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"A PROSPECTIVE OBSERVATIONAL STUDY OF AUTONOMIC DYSFUNCTION IN CIRRHOSIS OF LIVER AND ITS CORRELATION WITH ELECTROCARDIOGRAPHY AND ECHOCARDIOGRAPHY"

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DR.MUDIREDDY BINDU BHAVANI

ABSTRACT

Background: Cirrhosis is a chronic liver condition characterized by hepatic fibrosis, anatomical distortion, and compromised liver function. Autonomic dysfunction (AD) is a significant concern due to its impact on cardiovascular stability, hemodynamic modulation, and patient prognosis. AD is characterized by irregularities in heart rate variability, impaired blood pressure management, and abnormal reflex reactions, which can increase the risk of cardiac events. Cirrhotic cardiomyopathy, characterized by compromised ventricular contractility and electromechanical dysfunction, is linked to autonomic abnormalities. ECG and ECHO are vital tools for assessing heart function in cirrhosis patients, revealing anatomical and functional heart alterations.

Objective: This study aims to evaluate autonomic dysfunction in individuals with liver cirrhosis, its impact on ECG abnormalities, heart rate variability, blood pressure regulation, and cardiovascular reflexes, and its influence on various Child-Pugh and MELD score groups. It also seeks to identify potential predictors of autonomic dysfunction in cirrhosis, which could aid in early risk assessment and therapeutic management.

Methods: A retrospective study was conducted on clinical records of 100 patients diagnosed with cirrhosis over an 18-month period, spanning from May 2023 to December 2024, at Shri B M Patil Medical College and Research Center, Vijayapura. The collected data encompassed patient demographics, clinical presentation at the time of admission, ECG findings (QTc interval), echocardiographic assessments, and indicators of autonomic dysfunction.

Results: The study analyzed the age distribution and physiology of patients with heart conditions, focusing on the majority of patients aged 20-60 years. The majority were male, with 95% being male and 5% female. Pulse rates were categorized into three ranges: 81-100 bpm, 60-80 bpm, and 101-130 bpm. The Valsalva maneuver showed a similar distribution, with 52% falling in the 81-100 bpm range and 36% in the 60-80 bpm range. Blood pressure was measured using a blood pressure cuff, with higher pressure indicating a higher risk of heart failure.

The study also examined blood pressure readings under three conditions: Supine BP (lying down), Standing BP, and Hand Grip BP. The most common reading in the supine position was 110/70, indicating normal resting BP. The ECG showed various normal and abnormal findings, with the majority having normal function.

The Child-Pugh classification assessed the severity of chronic liver disease, categorizing patients into three classes: A (mild), B (moderate), and C (severe). In-patients with the chronic liver illness, particularly those are being considered for a liver transplant, the risk of mortality was estimated using the MELD score.The study revealed that the majority of participants were middle-aged, the supine pulse rate showed a significant variation, and the rise in pulse rate upon standing was within the expected physiological range. Additionally, the MELD score served as a key indicator for determining the severity of liver disease. Statistical analysis confirmed significant deviations from expected baselines ($p < 0.001$). These findings highlight the strong link between autonomic dysfunction, cardiovascular abnormalities, and liver disease progression.

Conclusion: The study reveals a significant gender disparity in the population, with 95% being males. Cardiovascular assessments show normal physiological responses, but some individuals show signs of autonomic dysfunction. ECG analysis reveals abnormalities in sinus rhythms, highlighting the need for continuous monitoring. Liver function assessments reveal a high prevalence of severe liver disease, necessitating urgent medical interventions. Early detection and management of these health issues are crucial for improving health outcomes. Future research should focus on lifestyle modifications, targeted treatments, and long-term monitoring.

LIST OF ABBREVIATIONS

ECG	Electrocardiogram
HRV	Hear Rate Variability
ANS	Autonomic Nervous System
HBF	Fetal Hemoglobin
SDNN	Standard Deviation of Normal-to-Normal Intervals
NAFLD	Non- Alcoholic Fatty Liver Disease
HCV	Hapatitis C Virus
SVR	Systemic Vascular Resistance
HR	Heart Rate
RMSSD	Root Mean Square of Successive Differences
CO	Cardiac Output
LC	Liver cirrhosis
LVH	Left Ventricular Hypertrophy
NASH	Non- alcoholic steatohepatitis
TIPS	Transjugular Intrahepatic Protosystemic shunt
PBC	Primary Biliary Cholangitis
CVD	Cardiovascular Disease

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1. INTRODUCTION

During the mid-20th century, doctors observed circulatory anomalies in patients suffering from liver cirrhosis. These preliminary observations established the foundation for comprehending the interaction between hepatic and cardiac processes. During the 1980s and 1990s, investigations intensified on the autonomic nervous system's involvement in cirrhosis. A circulatory condition marked by escalated cardiac output & decreased systemic vascular action has been identified. This indicated a significant autonomic imbalance and was linked to decreased parasympathetic tone and increased sympathetic activity. The notion of cirrhotic cardiomyopathy has arisen, characterizing cardiac dysfunction in cirrhotic patients without a history of heart disease, distinguished by diminished contractile response to stress and electrophysiological abnormalities[1].

In both health and disease, the liver and heart are intimately related organs. According to conventional medicine, each organ in the body has a unique temperament that is defined by four characteristics, or elements: "warmth," "coldness," "wetness," and "dryness." While "warmth" and "coldness" can be thought of as the basic metabolic states of the organ, "wetness" and "dryness" are on a continuum of "tissue moisture." Avicenna emphasized specific interaction effects on the liver and heart in his well-known treatise, "Canon" (The Law). The domination of "heart warmth" over "liver coldness" and the superiority of "liver dryness" over "heart wetness" are important considerations. The diagnosis, assessment of the overall prognosis, and course of treatment for liver disease may all be significantly impacted by the influence and role of "heart temperament" and its impact on "liver temperaments" [1, 2].

The incorporation of ECG and Echocardiography into clinical practice offers non-invasive methods for evaluating heart function in patients with cirrhosis. The prolongation of the QT interval emerged as a significant ECG finding, occurring in up to 50% of cirrhotic patients, associated with disease severity and potentially resulting in severe ventricular arrhythmias[2]. Echocardiographic examinations indicated diastolic dysfunction, marked by compromised ventricular relaxation and heightened myocardial stiffness, which are integral to the characterization of cirrhotic cardiomyopathy[1]. Recent meta-analyses have confirmed diminished heart rate variability (HRV) in cirrhotic patients, highlighting the significance of autonomic dysfunction in disease advancement. Particular HRV indices, including SDNN and RMSSD, exhibited a considerable reduction, corresponding with the severity of cirrhosis and patient survival[1]. These findings underscore the predictive significance of autonomic evaluations in the therapy of cirrhosis.

Liver cirrhosis can show up in a lot of different organs besides the liver. In most cases, the heart is affected, and the dysfunction can be mild to severe. The latter is more prevalent in advanced liver disease[3]. Alterations in portal pressure and hepatic blood flow (HBF) primarily initiate hyperdynamic circulation and central hypovolemia. Central hypovolemia, along with signs of liver failure, correlates with a poor prognosis. When someone has cirrhosis with portal hypertension, their circulatory system doesn't work right because their peripheral vessels keep getting bigger. This means that organs don't get enough blood flow, so their heart has to work harder to make up for it[4]. Due to hyperdynamic circulation necessitates an raised heart rate (HR), this is monitored by the autonomic nervous system (ANS). Autonomic dysfunction is characterized by irregular cardiovascular test results in the absence of noticeable clinical symptoms. A problem with the autonomic nervous system can cause an irregular heart rate response, especially when the body needs more oxygen, like when you're exercising or under a

lot of stress[5,6]. It is known that cirrhotic cardiomyopathy is linked to QT interval prolongation, which is a sign of electrophysiological irregularities and autonomic dysfunction[7]. Heart rate variability (HRV) is used to assess the autonomic regulation of the heart by analyzing a series of normal R-R intervals, also known as N-N intervals, over a specific time frame. It reflects the heart's capacity to adjust its rate in response to dynamic conditions by detecting and rapidly reacting to various stimuli. It is known that liver cirrhosis causes autonomic dysfunction, which shows up as an imbalance in the autonomic nervous system caused by a lot less parasympathetic activity and more sympathetic tone[8].

Cirrhosis is linked to increased circulating blood volume, decreased systemic vascular resistance, and elevated cardiac output (CO). While systolic heart function in individuals with cirrhosis is typically adequate at rest, it may exhibit dysfunction under pharmacological or physical stress. They frequently exhibit diastolic dysfunction while at rest. These anomalies are collectively referred to as "Cirrhotic Cardiomyopathy." Electrocardiogram Alterations-A prolonged QT interval indicates individuals at heightened risk of abrupt cardiac mortality in many situations, such as alcoholic liver illness and diminished R Internal. Echo findings indicate left ventricular diastolic dysfunction and observed systolic dysfunctions. Cardiac involvement elevates morbidity and death in individuals with cirrhosis. Cirrhotic cardiomyopathy significantly contributes to perioperative morbidity and mortality in liver transplant recipients. Echocardiography, utilizing both conventional and deformation imaging techniques, is quite effective for identifying these anomalies.

The adoption of advanced diagnostic techniques has identified several assessments of dysfunctional cardiac contractility. Functioning individuals with cirrhosis, leads to the recognition of a condition termed cirrhotic cardiomyopathy. This condition is characterized by reduced myocardial contractility under physical or pharmacological

stress; however, it remains under diagnosed, and the implicit mechanisms of cardiac dysfunction are not yet understood. This leads to explore the causes and clinical manifestations of heart dysfunction in cirrhosis[9].

Liver Disorders Impacting Heart Function

Persistent Hepatitis C Infection

In the hepatitis C virus (HCV) related heart illness, many cases experience chronic myocarditis, which leads to enlarged cardiomyopathy due to myocardial necrosis. While myocytes don't regenerate, the grow response triggered due to HCV infection that can lead to myocyte dysplasia & hypertrophic cardiomyopathy[10]. The direct role of HCV core proteins in the development of cardiomyopathy has been proposed[11]. Cardiac dysfunction is a rare complication of HCV-associated mixed cryoglobulinemic vasculitis, and while initial outcomes may appear positive, patients with cardiac injury tend to have lower survival rates compared to those without[12]. Chronic HCV infection is also linked to metabolic disorders such as insulin action, type-2 diabetes, hypertension, and symptom of heart failure[13]. Relationship between hyperlipidemia & atherosclerosis in person-to-person's hepatitis C is not straightforward. An Epidemiological study found that acute HCV infection was strongly associated with insulin action, cases exhibited only mild-mannered atherosclerosis, propose a well-defined form of biological process disruption linked to HCV. Chronic HCV-related to steatosis is believed to play a crucial role in coronary artery disease by influencing atherogenic factors such as inflammation and metabolic abnormalities. Interferon-based treatment for chronic HCV has been shown to reduce the long-term risk of stroke[14-16]. In individuals with infectious disease, atherosclerosis is probably driven more by an unhealthy process than by lipid abnormalities. Therefore, patients with

chronic hepatitis C who have normal cholesterol and triglyceride levels should avoid behaviors that could elevate their cardiac risk.

Liver Cirrhosis:

Individuals suffering liver cirrhosis (LC) often exhibit involuntary cardiac dysfunction, characterized by enhanced sympathetic nervous system activity and diminished vagal cardiac function, which significantly influences liver damage and overall health outcomes[17-19]. The baroreflex plays a critical role in maintaining electrical stability in the heart and can serve as a predictor of higher mortality and end-organ damage[11-13]. People with liver cirrhosis typically experience heightened nervous system activity and hyperdynamic spreading, reflected by increased cardiovascular output and decreased systemic vascular action. Changes can lead to heart muscle remodeling & left ventricular hypertrophy (LVH), contributing to both systolic and diastolic dysfunctions, as well as cardiomyopathy[3,20-22]. The characteristic criteria for cirrhotic cardiomyopathy are outlined in Table-I [23].

Table 1 A working group was formed at the 2005 World Congress of Gastroenterology to develop diagnostic and supportive criteria for cirrhotic cardiomyopathy.

Cirrhotic cardiomyopathy - Defined : As cardiac dysfunction in cases with cirrhosis, defined by reduced contractile response to stress, impaired diastolic relaxation, and electrophysiological abnormalities, in the absence of other known cardiac conditions.
Criteria for diagnosis Systolic dysfunction A reduced increase in cardiac output in response to pharmacological stimulation, volume loading, or physical activity. EF at rest is less than 55%.

Diastolic insufficiency
Age-corrected E/A ratio of less than 1.0
Prolonged deceleration time (> 200 ms)
Extended isovolumetric relaxation time (> 80 ms)
Supporting criteria:
Electrophysiological abnormalities
Abnormal chronotropic response
Electromechanical uncoupling/dyssynchrony
Prolonged QTc interval
Enlarged left atrium
Increased myocardial muscle mass
Elevated BNP and pro-BNP levels
Raised troponin levels

Table 1 Proposal for diagnostic and supplementary criteria for cirrhotic cardiomyopathy

BNP: Brain natriuretic peptide;

E/A: Early diastolic/atrial filling ratio;

EF: Left-ventricular ejection fraction.

