REFUSAL OR WITHDRAWAL OF PARTICIPATION:**

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

(Signature of patient)

(Signature of Guardian)

**STUDY SUBJECT CONSENT FORM:**

I confirm that **Dr.CHETHAN R RAJPUROHIT** has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all above in detail in my own language and I understand the same. I agree to give my consent to participate as a subject in this research project.

DATE:

DATE:

SIGNATURE OF PARTICIPANT :

SIGNATURE OF WITNESS:

## **ANNEXURE – III**

### **PROFORMA**

#### **SCHEME OF CASE TAKING**

Name:	CASE NO:
Age:	OP/IP NO:
Sex:	DOA:
Religion:	DOD:
Occupation:	
Address:	

**Presenting complaints with duration:**

**History of presenting complaints:**

**Past History:**

**Family History:**

**Personal History:**

**Treatment History:**

**General Physical Examination**

Pallor: present/absent

Icterus: present/absent

Cyanosis: present/absent

Clubbing: present/absent

Generalized lymphadenopathy: present/absent

Odema: present/absent

**VITALS:**

PR:

BP:        mm of mercury (mm hg)

RR:

Temp:

**SYSTEMIC EXAMINATION:**

1. Cardiovascular system
2. Respiratory system
3. Per abdomen
4. Central nervous system

## INVESTIGATIONS

### PATHOLOGY:

1.)Complete blood count:	
Hemoglobin	gm/dl
Total count	Cells/cumm
Differential countNeutrophils	%
Lymphocytes	%
Eosinophils	%
Basophils	%
Monocytes	%
2.) ESR	At the end of 1 <sup>st</sup> hour.
3.)Platelet Indices	
Platelet Count	
Mean Platelet Volume	
Platelet Distribution Width	
4.) Urine Routine	
Sugar	
Albumin	
Cell type	
Cell count	

**BIOCHEMISTRY:**

RBS	
B urea	
S creatinine	
serum sodium	
serum potassium	
serum chloride	
uric acid	

**ELECTROCARDIOGRAPHY:**

**CHEST X RAY PA VIEW**

Other relevant investigations will be done when required.