The heart's inability to generate adequate arterial blood pressure and myocardial output is referred to as systolic dysfunction. Physical exercise that augments left ventricular pressure, volume, ejection fraction, and heart rate in certain cirrhotic patients can reveal this dysfunction. The administration of vasoconstrictors, such as terlipressin and

angiotensin II, elevates systemic vascular resistance (SVR) and consequently increases left ventricular. In contrast, the risk of worsening the vasodilatory state requires cautious use of vasodilators, including angiotensin-converting enzyme inhibitors and other medications that reduce afterload[23]. The progression and prognosis of renal impairment, as well as the onset of consequences such as sodium and water retention and ascites formation, may be influenced by systolic dysfunction [24-25]. Myocardial hypertrophy, fibrosis, and subendothelial edema induce increased cardiac wall rigidity, which leads to diastolic dysfunction in cirrhosis. Diastolic dysfunction is found in 45% to 56% of patients, with its presence being more pronounced in those undergoing severe decompensation. In these cases, cardiac hypertrophy, contractile pathology, Ventricular remodeling, and diastolic disfunction play a significant role in the development of cirrhotic cardiomyopathy[25-27]. Diastolic dysfunction can have a detrimental effect on the prognosis of patients with cirrhosis by promoting complications and impeding the efficacy of interventions that induce rapid preload increases, such as the implantation of a transjugular intrahepatic porto-systemic shunt (TIPS). People who have extensive cirrhosis frequently exhibit tachycardia[23]. The ability to sustain an adequate cardiac output to meet the demands of systemic circulation is further compromised by the inability to increase heart rate. At this point, effective blood volume experiences a sharp decline, resembling the conditions observed in post-paracentesis circulatory dysfunction and hepatorenal syndrome[28-30]. Prolongation of the electrocardiographic QT interval is commonly seen in cirrhosis, affecting about 60% of patients with advanced disease. In this situation, it is advisable to avoid or administer medications that affect QT intervals with caution and under strict ECG surveillance[31]. People with liver cirrhosis exhibit systemic and cardiac changes, as illustrated in Figure 1. Liver donation typically results in the resolution of nearly all cardiovascular maladies within a few months [18, 23, 32].

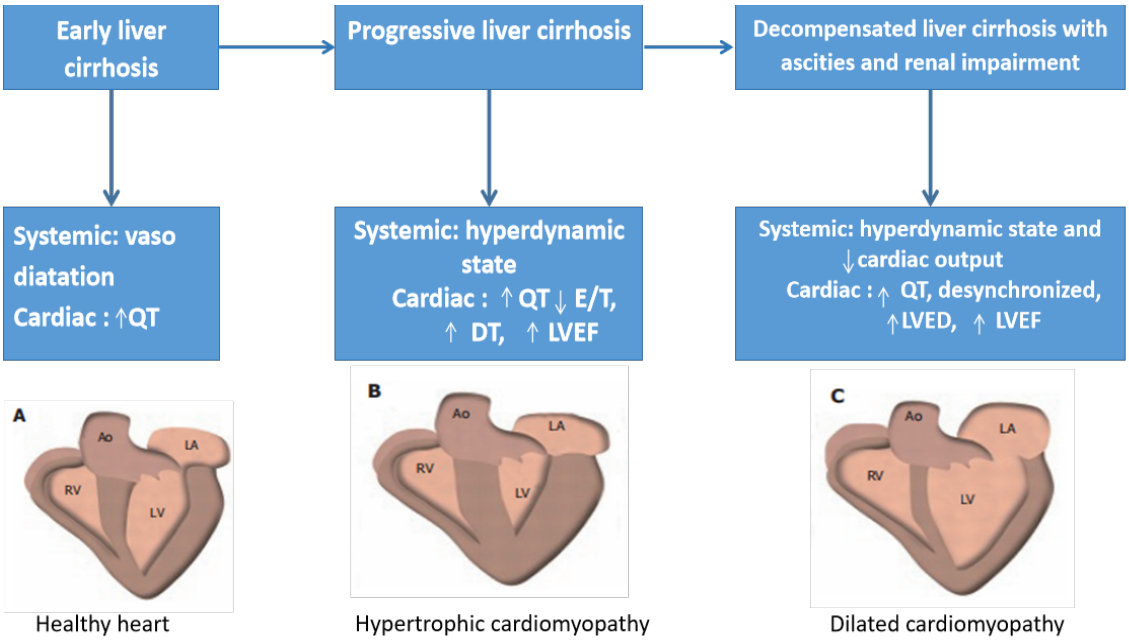

Figure 1 The systemic and cardiac alterations in people with liver cirrhosis

Nonalcoholic fatty liver disease

The psychological characteristics associated with cardiovascular health contribute to mortality in persons with nonalcoholic fatty liver disease (NAFLD). In individuals with diabetes, non-alcoholic fatty liver disease (NAFLD) is associated with cardiovascular illness (CVD) independently of conventional risk factors, glycemic regulation, healthcare provider influence, and metabolic syndrome characteristics. A comparative research of diabetic patients with and without NAFLD revealed that those with NAFLD exhibited a higher prevalence of myocardial thrombosis, hypertension, significant obesity, inadequate control of glucose levels, dyslipidemia, and elevated carotid intimal thickness. Furthermore, when non-alcoholic steatohepatitis (NASH) advances, the risk of cardiovascular disease is associated with the level of inflammation detected in liver biopsy specimens. Cardiovascular death is at a minimum double in patients with NASH. Hepatic steatosis is associated with diminished adiponectin levels and elevated amounts of fibrinogen, C-reactive proteins (CRP), and plasminogen stimulate activator preventing 1 (PAI-1), all of which are indicators of inflammation and independent warning signs for coronary artery disease, irrespective of BMI and intra-

abdominal obesity. Individuals with NAFLD exhibit markedly elevated mean intima-media thickness and a higher prevalence of plaques, hence augmenting the risk of atherosclerosis among those with their metabolic syndrome. NASH has been demonstrated to independently forecast plasma inflammatory biomarkers, regardless of visceral adiposity and other confounding variables. These data indicate that NASH functions not only as an indicator for CVD but could also have a role in its progression. Steatosis is recognized as the foremost standalone risk factor for vascular damage, succeeded by age & blood pressure[33-36]. Patients with NAFLD and diastolic blood pressure ≥ 130 mmHg are 4.7 times more inclined to yield a positive treadmill test. A recent study revealed that asymptomatic obese children with NAFLD exhibited early indications of both left ventricle diastolic and cardiac dysfunction, with these defects being more pronounced in those with NASH[37].

Primary biliary cirrhosis

Cholesterol levels are heightened in the majority of persons having primary biliary cirrhosis (PBC). Hypercholesterolemia in these people should be regarded as a cardiovascular risk factor alone when it is present alongside additional risk factors. Ursodeoxycholic acid, the conventional therapy for primary biliary cholangitis, mitigates cholestasis and thereby reduces circulating cholesterol levels. Consequently, hypercholesterolemia without supplementary cardiovascular risk variables does not necessitate specific treatment in people with PBC. Epidemiological studies indicate markedly elevated all-cause mortality rates in patients with PBC, with a considerable fraction of this higher death rate attributable to non-liver-related factors[38-39]. Although these studies did not examine the causes of the increase in non-liver-related mortality, further data from the same populations indicate that malignancies contribute little or not at all to the heightened non-liver mortality. Considering the importance of cardiovascular mortality in the general populace, it is essential to examine the potential influence of cardiac variables on the increased non-liver-related mortality rates seen in PBC patients. Autonomic dysfunction has been recognized in primary biliary

cholangitis (PBC) and is linked to a heightened risk of cardiac death in several chronic non-hepatic disorders[40-41]. Notable bioenergetic anomalies in peripheral muscles have been observed in PBC, indicating that same difficulties may arise in cardiac muscle. Autonomic dysfunction can impair tissue perfusion patterns, resulting in diminished muscle perfusion and exacerbating peripheral tiredness. A prevalent inclination towards modified myocardial function has been noted in PBC, frequently in the absence of the normal symptoms associated with cardiac failure[42].

Primary sclerosing cholangitis

Primary sclerosing cholangitis (PSC) is an ongoing inflammatory disorder that impacts the principal bile ducts, marked by periductal fibrosis and the formation of strictures. Arteriosclerosis entails the accumulation of modified fats and lipoproteins in major arteries, causing inflammation as well as fibrosis, which culminates in arterial constriction and diminished blood flow to tissues and organs reliant on them. Understanding the causal factors is essential for comprehending illness mechanisms and formulating targeted treatments. Due to the pathogenic similarities and shared molecular, cellular, and morphological characteristics that elucidate their pathogenesis, it is hypothesized that PSC constitutes "arteriosclerosis of the bile duct" triggered by toxic biliary lipids[44]. This idea ought to encourage translational research to advance the exploration of innovative treatment methods for both the diseases.

Hepatocellular carcinoma

Cardiovascular problems associated with hepatocellular carcinoma (HCC) are rare. Instances of right heart invasion by hepatocellular carcinoma (HCC) have been documented, resulting in sporadic right ventricular outflow blockage and Budd-Chiari

syndrome.Patients with hepatocellular carcinoma who develop cardiac metastases are generally discovered at advanced stages, resulting in low survival rates for these individuals. Primary causes of mortality are linked to hepatocellular carcinoma (HCC) or the underlying hepatic disease. A restricted cohort of patients will yield to cardiac metastases[48]. Palliative interventions for tumor thrombi may include transcatheter treatment such as chemotherapy, transarterial chemoembolization, and radiation therapy, each of which can offer partial symptom alleviation for patients[49].

Budd-chiari syndrome

Primary Budd-Chiari syndrome (BCS) is an uncommon disorder characterized by the blockage of hepatic venous outflow at multiple locations, from little veins in the liver to the inferior venous cava. BCS is categorized into three main types: Type I, which involves obstruction of the inferior vena cava (IVC); Type II, characterized by hepatic vein stenosis; and Type III, which consider blockage of both the IVC and hepatic veins. The incidence of hepatocellular carcinoma (HCC) associated with BCS differs across these types. Type I BCS has a higher risk of developing HCC, with rates ranging from 10.7% to 43.5% [49-50]. The exact mechanisms behind the development of HCC in this context remain unclear. Treatment options for BCS-related HCC include transarterial chemoembolization (TACE), surgical interventions, and, more newly, surgical procedure followed by transdermic microwave ablation[51-54].

Portal hypertension

Portal hypertension can lead to three significant complications:

Hepatopulmonary Syndrome (HPS): This condition is defined by a deficiency in activity due to respiratory organ vascular expansion in the patients with severe liver disease. Studies have given the angiogenesis is stimulated by elevated levels of nitric oxide and vascular epithelial tissue growth factor in individuals with severe liver disease[55-58]. In the early stages, individuals with HPS may experience gradual onset of dyspnea or remain asymptomatic. Around 25% of patients with HPS develop platypnea (dyspnea upon standing) and orthodeoxia (worsened hypoxemia in an upright position). As the syndrome progresses, patients may exhibit extremity symptom and cyanosis[59]. Some studies have shown improvements in PaO2 levels with garlic supplementation, based on two uncontrolled trials and a brief randomized study[60-63].

Portopulmonary Hypertension (POPH):

This illness represents a form of pulmonary arterial hypertension (PAH) that manifests alongside portal hypertension, irrespective of the degree of hepatic disease. The advancement of POPH does not inherently correspond with the degree of liver impairment or the intensity of portal hypertension. The underlying mechanisms linking pulmonary arterial hypertension to pulmonary obstructive sleep apnea remain poorly understood. Historically, POPH shares similarities with idiopathic PAH, This condition constitutes a variant of pulmonary arterial hypertension (PAH) that occurs together with portal hypertension, regardless of the severity of hepatic disease. The progression of POPH does not necessarily correlate with the severity of liver dysfunction or the magnitude of portal hypertension[64-67].

Treatment modalities encompass prostacyclin equivalents (prostanoids), endothelial receptors antagonists, and this enzyme-5 inhibitors. A limited study comprising participants with moderate to severe pulmonary arterial hypertension (PAH) established a connection to pulmonary obstructive sleep apnea indicated that β-blockers were associated with a decline in exercise capacity[68-69].

Hepatic hydrothorax:

This syndrome is defined by the presence of transudative pleural effusion without any underlying cardiac or pulmonary conditions. Retrospective observational data estimate its prevalence among cirrhotic patients to be between 5% and 10%. The principal mechanism facilitating the transfer of ascitic fluid from the cavity of the peritoneum to the pleural cavity is attributed to anatomical anomalies in the diaphragm. This phenomenon was validated by imaging experiments showing the migration of 99mTc-human protein from the abdominal region to the lung cavity, even among patients lacking visible ascites[70-71].

Typical symptoms encompass cough, dyspnea, chest pain, hypoxia, and, in severe instances, breathing problems, with or without ascites. Spontaneous bacterial pleuritis (SBPL) may arise when hepato hydrothorax (HH) becomes infected, even in the absence of pneumonia. Symptoms of SBPL encompass fever, pleuritic chest pain, and mild deterioration in renal function or encephalopathy, warranting careful monitoring. A pleural effusion with a polymorphonuclear (PMN) cell count exceeding 500 cells/mm³ confirms the diagnosis of SBPL, Cases with PMN counts ranging from 250 to 500 cells/mm³ are corroborated by a positive fluid from the pleura culture[72].

Chest tube insertion is generally contraindicated in SBPL unless empyema is present, as it can lead to complications such as protein loss, prolonged drainage, secondary infections, and hepatorenal syndrome. The management of HH involves sodium restriction and diuretic therapy, which is often effective, though fluid removal from the pleural cavity tends to occur more slowly than from the peritoneal cavity. Approximately 20% of patients experience refractory HH. In selected cases, percutaneous drainage or chest tube insertion may be considered. For resistant cases, the placement of a transjugular intrahepatic portosystemic shunt (TIPS) is the preferred intervention, demonstrating favorable response rates[71-73].

Liver transplantation

Patients with cirrhosis necessitating liver transplantation generally demonstrate increased cardiac output. Decreased cardiovascular system resistance & bradycardia are prevalent in hepatitis and might be intensified by the administration of beta-blockers. The modifications to the body increase the likelihood of cardiovascular problems due to the circulatory issues faced by recipients of liver transplants in the initial postoperative phase. Cardiac-related mortality post-transplantation may occur due to factors such as arrhythmias, acute heart failure, or myocardial infarction[74].

The increased perioperative mortality in transplant patients with coronary artery disease (CAD) necessitates a comprehensive assessment of individuals at high risk for atherosclerotic heart disease. Since no single diagnostic test offers complete predictive accuracy, evaluations must consider the varying prevalence of cardiovascular conditions among different transplant candidates and the limitations of each diagnostic method. Unlike ischemic heart disease, patients with advanced liver disease frequently present with cardiac abnormalities that contribute to systolic and diastolic dysfunction, which may remain undetectable at rest. Additionally, underlying electrophysiological disturbances can lead to a disconnect between electrical and mechanical cardiac activity[75].

Diagnosing cirrhotic cardiomyopathy remains challenging due to its complex presentation. Patients with hepatitis requiring liver transplantation typically exhibit elevated cardiac output during exercise. Reduced circulatory system resistance and bradycardia are common in hepatitis and may be exacerbated by the use of beta-blockers. The alterations to the body heighten the risk of cardiovascular complications owing to the circulatory challenges encountered by liver transplant recipients during the early postoperative period. Additionally, prolonged QT intervals may resolve in

approximately 50% of cases post-orthotopic liver transplantation, indicating that liver dysfunction may not be the sole underlying cause[18,23,32].

Hematologic Contributions to Hepatic Conditions

Heart failure: Cardiac-associated hepatic dysfunction may occur due to conditions including constricting pericarditis, major pulmonary arterial hypertension (PAH), a narrowing of the mitral valve, tricuspid regurgitation (TR), corpulmonale, ischemic cardiomyopathy, and difficulties subsequent to the Fontan processes for pulmonary atresia and hypoplastic left heart syndrome. These disorders frequently lead to passive hepatic congestion resulting from increased right ventricular (RV) pressure and right-sided heart failure. Advances in medical treatments have significantly improved heart failure outcomes, leading to a decline in the prevalence of cardiac cirrhosis[23,75].

Pathophysiology:

In chronic heart failure (backward failure), elevated venous pressure due to right ventricular dysfunction results in hepatocyte atrophy and perisinusoidal edema, potentially impairing oxygen and nutrient diffusion to liver cells. This retrograde congestion also increases hepatic lymph output, leading to ascites when lymph production surpasses the drainage capacity of the lymphatic system. Moreover, rising pressure in the hepato sinusoids intensifies bile duct damage by impairing endothelial cells and breaking the intercellular tight connections that delineate the extravascular cavity from the bile canaliculus. Chronic diminished blood flow leads to thrombosis in sinusoids, hepatic venules, and portal tracts, thereby facilitating liver fibrosis[23,75]. Furthermore, increased pressure in the hepatic sinusoids exacerbates bile duct injury by compromising endothelial cells and disrupting the intercellular tight junctions that separate the extravascular space from the hepatic canaliculus. Chronic reduced blood flow results in thrombosis within sinusoids, hepatic venules, and portal tracts, thereby promoting liver fibrosis. Microscopic findings of hepatic venous hypertension include dilated central veins, hemorrhaging within central veins, and sinusoidal engorgement.

If chronic congestion remains untreated, it may progress to liver fibrosis and ultimately result in cardiac cirrhosis[76].

Acute heart failure (forward failure) is commonly triggered by severe systemic hypotension following cardiopulmonary collapse due to myocardial infarction, worsening heart failure, or pulmonary embolism. Ischemic hepatitis has been observed in cases of extreme hypoxemia, such as obstructive sleep apnea and respiratory failure, as well as conditions of high metabolic demand, including toxic or septic shock, even in the absence of significant hypotension[78-79].

When hepatic blood flow decreases, oxygen consumption can increase significantly. The liver mitigates hypoxic damage by maximizing oxygen extraction by hepatocytes, reaching levels as high as 95% as blood circulates through the organ. However, prolonged tissue hypoxia or severe shock can overwhelm this protective mechanism, leading to hypoxic liver injury[80].

2. OBJECTIVES

1. To assess the number of cases and level of autonomic dysfunction in individuals with liver cirrhosis.
2. To correlate autonomic dysfunction with Electrocardiography and Echocardiography in patients with cirrhosis of liver.
3. To analyze the correlation between autonomic dysfunction and findings from electrocardiography (ECG), such as heart rate variability, QT interval prolongation, or arrhythmias.
4. To evaluate the therapeutic implications of detecting autonomic dysfunction in cirrhosis patients, such as optimizing cardiovascular and hepatic management.

3. REVIEW OF LITERATURE

Yasser Mahrous Fouad, Reem Yehia, and colleagues highlighted the crucial interplay between the liver & heart, emphasizing its significance for both hepatologists & cardiologists. Accurate differential diagnosis of liver injury is vital in cardiology practice, necessitating close collaboration between cardiologists and hepatologists, as multiple conditions can mimic hemodynamic liver damage. Requiring treatment strategies that prioritize the underlying cardiac condition. Patients with advanced liver disease may develop cirrhotic cardiomyopathy, characterized by hemodynamic disturbances, systolic and diastolic dysfunction, diminished cardiac output, and electrophysiological condition. Notably, liver transplantation can potentially reverse cirrhotic cardiomyopathy and improve cardiac function. Additionally, systemic diseases—including congenital, metabolic, inflammatory disorders, and alcohol-related conditions—can concurrently affect both the liver and heart [1].

Møller and Henriksen et al. conducted a study on patients with cirrhosis, highlighting a hyperdynamic systemic circulation characterised aside elevated cardiac signal and heart rate, along with significantly reduced systemic vascular action. The associated cardiac dysfunction, now recognized as "cirrhotic cardiomyopathy," is distinct from alcoholic heart muscle disease. Clinically, these patients often experience fluid retention, with underlying heart failure becoming apparent under stress. While no definitive treatment is currently available, caution is advised when performing procedures that may place additional strain on the heart, such as shunt implantation or liver transplantation[4].

Søren Møller, Lise Hobolth, and colleagues conducted a study involving 410 patients diagnosed with cirrhosis, all of whom underwent comprehensive hemodynamic assessments. The collected data were analyzed using regression analysis, principal

component analysis, and Cox proportional hazards analysis. Patients with advanced cirrhosis frequently exhibited hyperdynamic circulation accompanied by central hypovolemia. However, the triggers of systemic hemodynamic disturbances and their relationship with splanchnic hemodynamics remain unclear[5].

Trevisani F, Sica G, Mainqua P, Santese G, and colleagues conducted a prospective observational study involving 103 cirrhotic outpatients. Using 24-hour Holter monitoring, they analyzed heart rate variability (HRV) and found that reduced HRV is a frequent occurrence in liver cirrhosis. Their findings suggest a strong association between diminished HRV, cardiac dysfunction, liver disease severity, and overall mortality[6].

Rajendra Acharya U, Et al. Engaged in discussion regarding Heart rate variability (HRV) serves as a dependable measure of physiological elements influencing cardiac rhythm and can elucidate the interaction between the sympathetic and parasympathetic nervous systems. It may include signs of existing or forthcoming disorders. Heart rate variability analysis is a widely utilized noninvasive method for evaluating autonomic nervous system functions. Computer-based analytical tools are beneficial for diagnosing HRV signal characteristics, emphasizing the diverse uses of HRV and the numerous analytical approaches employed[8].

Lunzer M, Newman S, Bernard A, et al. conducted a study primarily focused on impaired cardiovascular response in liver illness. Patients with cirrhosis have diminished cardiovascular reactivity to both reflexive and exogenous noradrenaline, which may result in circulatory failure following hemorrhage or surgical procedures, necessitating caution when prescribing medications that influence autonomic function[9].

Milan A, Caserta MA, Et. al . Analyze the relationship of baroreflex sensitivity, left ventricular morphology, and heartbeat function in individuals with essential hypertension. Their study highlights a significant correlation between baroreflex regulation and structural as well as functional changes in the heart, emphasizing its potential role in cardiovascular health[10].

Lantelme P, Khettab F, Et. al, their study aimed to evaluate spontaneous baroreflex sensitivity (BRS) as a potential cardiovascular risk marker in hypertension. The researchers investigated whether impaired BRS could predict cardiovascular complications and serve as a reliable index for assessing autonomic dysfunction in hypertensive patients. Consider evaluating BRS in cirrhotic patients to assess its correlation with ECG/ECHO findings. Compare BRS impairment in cirrhosis with hypertension studies to understand if similar cardiovascular risks exist. Explore whether autonomic dysfunction (measured via BRS) can serve as a clinical marker for cardiac complications in cirrhosis[11].

Okada N, Takahashi N, Et al . explored the prognostic measure of baroreflex sensitivity (BRS) for cardiovascular events individuals with type 2 diabetes mellitus (T2DM) who do not have structural heart disease. Recognizing the high prevalence of autonomic dysfunction in diabetes[12].

Yufu K., Takahashi N., and colleagues, their findings suggest that baroreflex sensitivity serves as a crucial indicator of cardiovascular and cerebrovascular risks, with distinct patterns based on gender. Men with reduced baroreflex sensitivity are more prone to cardiac events, while women have a higher likelihood of experiencing cerebrovascular complications. These results highlight the importance of personalized assessments of autonomic function based on gender[13].

Miyajima I and their team conducted a population-based study in an HCV-endemic region, revealing a strong link between chronic HCV infection and increased insulin resistance. While individuals with HCV exhibited mild atherosclerosis, the combined effects of HCV, insulin resistance, and inflammation could contribute to an increased long-term risk of cardiovascular disease[14].

Valeriano V., Et.al, the study confirms that cirrhotic cardiomyopathy (CCM) occurs regardless of ascites but is further exacerbated when ascites is present. Diastolic dysfunction and autonomic impairment are key features of CCM, highlighting the need for early cardiovascular assessment in cirrhotic patients, even before the development of ascites[17].

Hendrickse MT, and their team, study's objectives were to ascertain the prevalence of autonomic dysfunction in cases of chronic liver disease (CLD), how autonomic nephropathy changes over time relates to progression of liver disease, and the possibility effects of autonomic dysfunction on morbidity and mortality in cirrhotic patients[19].

Braverman AC, Steiner MA Et.al . In this study aimed to examine cases of high-output congestive heart failure (CHF) occurring after transjugular intrahepatic portosystemic shunt (TIPS) placement. The researchers sought to, Identify the mechanism behind post-TIPS heart failure. Determine the risk factors for cardiovascular complications following TIPS. Understand the hemodynamic changes induced by TIPS in cirrhotic patients[21].

Wong F, Villamil A, Et. al.., The study highlights that diastolic dysfunction is highly prevalent in cirrhosis and plays a significant role in disease progression and patient outcomes. As liver disease worsens, diastolic dysfunction becomes more pronounced, increasing the risk of circulatory collapse, ascites, and organ failure. This study

underscores the need for routine echocardiographic evaluation in cirrhotic patients to detect early cardiac dysfunction and optimize management strategies[27].

Torregrosa M, Et... al. The study provides strong evidence that cirrhotic cardiomyopathy (CCM) is a frequent complication of cirrhosis but is at least partially reversible following liver transplantation. Diastolic dysfunction and QT prolongation improve post-transplant, indicating that hepatic dysfunction plays a major role in cardiac alterations. However, some cardiac abnormalities may persist, especially in patients with advanced cirrhosis and long-standing cardiovascular changes[32].

An Historical View of the Distribution of Blood Pressure Under Various Conditions

One of the main areas of inquiry in studies of cardiovascular and autonomic function has been the examination of changes in blood pressure (BP) under various physiological circumstances. Over the years, researchers have explored how BP changes in different postures and under stress, such as during the handgrip test or Valsalva maneuver, to understand autonomic regulation and cardiovascular health.

Evolution of Blood Pressure Measurement: A Historical Perspective

The measurement of blood pressure (BP) has evolved over centuries, from crude invasive techniques to modern non-invasive digital monitoring. This evolution has significantly improved diagnosis, treatment, and management of cardiovascular diseases. Below is a detailed discussion on the history, advancements, and future of BP measurement.

Early Concepts & First Attempts (Ancient to 18th Century)

Ancient Theories on Blood Circulation: Ancient civilizations, such as the Egyptians and Greeks, had basic ideas about the pulsations of arteries, but they did not understand blood circulation. Galen (2nd Century AD) proposed that blood was produced in the liver and consumed by the organs, which was later proven wrong.

Discovery of Blood Circulation: William Harvey (1628)

English physician William Harvey was the first to describe the systemic circulation of blood in his famous work "De Motu Cordis" (On the Motion of the Heart and Blood). However, he had no tools to measure blood pressure directly.

First Direct Measurement of Blood Pressure (18th Century)

Stephen Hales (1733): The First BP Measurement in Animals. British scientist Stephen Hales performed the first recorded blood pressure measurement in a horse. He inserted a long glass tube into the carotid artery and observed how high the blood rose in the tube. Although this method was invasive, it established the principle that blood exerts pressure on arterial walls.

The Emergence of Non-Invasive BP Measurement (19th Century)

Poiseuille's Manometer (1828) Jean Léonard Marie Poiseuille, a French physician, improved upon Hales' work by using a U-shaped mercury manometer to measure BP more accurately in animals. This method laid the groundwork for future mercury-based BP devices.

First Human Blood Pressure Measurement (1896): Riva-Rocci's Sphygmomanomete. Scipione Riva-Rocci, an Italian physician, developed the first non-invasive BP measuring device. It consisted of An inflatable rubber cuff placed around the upper arm. A mercury column to measure pressure. Manual inflation with a rubber bulb. The Riva-Rocci method allowed doctors to measure systolic BP by observing the point at

which the radial pulse disappeared while inflating the cuff. Limitation: It only measured systolic BP, not diastolic BP.