**DIAGNOSIS:**

**DATE:**

**SIGNATURE**









38	27	68	0.08	1	0.12	0.08	ISOELECTRIC	0.34	0.39	N	N	N	N	N	N	I	N	N	N	N	N	+	-	+	-	-	-	-	-	-	-	+	+
39	29	96	0.08	1	0.12	0.08	ISOELECTRIC	0.34	0.406	N	N	N	N	N	N	I	N	N	N	N	N	+	-	-	+	-	+	-	-	-	-	+	+
40	30	86	0.08	1	0.16	0.08	ISOELECTRIC	0.34	0.42	N	N	N	N	N	N	I	N	N	I	N	N	-	+	-	-	-	+	-	-	-	-	-	+
41	32	88	0.08	1	0.12	0.08	ISOELECTRIC	0.4	0.392	N	N	N	N	N	N	N	N	N	N	N	N	-	+	-	-	-	+	-	-	-	-	-	-
42	33	90	0.08	1	0.12	0.08	ISOELECTRIC	0.4	0.412	N	N	N	N	N	N	I	I	N	N	N	N	-	+	-	-	-	-	-	-	-	-	+	+
43	35	96	0.08	1	0.12	0.08	ISOELECTRIC	0.34	0.382	N	N	N	N	N	N	I	N	N	N	N	N	-	-	-	-	+	-	-	-	-	-	-	-
44	39	84	0.08	1.2	0.2	0.1	ISOELECTRIC	0.36	0.396	N	N	N	N	N	N	I	N	I	N	N	N	-	-	+	-	-	-	-	-	-	-	-	-
45	28	96	0.08	1.2	0.14	0.08	ISOELECTRIC	0.34	0.4	N	N	I	N	N	N	I	N	N	N	N	N	-	-	+	-	-	-	-	-	-	-	+	-
46	32	72	0.08	1	0.14	0.08	ISOELECTRIC	0.32	0.391	N	N	I	N	N	N	N	N	N	N	N	N	-	+	+	-	-	-	-	-	-	-	+	-
47	35	84	0.08	1.6	0.16	0.08	ISOELECTRIC	0.34	0.412	N	N	I	N	N	N	I	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	+	-
48	38	96	0.08	1	0.16	0.08	ISOELECTRIC	0.36	0.382	N	N	N	N	N	N	I	N	N	N	N	N	-	-	-	+	-	+	-	-	-	-	+	+
49	33	102	0.08	1	0.12	0.1	ISOELECTRIC	0.34	0.392	N	N	I	N	N	N	I	N	N	N	N	N	+	-	-	-	-	-	-	-	-	-	+	+
50	35	88	0.08	1	0.12	0.08	ISOELECTRIC	0.34	0.4	N	N	N	N	N	N	N	N	N	N	N	N	-	+	-	-	-	-	-	-	-	-	-	-
51	36	72	0.08	1	0.12	0.08	ISOELECTRIC	0.32	0.398	N	N	I	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
52	37	76	0.08	1	0.14	0.08	ISOELECTRIC	0.34	0.392	N	N	I	N	N	N	I	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
53	23	84	0.08	1	0.14	0.08	ISOELECTRIC	0.36	0.394	N	N	I	N	N	N	I	I	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
54	28	86	0.08	1	0.16	0.06	ISOELECTRIC	0.34	0.412	N	N	I	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
55	26	88	0.08	1.2	0.12	0.08	ISOELECTRIC	0.34	0.422	N	N	I	N	N	N	I	N	N	N	N	N	-	-	-	-	+	-	-	-	-	-	-	-
56	26	76	0.08	1	0.18	0.08	ISOELECTRIC	0.34	0.424	N	N	I	N	N	N	I	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
57	32	74	0.08	1	0.14	0.08	ISOELECTRIC	0.4	0.392	N	N	I	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
58	36	86	0.09	1.2	0.12	0.08	ISOELECTRIC	0.34	0.396	N	N	I	N	N	N	I	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
59	38	112	0.08	1	0.16	0.08	ISOELECTRIC	0.34	0.391	N	N	I	N	N	N	N	N	I	N	N	N	-	-	-	+	-	-	-	-	-	-	-	-
60	32	104	0.08	1	0.14	0.08	ISOELECTRIC	0.32	0.396	N	N	I	N	N	N	N	N	N	N	N	N	-	+	-	-	-	-	-	-	-	-	-	-
61	25	98	0.08	1	0.12	0.1	ISOELECTRIC	0.34	0.412	N	N	N	N	N	N	N	I	N	N	N	N	-	-	+	-	-	-	-	-	-	-	-	-
62	26	86	0.08	1	0.16	0.08	ISOELECTRIC	0.34	0.396	N	N	I	N	N	N	N	N	N	N	N	N	+	-	+	-	-	-	-	-	-	-	-	-
63	30	84	0.08	1	0.16	0.08	ISOELECTRIC	0.34	0.4	N	N	I	N	N	N	N	N	N	I	N	N	-	-	-	-	-	+	-	-	-	-	-	+
64	31	88	0.09	1	0.14	0.08	ISOELECTRIC	0.32	0.42	N	N	I	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	+	-
65	28	86	0.08	1.2	0.12	0.08	ISOELECTRIC	0.34	0.391	N	N	I	N	N	N	N	N	N	N	N	N	-	+	-	-	-	-	-	-	-	-	-	-
66	26	92	0.08	1	0.14	0.06	ISOELECTRIC	0.34	0.396	N	N	N	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
67	32	90	0.1	1	0.12	0.08	ISOELECTRIC	0.34	0.42	N	N	I	N	N	N	N	N	N	N	N	N	+	-	-	-	+	-	-	-	-	-	-	-
68	35	86	0.08	1	0.14	0.08	ISOELECTRIC	0.36	0.42	N	N	I	N	N	N	N	N	N	N	N	N	-	-	-	+	-	-	-	-	-	-	-	-
69	27	74	0.08	1	0.12	0.08	ISOELECTRIC	0.34	0.392	N	N	I	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
70	33	76	0.08	1	0.16	0.08	ISOELECTRIC	0.36	0.396	N	N	N	N	N	N	I	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
71	29	82	0.08	1	0.12	0.08	ISOELECTRIC	0.34	0.412	N	N	I	N	N	N	I	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	+	-
72	36	96	0.09	1	0.14	0.1	ISOELECTRIC	0.34	0.398	N	N	N	N	N	N	I	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
73	35	102	0.08	1	0.12	0.08	ISOELECTRIC	0.34	0.392	N	N	I	N	N	N	N	N	I	N	N	N	+	-	-	-	-	-	-	-	-	-	-	-
74	32	68	0.08	1	0.14	0.08	ISOELECTRIC	0.34	0.4	N	N	I	N	N	N	N	N	N	I	N	N	-	-	+	-	-	+	-	-	-	-	-	-
75	34	74	0.08	1	0.12	0.08	ISOELECTRIC	0.4	0.396	N	N	N	N	N	N	N	I	N	N	N	N	+	-	+	-	-	-	-	-	-	-	-	-
76	33	86	0.08	1	0.14	0.08	ISOELECTRIC	0.34	0.382	N	N	I	N	N	N	N	N	N	N	N	N	-	-	+	-	-	-	-	-	-	-	-	-
77	26	94	0.1	1.2	0.12	0.08	ISOELECTRIC	0.34	0.412	N	N	I	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
78	25	96	0.08	1	0.16	0.1	ISOELECTRIC	0.34	0.396	N	N	N	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	+	-
79	33	86	0.08	1	0.18	0.08	ISOELECTRIC	0.34	0.412	N	N	N	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	+	-
80	35	72	0.08	1	0.12	0.08	ISOELECTRIC	0.32	0.391	N	N	N	N	N	N	N	N	N	N	N	N	-	-	-	-	-	+	-	-	-	-	-	+
81	33	84	0.08	1	0.14	0.08	ISOELECTRIC	0.32	0.396	N	N	I	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	+	-	-
82	29	86	0.1	1	0.12	0.08	ISOELECTRIC	0.34	0.392	N	N	I	N	N	N	N	N	N	N	N	N	-	-	-	-	-	-	-	-	-	-	-	-
83	32	72	0.08	1	0.14	0.08	ISOELECTRIC	0.34	0.422	N	N	I	N	N	N	N	I	N	N	N	N	-	-	-	+	-	-	-	-	-	-	-	-