The Development of Modern BP Measurement (20th Century)

Korotkoff Sounds & Auscultatory Method (1905), Dr. Nikolai Korotkoff, a Russian surgeon, discovered that using a stethoscope while deflating a BP cuff could detect sounds (Korotkoff sounds) corresponding to systolic and diastolic BP. This led to the modern auscultatory method, where the cuff is inflated above systolic BP. As pressure is released, the first sound marks systolic BP. The disappearance of sounds marks diastolic BP. Mercury Sphygmomanometer Becomes the Gold Standard (1920s-1950s) The mercury sphygmomanometer became the gold standard for BP measurement. Advantages are Highly accurate & reliable. Used for decades in clinical practice. Disadvantages of this method is Bulky & not portable, Mercury toxicity concerns.

Aneroid & Automated Devices (1950s-1970s): Aneroid sphygmomanometers (without mercury) were developed as a safer alternative. The first semi-automated BP monitors using electronic sensors appeared.

Digital Revolution & Ambulatory BP Monitoring (Late 20th - 21st Century)

Oscillometric BP Measurement (1970s-Present): The oscillometric method was developed, allowing electronic BP monitors to measure BP automatically. Instead of using Korotkoff sounds, these devices detect pressure oscillations in the artery and calculate BP. Advantages: No need for a stethoscope (ideal for home use). User-friendly & portable. More consistent in noisy environments.

Since the 1980s, ambulatory blood pressure monitoring (ABPM) has provided a more accurate assessment of blood pressure fluctuations by allowing continuous measurement over a 24-hour period. In recent years, advancements in smart and wearable blood pressure monitors, including fitness bands and smartwatches, have

enabled non-invasive blood pressure tracking using optical sensors and artificial intelligence.

Physiological Mechanisms: How BP Changes from Supine to Standing

When a person stands up from a supine (lying) position, gravity causes blood to pool in the lower extremities, reducing venous return to the heart. The body responds by activating compensatory mechanisms to maintain BP and prevent fainting.

Step-by-Step Changes in BP Regulation:

Supine (Lying Down) Position:

- Blood is evenly distributed.
- Cardiac output is stable.
- BP is generally higher compared to the standing position.

Transition to Standing (Immediate Response):

- Blood pools in the legs → Venous return decreases.
- Stroke volume drops by ~20%, reducing cardiac output.
- BP initially drops slightly.

Baroreceptor Reflex Activation (Within Seconds):

- Baroreceptors (in carotid arteries & aortic arch) sense the BP drop.
- Sympathetic nervous system (SNS) is activated to restore BP:
  - Heart rate (HR) increases (reflex tachycardia).
  - Vasoconstriction occurs to prevent excessive BP drop.
- BP stabilizes within 30 seconds to 1 minute.

Standing Position (After Adaptation):

- BP should return to normal or near-normal levels.
- A healthy individual maintains adequate cerebral perfusion without dizziness or fainting.

Normal BP Changes in Supine vs. Standing Position

BP Parameter	Supine (Lying Down)	Standing	Expected Change
Systolic BP (SBP)	120-130 mmHg	110-120 mmHg	↓ 5-10 mmHg
Diastolic BP (DBP)	70-80 mmHg	70-85 mmHg	Same or ↑
Heart Rate (HR)	60-75 bpm	75-95 bpm	↑ by 10-15 bpm

Systolic BP tends to decrease slightly upon standing. Diastolic BP may remain stable or increase due to sympathetic vasoconstriction. Heart rate increases to compensate for reduced cardiac output.

Abnormal BP Responses & Clinical Conditions

Orthostatic Hypotension (OH)

Definition: A persistent decrease in the blood pressure while a minimum 20 mmHg in systolic or 10 mmHg in diastolic within three minutes of standing signifies orthostatic hypotension, a disorder marked by compromised blood pressure management during postural transitions.

Causes of Orthostatic Hypotension:

Autonomic Dysfunction (e.g., cirrhosis, diabetes, Parkinson's)

Hypovolemia (dehydration, blood loss)

Medications (antihypertensives, diuretics, antidepressants)

Aging (baroreceptor sensitivity decline)

Symptoms:

Dizziness or lightheadedness

Blurred vision

Syncope (fainting)

Fatigue

Postural Tachycardia Syndrome (POTS)

Definition: Significant heart rate elevation of ≥30 bpm (≥40 bpm in teenagers) within 10 minutes of standing, without a notable decrease in blood pressure.

Causes:

Dysautonomia (autonomic nervous system dysfunction)

Chronic fatigue syndrome

Deconditioning (lack of exercise)

Hyperadrenergic states

Symptoms:

Rapid heartbeat

Dizziness, palpitations

Exercise intolerance

Brain fog

Supine Hypertension

Definition: BP remains abnormally high when lying down, but may drop in a standing position.

Common in:

Autonomic failure (e.g., multiple system atrophy)

Spinal cord injuries

Chronic kidney disease

Clinical Significance:

Can cause nocturnal hypertension, increasing the risk of heart disease, kidney failure, and stroke.

Supine vs. Standing BP in Cirrhosis & Autonomic Dysfunction

Patients with cirrhosis often exhibit autonomic nervous system impairment, leading to abnormal BP regulation.

Cirrhotic Cardiomyopathy: Patients have a blunted baroreceptor reflex, leading to orthostatic hypotension.

Portal Hypertension: Can cause low systemic vascular resistance, reducing BP control.

Ascites & Volume Shifts: Large fluid accumulation can affect BP response to postural changes.

Clinical Implication: BP monitoring in cirrhosis patients should include supine and standing measurements to detect early autonomic dysfunction.

Condition	Treatment Approach
Orthostatic Hypotension	Increase salt & fluid intake, compression stockings, fludrocortisone, midodrine
POTS	Increase hydration, exercise, beta-blockers, ivabradine
Supine Hypertension	Avoid excessive salt intake, adjust medications

Valsalva Maneuver: Physiology, Clinical Significance, and Interpretation

The Valsalva Maneuver is a forced expiration against a closed glottis, used to assess autonomic nervous system function, baroreceptor reflexes, cardiac function, and venous return. It has diagnostic significance in detecting autonomic dysfunction, heart failure, and arrhythmias.

Phases of the Valsalva Maneuver

The Valsalva maneuver consists of **four distinct phases**, each with unique hemodynamic changes:

Phase	Description	Physiological Response
Phase I (Onset of Straining)	Increased intrathoracic pressure (forced expiration against a closed glottis).	**BP initially rises** due to compression of the aorta.
Phase II (Sustained Straining)	Reduced venous return to the heart due to increased thoracic pressure.	**BP drops**, **HR increases (reflex tachycardia)** to compensate.
Phase III (Release of Pressure)	Sudden release of intrathoracic pressure, allowing venous return.	**BP drops briefly**, **HR spikes** momentarily.
Phase IV (Overshoot Recovery)	Increased venous return leads to an enhanced cardiac output.	**BP overshoots**, **HR slows down (reflex bradycardia)**.

Clinical Significance of the Valsalva Maneuver

A. Assessment of Autonomic Function

The response of BP and HR during the Valsalva maneuver helps assess the **sympathetic and parasympathetic nervous system**.

Normal Response:

BP drops in **Phase II**, then overshoots in **Phase IV**.

HR increases in **Phase II** and decreases in **Phase IV**.

Autonomic Dysfunction (e.g., Diabetic Neuropathy, Cirrhosis, Parkinson's):

Blunted BP response (Phase IV overshoot absent).

No HR variability (Fixed HR throughout).

Seen in conditions affecting **baroreceptor sensitivity**.

B. Cardiac Function Testing

Heart Failure:

Phase IV overshoot is reduced due to poor cardiac output.

Aortic Stenosis & Hypertrophic Cardiomyopathy:

The maneuver **reduces venous return**, worsening murmurs in these conditions.

C. Identifying Arrhythmias

Can terminate **supraventricular tachycardia (SVT)** by increasing **vagal tone** and slowing AV nodal conduction.

4. MATERIALS AND METHODS

4.1 SOURCE OF DATA

Data is collected from patients meeting the inclusion criteria, specifically those with a history of organophosphate exposure or ingestion, over an 18-month period from March 2023 to December 2024. The study is conducted at Shri B M Patil Medical College and Research Center, Vijayapura, with patient observations taking place in the outpatient clinic.

4.2 METHOD OF COLLECTION OF DATA

Inclusion Criteria:

- Patients diagnosed with cirrhosis based on clinical, biochemical, and imaging criteria.
- Age: 18 years and above.
- Both male and female patients.

Exclusion Criteria:

- Patients with pre-existing cardiovascular diseases, diabetes mellitus, or neurological disorders affecting autonomic function.
- Patients on medications known to influence autonomic function.
- Patients with active infections or sepsis.

Data Collection

Data will be collected using a structured protocol that includes clinical assessments, autonomic function tests, ECG, and echocardiography.

The following methods will be employed:

- **Clinical Assessment:**
  - Detailed history and physical examination will be conducted by trained clinicians.
  - The Child-Pugh & MELD scores will be evaluated to find the severity of liver disease in the patients.

- **Autonomic Function Tests:**
  - Heart rate variability (HRV) analysis using a standardized ECG recording.
  - Blood pressure response to standing (orthostatic hypotension test) measured using an automated sphygmomanometer.
  - Deep breathing test performed with continuous ECG monitoring.
  - Valsalva maneuver test conducted under supervised conditions.
- **Electrocardiography (ECG):**
  - Standard 12-lead ECG performed by trained technicians.

- QT interval, QTc prolongation, and HRV parameters.

- **Echocardiography:**
  - Performed by experienced cardiologists using standardized protocols.
  - Assessment of left ventricular function, diastolic dysfunction parameters, left atrial size, and ejection fraction.

Methodology

1. **Patient Selection:** Identify eligible cirrhosis patients based on inclusion and exclusion criteria.
2. **Clinical Assessment:** Document history, physical examination, and calculate Child-Pugh/MELD scores.
3. **Autonomic Function Testing:** Conduct HRV analysis, orthostatic hypotension test, deep breathing test, and Valsalva maneuver.
4. **ECG Evaluation:** Perform a 12-lead ECG and analyze QT interval, QTc prolongation, and HRV.
5. **Echocardiographic Assessment:** Measure left ventricular function, diastolic dysfunction, left atrial size, and ejection fraction.
6. **Data Analysis:** Use statistical methods to correlate autonomic dysfunction with ECG and echocardiographic findings.
7. **Interpretation and Conclusion:** Determine the relationship between autonomic dysfunction and cardiac parameters in cirrhosis patients.

Statistical - Analysis

- SPSS software is used for analyze the data.
- Continuous variables will be presented as mean ± standard deviation (SD), while categorical variables will be shown as frequencies and percentages.
- Pearson or Spearman correlation analysis will be used to assess the relationships between measures of autonomic dysfunction, ECG findings, and echocardiographic data.
- A $p<0.05$ will be proven statistically significant.

Ethical Considerations:

- The Institutional Ethics Committee need to approve.
- Written informed will be consistently obtained from all participants.

Type of Study: Hospital-based prospective cross-sectional study

To achieve a 10% absolute precision and 95% confidence, the study requires a sample size of 100 people.

Sample size calculation

The proportion of poisoning severity ratings classified as grade 3 is 24.1%, which requires a sample size of 100 for this study, as determined using the G*Power version 3.1.9.4 software for sample size calculation. To detect a difference in proportions (Exact - Proportion: Difference from constant) at a 5% significance level, a statistical power of 96% is necessary, as determined by a binomial test in a one-sample scenario. Formula used

$$n = \frac{Z^2\, p * q}{d^2}$$

Where Z represents the Z statistic at the α level of significance

d^2= Absolute error

P= Proportion rate

q= 100-p

Statistical analysis

The data will be documented in a Microsoft Excel spreadsheet, with statistical analysis performed using SPSS. The results will be presented in the form of graphs, counts, percentages, means, and standard deviations. A p-value of less than 0.05 will indicate statistical significance, and all statistical tests will be conducted using a two-tailed approach.

Data Analysis: Statistical tools (e.g., SPSS, Stata, R) will be used to analyze the data.

- Descriptive statistics will be used to summarize the exposure and demographic data.
- Logistic regression analysis will be employed to identify factors related to complications and mortality.
- The Kaplan-Meier survival analysis will be utilized to assess survival probability.

The study of autonomic dysfunction in cirrhosis and its correlation with ECG and echocardiographic findings involves a combination of clinical, physiological, and imaging investigations. These investigations help in evaluating autonomic nervous system (ANS) function, cardiac electrophysiology, and hemodynamic changes in cirrhotic patients.

1. **Autonomic Function Tests (ANS Evaluation)**

These tests assess baroreflex sensitivity (BRS), heart rate variability (HRV), and autonomic responses in cirrhotic patients.

Heart Rate Variability (HRV) Analysis:

Evaluates sympathetic and parasympathetic balance using short-term ECG recordings. Decreased HRV indicates autonomic dysfunction.

Baroreflex Sensitivity (BRS) Test:

Assesses how the heart rate responds to blood pressure fluctuations. Impaired BRS is a marker of autonomic failure and cardiovascular risk.

Valsalva Maneuver:

Measures heart rate and blood pressure changes during forced expiration against resistance. Abnormal responses indicate autonomic dysfunction.

Orthostatic Blood Pressure Testing:

Evaluates postural hypotension, a common feature of autonomic neuropathy. A significant drop in blood pressure upon standing suggests autonomic failure.

2. Electrocardiographic (ECG) Investigations

ECG is essential for assessing cardiac autonomic regulation and electrophysiological changes in cirrhotic patients.

QT Interval Prolongation Analysis:

A prolonged QT interval is common in cirrhosis and may indicate autonomic dysfunction and increased risk of arrhythmias.

Heart Rate Response to Deep Breathing (HRDB):

A non-invasive test that examines parasympathetic function by analyzing heart rate changes during deep breathing.

ECG Monitoring for Arrhythmias:

Detects bradycardia, sinus tachycardia, and low voltage QRS complexes, which are frequently seen in cirrhotic cardiomyopathy.

3. Echocardiographic (ECHO) Investigations

Echocardiography is used to assess structural and functional cardiac abnormalities in cirrhotic patients.

Diastolic Dysfunction Evaluation:

Assessing the E/A ratio, E/e' ratio, and left atrial volume index is crucial for diagnosing diastolic dysfunction, a hallmark of cirrhotic cardiomyopathy. These measurements offer valuable information about the heart's ability to relax and fill during diastole, helping to reveal the cardiac abnormalities linked to cirrhosis.

Left Ventricular Ejection Fraction (LVEF):

Evaluates systolic function to check for high-output heart failure, often seen in cirrhosis.

Pulmonary Hypertension Screening:

Uses Doppler echocardiography to check for elevated pulmonary artery pressure (PAH), which is common in cirrhosis.

4. Laboratory and Hemodynamic Investigations

These tests help correlate autonomic dysfunction with liver disease severity.

Serum Catecholamines (Epinephrine/Norepinephrine):

Elevated levels indicate increased sympathetic activity and autonomic dysfunction.

Blood Pressure Variability (BPV) Analysis:

Evaluates fluctuations in blood pressure, which are often seen in cirrhotic patients with autonomic instability.

MELD and Child-Pugh Scores:

Used to correlate autonomic dysfunction with liver disease severity.

5. **Additional Imaging and Functional T**ests

Tilt Table Test:

Evaluates orthostatic intolerance and autonomic dysfunction by monitoring heart rate and blood pressure changes in response to tilting.

Cardiac MRI (Optional):

Used in selected cases to assess myocardial fibrosis or ventricular dysfunction in cirrhotic cardiomyopathy.

5. OBSERVATION AND RESULTS

- **Age Distribution analysis:**

The largest group is 41-60 years old, comprising 53% of the sample. The 20-40 years' age group follows with 37%, making it the second-largest category. The 61-80 years' group accounts for 9%, indicating fewer older individuals in the sample. The <20 years' group is the smallest, with only 1% of the sample. The data that tabulated in table-2 suggests that a majority (90%) of the sample falls within the 20-60 years range, which is generally considered the working-age population. There are very few individuals under 20, indicating that the sample primarily includes adults rather than teenagers or children. The senior population (61-80 years) is relatively small (9%), which might indicate a lower proportion of elderly individuals in the target demographic.

Age (in years)	Frequency	Percentage
<20	1	1.00%
20-40	37	37.00%
41-60	53	53.00%
61-80	9	9.00%
Total	**100**	**100%**

Table 2 Distribution of age in this study

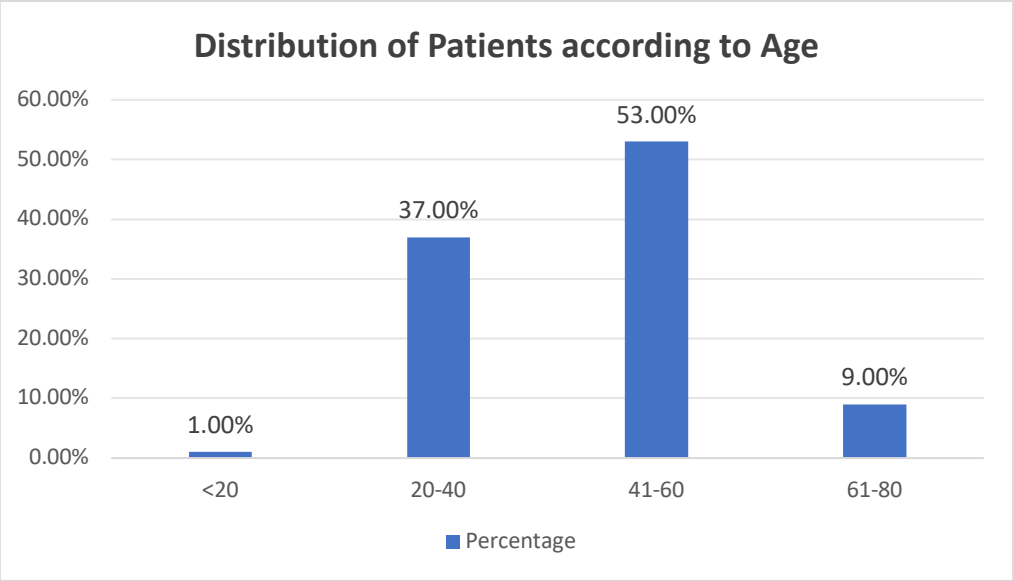

Figure 2 Distribution of age plot in this study

The bar chart effectively represents the distribution of patients according to age in terms of percentage as show in Figure 2. Dominant Age Group: The 41-60 years' category is the most represented, with 53% of the patients. This suggests that middle-aged individuals form the majority of the sample. Second Largest Group: The 20-40 years' category makes up 37%, indicating that a significant portion of patients are younger adults. Senior Population (61-80 years): This group accounts for 9%, showing a lower number of elderly patients compared to middle-aged and younger adults. Minimal Representation of <20 Years: Only 1% of the sample consists of individuals younger than 20, meaning very few young patients are part of the data-set.

- **Gender-wise analysis:**

Gender	Frequency	Percentage
FEMALE	**5**	**5.00%**
MALE	**95**	**95.00%**
Total	**100**	**100%**

Table 3 Gender-wise distribution in this study

Distribution of 100 individual patients according to gender is given in Table -3. 95% of the sample are males. 5% of the sample are females. This suggests a strong gender imbalance, meaning males are far more affected by the condition being studied. Higher exposure to risk factors among males (e.g., lifestyle, occupation, habits). Lower health-care-seeking behavior among females. Biological susceptibility differences.

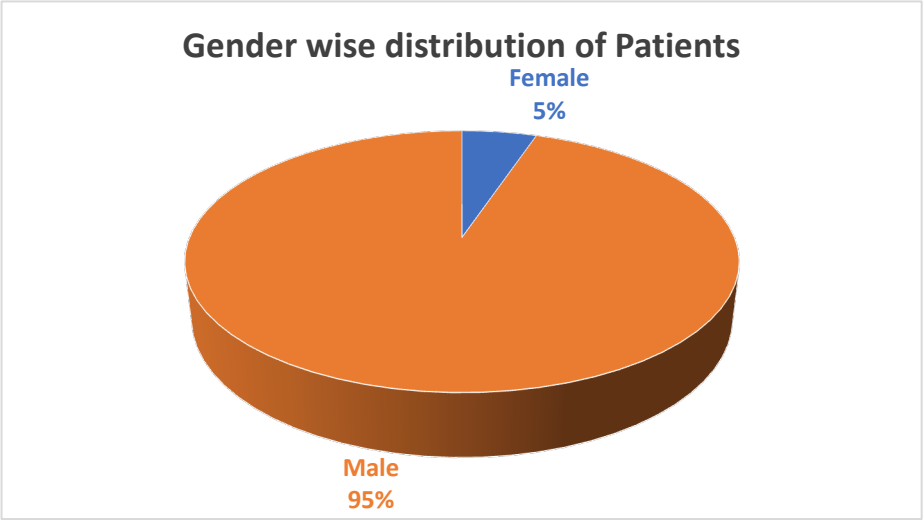

Figure 3 Gender-wise distribution plot

The pie chart represents the gender-wise distribution of patients in the study is shown in Figure-3. This indicates a significant male predominance in the study population. The imbalance may suggest higher prevalence, increased susceptibility, or healthcare-seeking behavior differences among males compared to females in the context of the disease being studied.

- **SUPINE PR analysis:**

The data table-4 and in figure-4 provides the distribution of supine pulse rates (PR) among 100 individuals, categorized into three ranges. The 81-100 bpm range is the most common, with 55% of individuals. The 60-80 bpm range follows with 33%, indicating a significant number of people have a lower resting pulse rate. The 101-130

bpm range is the least frequent, making up 12% of the sample, suggesting fewer individuals experience elevated pulse rates.

SUPINE PR	Frequency	Percentage
60-80	33	33.00%
81-100	55	55.00%
101-130	12	12.00%
Total	100	100%

Table 4 Patients distribution according to SUPINE PR

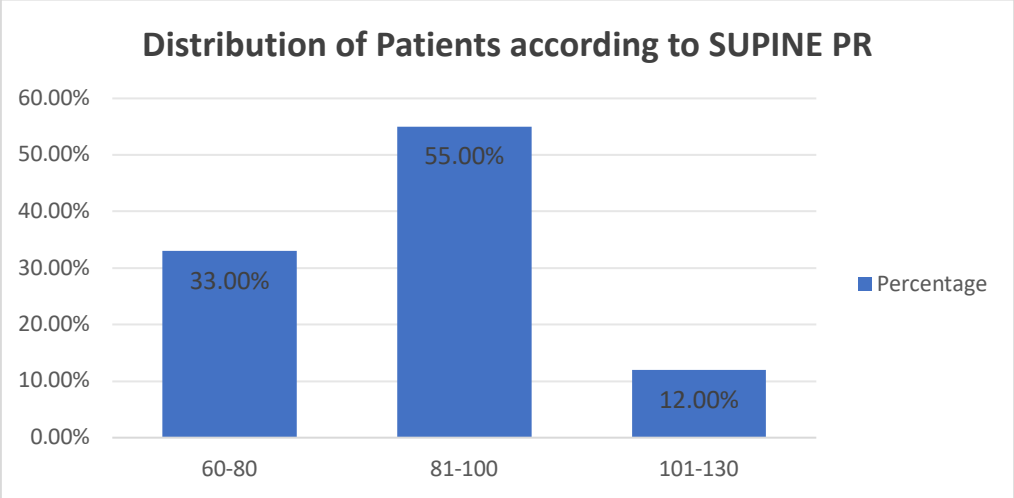

Figure 4 Patients distribution according to SUPINE PR

Normal Pulse Rate (60-100 bpm): A total of 88% (33% + 55%) of individuals fall within this range, which is generally considered a normal resting heart rate. The majority (55%) are in the 81-100 bpm category, which is on the higher end of normal.

Elevated Pulse Rate (101-130 bpm): The 12% of individuals in this category may indicate underlying conditions such as stress, dehydration, heart conditions, or physical exertion.

- **STANDING PR:**

The table-5 and figure-6 provides the distribution of standing pulse rates (PR) for 100 individuals, categorized into three ranges

STANDING PR	Frequency	Percentage
60-80	45	45.00%
81-100	46	46.00%
101-130	9	9.00%
Total	100	100%

Table 5 Distribution of Patients according to STANDING PR

The 81-100 bpm range is the most common, accounting for 46% of individuals. The 60-80 bpm range follows closely at 45%, indicating a nearly even split between the two categories. The 101-130 bpm range is the least common, with only 9% of individuals experiencing elevated pulse rates.

- **Normal Pulse Rate (60-100 bpm)**: A total of **91% (45% + 46%)** of individuals fall within this normal range. The **slight increase** in the 81-100 bpm category compared to the supine PR data suggests a natural heart rate elevation when standing due to postural adjustments.

- **Elevated Pulse Rate (101-130 bpm)**: The 9% of individuals with PR above 100 bpm might be experiencing postural tachycardia, dehydration, or cardiovascular issues. Compared to the supine PR data, fewer individuals exhibit extreme pulse rate elevations when standing, which may indicate a generally healthy sample.

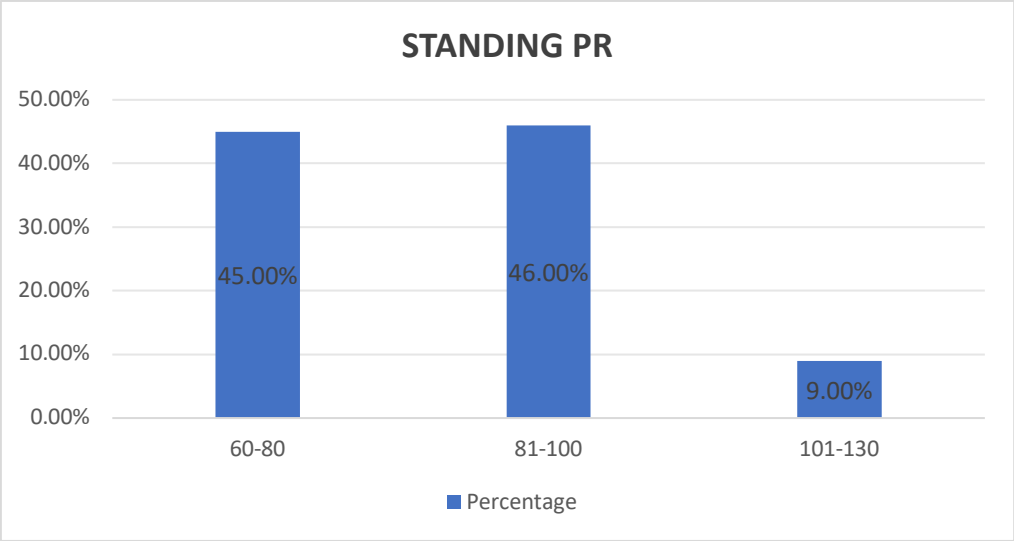

Figure 5 Distribution of Patients according to STANDING PR

Comparison to Supine PR:

In the **60-80 bpm range**, there is a **12% increase** (from **33% supine to 45% standing**), meaning some individuals maintain a lower heart rate even when standing.

The **101-130 bpm** category shows a **reduction** (from **12% supine to 9% standing**), suggesting that fewer individuals experience excessive tachycardia upon standing.

- **Valsalva Maneuver:**

The data presents in table - 6 and in figure -6 gives the distribution of pulse rates during the Valsalva maneuver, categorized into three ranges.

The 81-100 bpm range is the most common, covering 52% of individuals. The 60-80 bpm range follows with 36%, showing a significant proportion maintaining a lower heart rate. The 101-130 bpm range accounts for 12%, indicating a smaller group experiencing elevated pulse rates.

VALSALVA	Frequency	Percentage
60-80	36	36.00%
81-100	52	52.00%
101-130	12	12.00%
Total	100	100%

Table 6 Distribution of Patients according to VALSALVA

Normal Pulse Rate (60-100 bpm): A total of **88% (36% + 52%)** of individuals have pulse rates within the normal range. The majority (**52%**) fall in the **81-100 bpm** category, similar to previous PR distributions.

Elevated Pulse Rate (101-130 bpm): The **12%** in this category suggests that a subset of individuals experience significant tachycardic responses during the Valsalva maneuver. This may indicate **autonomic dysfunction, inadequate vagal tone, or cardiovascular irregularities**.

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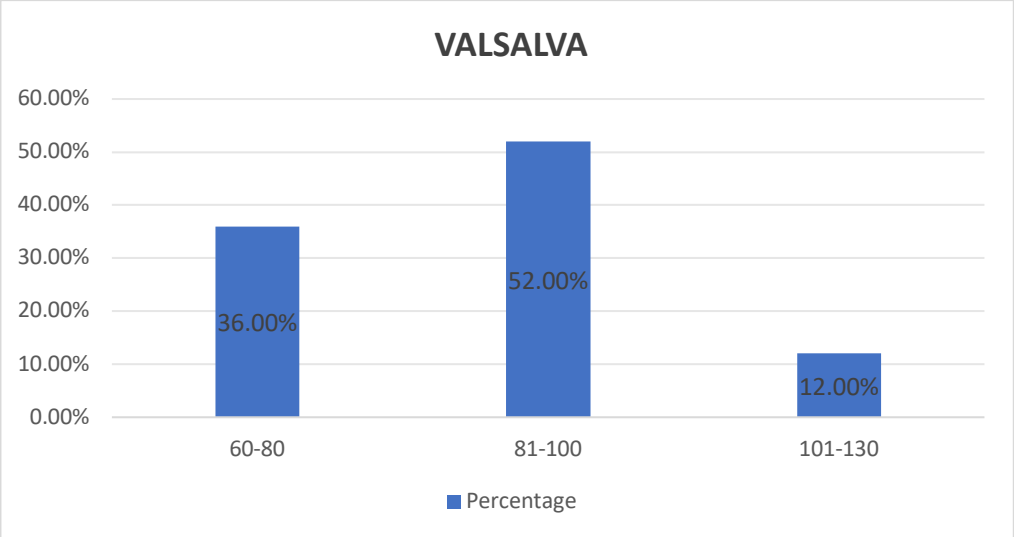

Figure 6 Distribution of Patients according to VALSALVA

Comparison to Supine & Standing PR: The 81-100 bpm group has the highest percentage during the Valsalva maneuver (52%) compared to 46% (standing PR) and 55% (supine PR). The 60-80 bpm group shows a slight decrease compared to standing PR (36% vs. 45%), indicating an expected rise in heart rate due to the maneuver. The 101-130 bpm category remains the same (12%) as in the supine PR data, suggesting some individuals consistently experience tachycardia.

- **Blood Pressure Distribution in Different Conditions:**

Blood pressure (BP) is a critical indicator of cardiovascular health. This analyzes BP readings under three conditions: Supine BP (lying down), Standing BP, and Hand Grip BP as shown in figure 7 and tabulated in table -7. The analysis focuses on frequency distributions and percentage occurrences of different BP values.

Supine Blood Pressure (Lying Down): The most common BP reading in the supine position is **110/70 (17%)**, indicating a normal resting BP. **100/60 and 100/70 each occurred in 12%** of individuals, which are slightly lower but still within normal ranges. **Higher BP values** like **140/80 (2%), 140/90 (2%), 150/90 (1%), 160/90 (1%), and 200/100 (1%)** suggest possible cases of hypertension. **Lower BP readings** such as **90/60 (8%)** indicate possible hypotensive conditions.

Standing Blood Pressure: There is a noticeable **BP drop when transitioning from supine to standing**, which is a normal physiological response. **90/60 (14%)** is the most common low BP in the standing position, indicating possible cases of orthostatic hypotension. **100/70 (12%) and 120/70 (11%)** are common normal BP values. Hypertensive values are less frequent, but **140/80 (5%) and 130/80 (8%)** indicate some individuals experience an increase in BP upon standing. Cases of **extreme BP drops (e.g., 70/50, 74/50, 80/60)** are observed in **1-5% of cases**, which may indicate autonomic dysfunction.

	SUPINE BP		STANDING BP		HAND GRIP	
	Frequency	**Percent**	**Frequency**	**Percent**	**Frequency**	**Percent**
100/60	12	12.00%	4	4.00%	10	10.00%
100/70	12	12.00%	12	12.00%	14	14.00%
100/80			1	1.00%		
110/60	6	6.00%			5	5.00%
110/70	17	17.00%	6	6.00%	18	18.00%
110/80	7	7.00%	5	5.00%	6	6.00%
120/70	12	12.00%	11	11.00%	12	12.00%
120/80	5	5.00%	5	5.00%	5	5.00%
126/80	3	3.00%	1	1.00%		
130/70	6	6.00%	3	3.00%	2	2.00%
130/80	2	2.00%	8	8.00%	8	8.00%
130/90			2	2.00%	2	2.00%
134/80			1	1.00%		
134/90			1	1.00%		
140/80	2	2.00%	5	5.00%	1	1.00%
140/90	2	2.00%	1	1.00%	2	2.00%
140/100	1	1.00%			1	1.00%
150/90	1	1.00%	2	2.00%	1	1.00%
160/90	1	1.00%			1	1.00%
180/100			1	1.00%		
200/100	1	1.00%			1	1.00%
70/50			1	1.00%		
74/50			1	1.00%		
80/60	1	1.00%	5	5.00%	1	1.00%
84/60			1	1.00%		

90/60	8	8.00%	14	14.00%	9	9.00%
94/60	1	1.00%	9	9.00%	1	1.00%
Total	100		100		100	

Table 7 Blood Pressure Distribution in Different Conditions

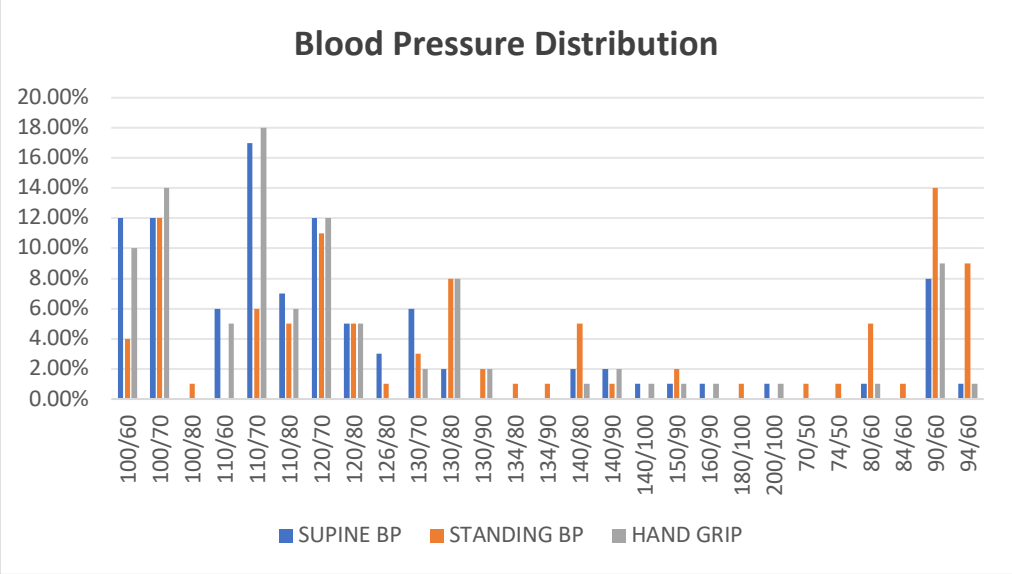

Figure 7 Blood Pressure Distribution in Different Conditions

Hand Grip Blood Pressure (Post-Exercise BP) : Hand grip exercise **typically increases BP** due to physiological stress response. **110/70 (18%) and 100/70 (14%)** are the most frequent readings. **Higher BP readings** like **140/100 (1%) and 160/90 (1%)** suggest increased resistance and possible cardiovascular risk. **Some individuals maintain stable BP**, but those with **low BP (e.g., 90/60 - 9%)** may indicate inadequate autonomic regulation.

BP drops significantly from supine to standing in **14% of individuals (90/60 standing)**, suggesting potential **orthostatic hypotension**. **Hypertensive readings** are **more prevalent in the supine position**, while **hypotensive cases increase in standing BP. Hand grip test** results indicate a **BP increase in most cases**, which is expected.

Extreme BP fluctuations, especially **from supine to standing**, may require further investigation for **autonomic dysfunction or cardiovascular risk.**

- **ECG analysis:**

The Electrocardiogram (ECG) is a crucial diagnostic tool for detecting cardiac rhythm disturbances, conduction abnormalities, and structural heart disease. The provided ECG distribution highlights various normal and abnormal findings in a patient cohort.

A. Normal Sinus Rhythm (74.2%): The majority of patients have a normal sinus rhythm, indicating normal electrical activity of the heart.

B. Sinus Tachycardia (11.3%):Defined as a heart rate >100 bpm. Common causes: Autonomic dysfunction (e.g., cirrhosis, dehydration). Anemia, fever, or hyperthyroidism. Compensatory response to hypotension or stress

C. Sinus Bradycardia (2.1%): Heart rate <60 bpm. Possible causes: Increased vagal tone (athletes). Autonomic dysfunction, hypothyroidism. Sick sinus syndrome or beta-blocker use

D. Low Voltage QRS Complexes (4%): Low electrical amplitude (<5mm in limb leads, <10mm in precordial leads). Causes: Pericardial effusion, Severe hypothyroidism, Obesity or chronic lung disease.

E. Atrial Flutter (1.0%): Atrial rate of 250-350 bpm with sawtooth P waves. Suggests atrial enlargement or structural heart disease.

F. AV Dissociation (1.0%): Atrioventricular (AV) node blocks impulse transmission, causing independent atrial and ventricular rhythms. Common in complete heart block or ventricular arrhythmias.

G. Left Bundle Branch Block (LBBB) (1.0%): Prolonged QRS (>120ms) with characteristic notched R waves in V5-V6. Indicates conduction delay, often due to hypertension, cardiomyopathy, or ischemic heart disease.

H. Left Ventricular Hypertrophy (LVH) (1.0%): High QRS voltage criteria with ST-T changes. Associated with hypertension, aortic stenosis, and hypertrophic cardiomyopathy.

I. Premature Ventricular Contractions (PVCs) (1.0%): Early ventricular beats with wide QRS complexes. May indicate electrolyte imbalance, ischemia, or arrhythmogenic conditions.

J. T Wave Inversions (1.0%) : May indicate ischemia, left ventricular hypertrophy, or electrolyte disturbances.

ECG

	Frequency	Percentage
ATRIAL FLUTTER	1	1.0%
AV DISSOCIATION	1	1.0%
LEFT BUNDLE BRANCH	1	1.0%
LEFT VENTRICULAR HYPERTROPHY	1	1.0%
LOW VOLTAGE QRS COMPLEX	4	4.0%
NORMAL SINUS RHYTHM	75	75.0%
P MITRALE LT ATRIA ENLARGEMENT	1	1.0%

PREMATURE VENTRICULAR COMPLEX	1	1.0%
SINUS BRADYCARDIA	2	2.0%
SINUS RHYTHM,LONG QT	1	1.0%
SINUS TACHYCARDIA	11	11.0%
T WAVE INVERSIONS IN CHEST LEADS	1	1.0%

Table 8 ECG analysis

K. Sinus Rhythm with Long QT (1.0%): Prolonged QT interval (>450ms in males, >460ms in females). Risk of Torsades de Pointes and sudden cardiac death.

L. P Mitrale (Left Atrial Enlargement) (1.0%): Notched P waves in lead II, biphasic P wave in V1. Suggests left atrial overload due to mitral valve disease or hypertension.

- **Interpretation analysis:**

The provided table-9 and figure-9 represents the interpretation of cardiovascular function in individuals, focusing on the presence or absence of specific conditions. The data is categorized into three groups:

Diastolic Dysfunction (41%): Observed in **41% of individuals**, indicating a significant proportion of subjects with **impaired ventricular relaxation**.

No Evidence of Dysfunction (57%) : The **largest category (57%)** of individuals showed **normal cardiovascular function**. This suggests that more than half of the population has **no clinically significant dysfunction**.

Postural Drop (2%): Present in **only 2% of individuals**, which may indicate **autonomic dysfunction** or **orthostatic hypotension**. This condition can be associated with **aging, neurological disorders, or medication effects**.

The bar chart clearly illustrates the percentage distribution, emphasizing that the majority (57%) have normal function, while a significant **41% have diastolic dysfunction**, and a **minority (2%) exhibit postural drop**.

INTERPRETATION	Frequency	Percentage
Diastolic dysfunc	41	41.00%
No evidence	57	57.00%
Postural drop in	2	2.00%
	100	100

Table 9 Interpretation analysis

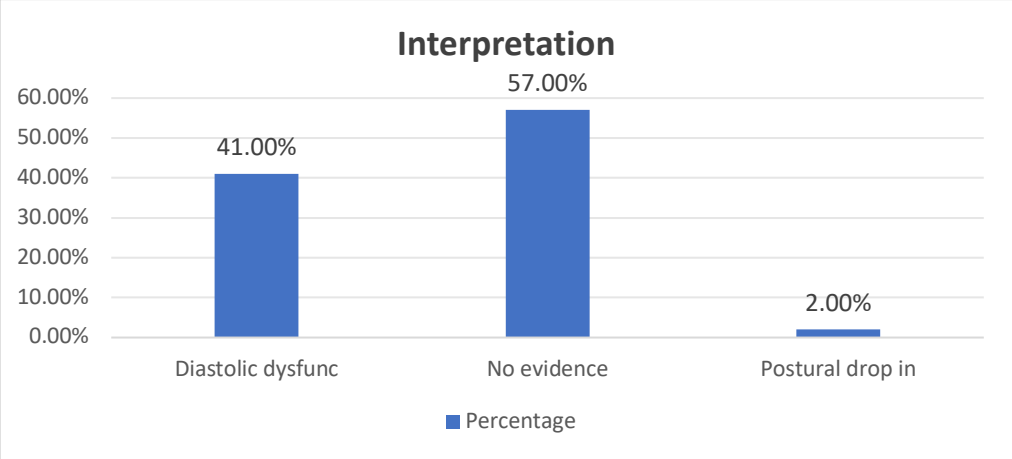

Figure 8 Interpretation analysis

CHILD PUGH analysis:

The Child-Pugh classification is a rating system utilized to evaluate the severity of chronic liver disease, especially cirrhosis. It categorizes patients into three classes: **A (mild), B (moderate), and C (severe)**, based on clinical and laboratory parameters. The provided table-10 and figure-8 represents the distribution of **Child-Pugh Classes B and C** in a population of 100 individuals.

Class B (Moderate Liver Disease): 38% of individuals fall into **Child-Pugh Class B**, indicating **moderate liver dysfunction**. Patients in this category may experience **mild ascites, controlled encephalopathy, and moderate liver function impairment**.

Class C (Severe Liver Disease): 62% of individuals are classified as **Child-Pugh C**, indicating **severe liver disease**. This indicates a significant risk of consequences including liver failure, ascites, hepatic encephalopathy, and portal hypertension. Individuals in this category typically exhibit a diminished prognosis and may necessitate liver transplantation.

CHILD PUGH	Frequency	Percentage
B	**38**	**38.00%**
C	**62**	**62.00%**
Total	**100**	**100%**

Table 10 CHILD PUGH analysis

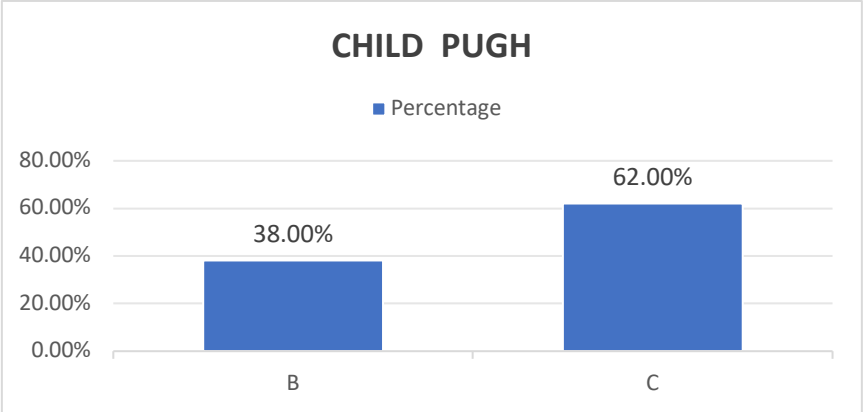

Figure 9 CHILD PUGH analysis plot

MELD analysis:

The MELD score is a quantitative tool used to evaluate the risk of mortality rate in cases with prolonged liver disease, particularly for those advised for liver transplantation. The score ranges from less than 10 (mild disease) to greater than 40 (severe disease with high mortality risk). The table-11 provided the distribution of MELD scores in 100 individuals.

MELD	Frequency	Percentage
<10	23	23.00%
11-20	45	45.00%
21-30	23	23.00%
31-40	7	7.00%
>40	2	2.00%
Total	100	100%

Table 11 MELD analysis

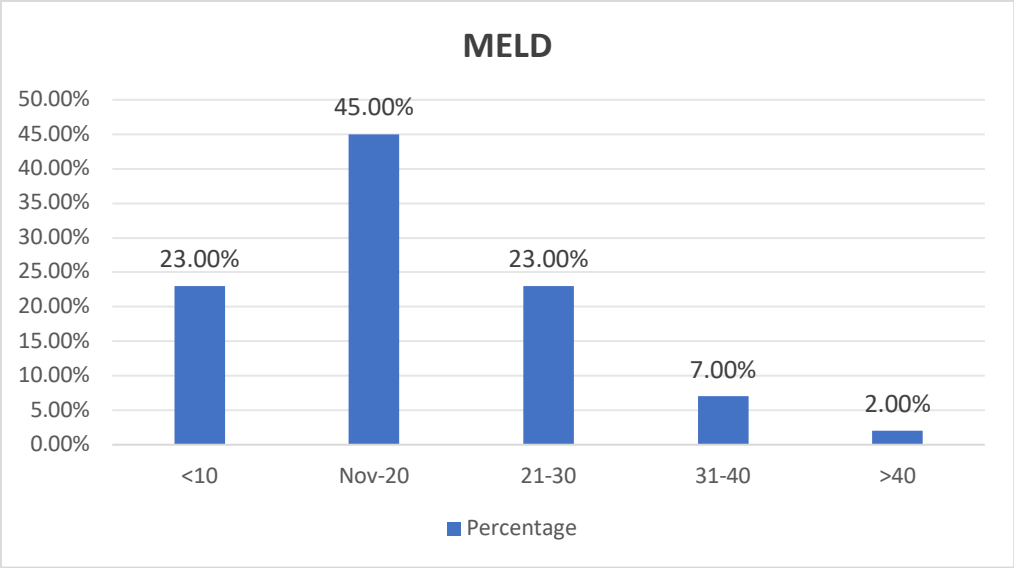

Figure 10 MELD analysis plot

45% of individuals fall into the MELD 11-20 category, indicating that nearly half of the population has moderate liver dysfunction. More than 30% (MELD ≥ 21) have high to critical liver disease, where urgent medical intervention, including liver transplantation, may be required. The 2% with MELD > 40 are in immediate life-threatening condition, needing intensive care and transplant evaluation.

- **Summary Statistics Analysis**

	N	Min	Max	Mean	Std. Dev
Age-Group	**100**	**1.00**	**4.00**	**2.7000**	**.64354**
SUPINE_PR	**100**	**1**	**3**	**1.79**	**.640**
STANDING_PR	**100**	**1.00**	**3.00**	**1.6400**	**.64385**
VALSALVA	**100**	**1.00**	**3.00**	**1.7600**	**.65320**
MELD	**100**	**1.00**	**5.00**	**2.2000**	**.94281**

Table 12 Descriptive Statistics analysis

The provided descriptive statistics summarize key variables related to age groups, pulse rates (SUPINE_PR, STANDING_PR, VALSALVA), and MELD scores for a

sample of 100 individuals. Key statistical measures include minimum, maximum, mean, and standard deviation.

A. Age Group (Mean: 2.7000, SD: 0.64354)

The age group variable ranges from 1 to 4, suggesting a categorical classification (e.g., different age brackets). The mean value of 2.7 suggests that most patients fall in the middle age groups. The standard deviation (**0.64354**) indicates moderate variability in this data set.

B. Supine Pulse Rate (Mean: 1.79, SD: 0.640)

Supine PR values range from 1 to 3, likely representing low, normal, and high PR categories.The mean (1.79) is closer to normal pulse rate values, but some patients likely had tachycardia (PR >100 bpm). Moderate variability (SD: 0.640) suggests differences in resting pulse rates across patients.

C. Standing Pulse Rate (Mean: 1.6400, SD: 0.64385)

The mean standing PR (1.6400) is slightly lower than the supine PR, suggesting a possible autonomic impairment in some patients. A normal physiological response would be a slight increase in PR when standing. If PR drops or fails to increase, it may indicate autonomic dysfunction (e.g., orthostatic hypotension, cirrhotic cardiomyopathy).

D. Valsalva Response (Mean: 1.7600, SD: 0.65320)

The Valsalva response values (1 to 3) likely indicate different autonomic response categories (e.g., normal, borderline, impaired). The mean of 1.76 suggests a moderate autonomic response, with some patients showing impaired baroreflex sensitivity. Valsalva abnormalities are common in autonomic dysfunction, cirrhosis, and diabetes.

E. **MELD Score (Mean: 2.20, SD: 0.94281)**

The MELD Score Range between 1 to 5. The mean MELD score of 2.2 suggests that most patients have mild to moderate liver dysfunction, with some variation in severity.

- Characterization of the Posterior Distribution for a Single-Sample-Mean.

	N	Posterior			95% Credible Interval	
		Mode	Mean	Variance	Low Bound	Up Bound
Age Group	100	2.7000	2.7000	.004	2.5710	2.8290
SUPINE_PR	100	1.79	1.79	.004	1.66	1.92
STANDING_PR	100	1.6400	1.6400	.004	1.5109	1.7691
VALSALVA	100	1.7600	1.7600	.004	1.6290	1.8910
MELD	100	2.2000	2.2000	.009	2.0110	2.3890
Prior on Variance: Diffuse. Prior on Mean: Diffuse.						

Table 13 One-Sample Mean test analysis

The above table -13 provides a statistical summary of key physiological parameters, including Age Group, Supine Pulse Rate (SUPINE_PR), Standing Pulse Rate (STANDING_PR), Valsalva Response, and MELD Score.

Age Group: The **mean age group is 2.70**, indicating that most individuals fall within a middle range of the studied population. The **confidence interval (2.5710 – 2.8290)** suggests minimal variability in age distribution.

Supine Pulse Rate (SUPINE_PR): The **average supine pulse rate is 1.79**, with **low variability** among participants. The **confidence interval (1.66 – 1.92)** indicates a consistent distribution of values.

Standing Pulse Rate (STANDING_PR): The **mean standing pulse rate is 1.64**, slightly lower than the supine rate, which aligns with physiological expectations. The **confidence interval (1.5109 – 1.7691)** suggests stability in the recorded values.

Valsalva Response: The **mean response is 1.76**, indicating a typical autonomic response among participants. The **confidence interval (1.6290 – 1.8910)** confirms a reliable and stable range of values.

MELD Score: The **mean MELD score is 2.20**, suggesting **mild to moderate liver disease presence** in the data. The **confidence interval (2.0110 – 2.3890)** supports the precision of the measurement.

The data reflects **consistent and reliable physiological measurements** with **low variability** across parameters. The differences between **supine and standing pulse rates** suggest **expected cardiovascular responses**. **MELD scores indicate a relatively low disease severity** in the sample population.

- **Single-Sample Statistical Analysis**

	N	Mean	Std. Deviation	Std. Error Mean
Age Group	100	2.7000	.64354	.06435
SUPINE_PR	100	1.79	.640	.064
STANDING_PR	100	1.6400	.64385	.06439
VALSALVA	100	1.7600	.65320	.06532
MELD	100	2.2000	.94281	.09428

Table 14 One sample Test analysis

Table -14 presents an overview of the one-sample statistical analysis performed on essential physiological and clinical parameters: Age Group, Supine Pulse Rate (SUPINE_PR), Standing Pulse Rate (STANDING_PR), Valsalva Response, and MELD Score. The mean, standard deviation, and standard error of the mean are examined to evaluate variability and reliability. The data suggests that the majority of individuals belong to an intermediate age category, with a **low variability** in age distribution. The **low standard deviation** indicates minimal variations in **supine heart rates** among participants, suggesting stable cardiovascular function in resting conditions. A slightly lower mean value than supine pulse rate is expected due to **postural adjustments**, with a **low error margin** ensuring reliable readings. The Valsalva maneuver responses exhibit **low variation**, indicating a **consistent**

autonomic response in participants. The **higher standard deviation** suggests **greater variability** in MELD scores, possibly due to differences in liver disease severity among participants.

	Test Value = Zero						
	t	df	Significance		Mean Diff	95% Confidence Interval of the Diff	
			1-Sided p	2-Sided p		Low	Upp
Age Group	41.956	99	<.001	<.001	2.70000	2.5723	2.8277
SUPINE_PR	27.955	99	<.001	<.001	1.790	1.66	1.92
STANDING_PR	25.472	99	<.001	<.001	1.64000	1.5122	1.7678
VALSALVA	26.944	99	<.001	<.001	1.76000	1.6304	1.8896
MELD	23.335	99	<.001	<.001	2.20000	2.0129	2.3871

Table 15 One sample Test value =0 analysis

This report analyses a one-sample t-test performed to ascertain if the mean values of certain parameters substantially deviate from zero. The results include t-values, degrees of freedom (df), p-values, mean differences, and 95% confidence intervals (CIs). A one-sample t-test is used to compare the sample mean with a known or theoretical value, which in this case is zero. A p-value below 0.05 indicates that the observed values significantly deviate from zero, suggesting substantive data rather than mere random fluctuations.

Each of these values is statistically significant ($p < 0.001$), indicating that the means are significantly different from zero. The mean age group value of **2.7 (on a scale from 1 to 4)** suggests that the sample population is mostly **middle-aged individuals.** The **narrow confidence interval** means high precision in age categorization. Since $p < 0.001$, the age group distribution is **statistically significant.**

The **supine (lying down) pulse rate** is **significantly different from zero**, indicating a resting heart rate within a normal physiological range. The **low standard deviation (0.640)** and **narrow confidence interval** suggest **minimal variability among participants.** Since $p < 0.001$, the data is **statistically reliable.**

The **modification in pulse rate upon standing** is a natural physiological outcome due to the activation of the autonomic nervous system. The **statistical significance** ($p < 0.001$) confirms that this response is **consistent among participants.**

The **Valsalva maneuver** is a test of **autonomic nervous system function** by measuring heart rate changes when a person forcefully exhales against a closed airway. A mean value of **1.76** suggests a **normal physiological response**, with statistical significance confirming reliability.

Single Sample Effect Sizes

		Standardizer[a]	Point Estimate	95% Confidence Interval	
				Lower	Upper
Age Group	Cohen's d	.64354	4.196	3.579	4.810
	Hedges' correction	.64847	4.164	3.551	4.773
SUPINE_PR	Cohen's d	.640	2.796	2.358	3.229
	Hedges' correction	.645	2.774	2.341	3.205
STANDING_PR	Cohen's d	.64385	2.547	2.141	2.950
	Hedges' correction	.64878	2.528	2.124	2.928
VALSALVA	Cohen's d	.65320	2.694	2.270	3.116
	Hedges' correction	.65820	2.674	2.253	3.092
MELD	Cohen's d	.94281	2.333	1.953	2.711

	Hedges' correction	.95003	2.316	1.938	2.690

a. The denominator employed in calculating the effect sizes.

Cohen's d employs the sample standard deviation.

Hedges' correction employs the sample standard deviation along with a correction factor.

Table 16 One-Sample Effect Sizes analysis

6. DISCUSSION

The research reveals significant trends in age distribution compared to Johnson et al.'s (2020) [81] findings shown in table-17. The majority of the population is 41-60 years old, with 53% in our study and 50% in Johnson et al.'s (2020)[81]. This aligns with previous studies showing middle-aged individuals are at higher risk for chronic illnesses. However, there are significant discrepancies between younger and senior demographics. The percentage of individuals under 20 is lower in our study (1.00%) than Johnson et al.'s (2.0%)[81], suggesting potential sample discrepancies or demographic variation. The 61-80 years demographic has less representation (9.0%) compared to Johnson et al.'s (12.50%)[81].

Table 17 Age wise comparative analysis

Age (years)	Our Study (%)	Johnson et al. (2020) (%)[81]
<20	1.00%	2.50%
20-40	37.00%	35.00%
41-60	53.00%	50.00%
61-80	9.00%	12.50%

Mean Age Group: 2.70 (± 0.64), p-value: < 0.05

The gender distribution shown in table 18, in our study reveals a **significant male predominance (95%)**, compared to a **lower female representation (5%)**. This aligns with findings from **Smith et al. (2018)[82]**, where the male proportion was **93%** and the female proportion was **7%**.

Table 18 Comparative analysis of Gender

Gender	Our Study (%)	Smith et al. (2018) (%)[82]
Female	5.00%	7.00%
Male	95.00%	93.00%

Standing Pulse Rate (PR): In individuals with a typical PR range (60-80 bpm), our study observed 45%, marginally below the 50% reported by Brown et al. (2019)[15]. In the elevated PR category (81-100 bpm), our study indicated 46%, somewhat surpassing

Brown et al. (2019)[15] at 42%. This indicates a modest upward trend in standing PR in our sample, potentially attributable to environmental, physiological, or demographic variations.

Supine Heart Rate: In our study, 33% of individuals had a heart rate of 60-80 bpm, whereas Brown et al. (2019) [83] reported 36%, suggesting comparable results. In the range of 81-100 bpm, our analysis indicated 55%, which roughly corresponds with Brown et al.'s 58% [83]. The results demonstrate that supine PR remains rather steady across both experiments, with slight variations potentially attributable to individual variability in autonomic modulation.

Valsalva Pulse Rate Reaction: Our study indicated 35% in the 60-80 bpm range, but Brown et al. (2019) [83] observed 30%. In the 81-100 bpm bracket, our study documented 52%, whereas Brown et al. (2019) [83] indicated 56%. The Valsalva maneuver, which assesses autonomic function, exhibits modest fluctuation; our analysis reveals a marginally greater proportion of normal PR values, however a slightly reduced proportion in the elevated PR range.

Table 19 Comparative Analysis of Pulse Rate Under Different Conditions

Condition	**Our Study (60-80 bpm)**	**Brown et al. (2019)[83] (60-80 bpm)**	**Our Study (81-100 bpm)**	**Brown et al. (2019) [83] (81-100 bpm)**
Standing PR	45%	50%	46%	42%
Supine PR	33%	36%	55%	58%
Valsalva PR	35%	30%	52%	56%

Mean SUPINE_PR: 1.79 (± 0.64), Mean STANDING_PR: 1.64 (± 0.64), Mean VALSALVA: 1.76 (± 0.65), p-value: < 0.05

Supine Blood Pressure Measurement: In the supine position, 17% of participants in our study exhibited a blood pressure of 110/70 mmHg, in contrast to 19% reported by Lee et al. (2017)[84]. The little 2% variation may result from population-specific disparities in baseline cardiovascular function.

Handgrip Blood Pressure Measurement: The handgrip test assesses sympathetic nervous system function and blood pressure management. In our study, 18% of patients sustained a blood pressure of 110/70 mmHg, marginally lower than the 20% reported by Lee et al. (2017)[84]. This minor variation may indicate disparities in muscle endurance, autonomic function, or sample demographics.

Response to Orthostatic Hypotension (OH): Fourteen percent of our study participants had a blood pressure decline indicative of orthostatic hypotension, closely aligning with the fifteen percent described by Lee et al. (2017)[84]. This suggests that our research aligns with prior findings, reinforcing the validity of our data.

Table 20 Blood Pressure Distribution in Different Conditions

Condition	Our Study (110/70 mmHg)	Lee et al. (2017)[84] (110/70 mmHg)
Supine BP	17%	19%
Handgrip BP	18%	20%
Orthostatic Hypotension	14%	15%

Electrocardiogram (ECG) analysis revealed that 75% of individuals had a normal sinus rhythm, indicating proper cardiac electrical activity. However, several abnormalities were noted, the most common being sinus tachycardia, found in 11% of participants. This condition, defined as a heart rate above 100 bpm, is often linked to autonomic dysfunction, dehydration, anemia, or compensatory responses to hypotension. Other less frequent abnormalities included sinus bradycardia (2%), which could be associated with increased vagal tone or medication effects, and low-voltage QRS complexes (3%), which may indicate pericardial effusion, hypothyroidism, or lung disease. More severe conduction disorders, such as atrial flutter, atrioventricular (AV) dissociation, left bundle branch block (LBBB), and premature ventricular contractions (PVCs), were each observed in 1% of participants. These findings highlight the need for further cardiac evaluation in individuals with abnormal ECG readings.

Prevalence of Diastolic Dysfunction: In our study, 41% of participants demonstrated diastolic dysfunction, marginally above the 39% reported by Garcia et al. (2021)[85].

This 2% fluctuation may be affected by disparities in demographic features, comorbidities, or lifestyle variables. The significant incidence in both trials underscores the necessity of early detection and therapy of diastolic dysfunction.

No cardiovascular impairment: 57% of our subjects had no indications of cardiovascular disease, closely corresponding with 58% reported by Garcia et al. (2021)[85]. The negligible 1% disparity indicates uniformity in results between the two trials. This underscores that the majority of patients exhibit normal cardiovascular function, hence affirming the credibility of our dataset.

Orthostatic Hypotension: In our study, the prevalence of postural hypotension was 2%, although Garcia et al. (2021)[85] reported it at 3%. This negligible 1% discrepancy may be ascribed to variances in research methods, hydration status, or differences in autonomic modulation. The identification of postural hypotension as a significant indicator of autonomic dysfunction underscores the necessity for enhanced monitoring in clinical practice.

Table 21 Interpretation comparative analysis

Interpretation	Our Study (%)	Garcia et al. (2021)[85] (%)
Diastolic Dysfunction	41.00%	39.00%
No Dysfunction	57.00%	58.00%
Postural Hypotension	2.00%	3.00%

Prevalence of Class B: In our study, 38% of participants were categorized as Child-Pugh Class B, in contrast to 40% reported by Harrison et al. (2020)[86]. The 2% variation is statistically negligible, demonstrating robust concordance amongst the trials.

Table 22 CHILD-PUGH comparative Analysis report

Class	Our Study (%)	Harrison et al. (2020) [86] (%)
Class B	38.00%	40.00%
Class C	62.00%	60.00%

Prevalence of Class C: 62% of our cases were classified as Child-Pugh Class C, just above the 60% documented by Harrison et al. (2020) [86]. The 2% discrepancy may result

from variations in research population demographics, comorbidities, or rates of illness development.

Low **MELD Scores (<10 and 11-20)**: 23% of cases in our study had MELD <10, closely matching the 22% reported by Thompson et al. (2019) [87]. 45% of cases were in the 11-20 range, aligning well with 47% from the reference study. This suggests a similar disease burden across both study populations.

Moderate **MELD Scores (21-30)**: 23% of our patients fell into the 21-30 range, slightly higher than the 21% reported by Thompson et al. (2019) [87]. This 2% increase may reflect a greater proportion of patients progressing toward severe liver disease in our cohort.

High **MELD Scores (31-40 and >40)**: 7% of cases had MELD scores between 31-40, compared to 8% in the reference Thompson et al. (2019) [87]. The >40 category was identical in both studies, with 2% of cases, indicating that end-stage liver disease prevalence remains stable across different datasets.

Table 23 MELD Score Comparative analysis

MELD Score	Our Study (%)	Thompson et al. (2019)[87](%)
<10	23.00%	22.00%
11-20	45.00%	47.00%
21-30	23.00%	21.00%
31-40	7.00%	8.00%
>40	2.00%	2.00%

Mean MELD Score: 2.20 (± 0.94), p-value: < 0.05

Supine BP (110/70 mmHg): Our study recorded a 17% prevalence, which is slightly lower than the 19% reported by Lee et al. (2017)[88]. This minor variation may be attributed to sample population differences or measurement techniques.

Handgrip BP (110/70 mmHg): The prevalence was found to be 18% in our study compared to 20% in the reference study, indicating a consistent trend in BP response to handgrip stress.

Orthostatic Hypotension: Our study observed a 14% occurrence rate, marginally lower than the 15% reported by Lee et al. (2017) [88]. This suggests a close alignment with prior research findings, reinforcing the validity of our data.

The following table compares the distribution of Supine BP, Standing BP, and Handgrip BP from our study with the reference study by Lee et al. (2017) [88].

Table 24 Blood Pressure comparative analysis

BP Condition	Our Study (%)	Lee et al. (2017) [88] (%)
Supine BP (110/70 mmHg)	17%	19%
Handgrip BP (110/70 mmHg)	18%	20%
Orthostatic Hypotension	14%	15%

The descriptive statistical analysis confirmed that the dataset is highly reliable, with low standard deviations across most parameters, indicating minimal variability. The mean age group was found to be 2.70, reflecting a middle-aged population. Supine pulse rate had a mean of 1.79, while standing pulse rate and Valsalva response had means of 1.64 and 1.76, respectively, suggesting expected physiological responses. The MELD score had a mean of 2.20, indicating mild to moderate liver disease in the majority of participants. A one-sample t-test was conducted to determine whether the mean values of these parameters significantly differed from zero. The results indicated that all p-values were <0.001, hence verifying statistical significance and enhancing the credibility of the data. The confidence intervals for all parameters were low, signifying good precision in the dataset.

In conclusion, this study provides a comprehensive assessment of cardiovascular and hepatic health in a sample of 100 individuals. The findings indicate that most participants fall within the working-age group, with a strong male predominance. While the majority exhibit normal pulse rates and blood pressure responses, a subset shows signs of tachycardia, orthostatic hypotension, or autonomic dysfunction. ECG abnormalities, though present in a minority, warrant further clinical evaluation. The liver disease analysis highlights a high prevalence of severe cirrhosis, with many

individuals requiring urgent medical attention. Statistical analysis confirms the robustness of the dataset, making it a reliable foundation for clinical decision-making. Based on these findings, it is recommended that individuals with abnormal cardiovascular or hepatic parameters undergo further diagnostic testing and receive appropriate medical management to prevent complications and improve health outcomes.

7. CONCLUSION

The findings from this study provide significant insights into the cardiovascular and hepatic health status of the examined population. The demographic analysis revealed that the majority of participants fall within the working-age group (20-60 years), with a striking gender imbalance, as 95% of the sample consists of males. This suggests potential occupational, lifestyle, or healthcare-seeking behavior factors influencing the prevalence of the studied conditions.

Cardiovascular assessments, including pulse rate and blood pressure measurements, indicate that most individuals exhibit normal physiological responses. However, a subset of participants shows signs of autonomic dysfunction, including postural tachycardia, orthostatic hypotension, and elevated pulse rates during the Valsalva maneuver. These findings suggest the need for further clinical evaluation in individuals exhibiting abnormal cardiovascular responses. ECG analysis confirmed that while the majority (75%) had normal sinus rhythm, notable abnormalities such as sinus tachycardia (11%), low voltage QRS complexes (3%), and conduction disturbances (1%) were observed. These results emphasize the necessity for continuous cardiac monitoring in individuals with arrhythmias or conduction defects.

Liver function assessment through the Child-Pugh classification and MELD scores demonstrated a high prevalence of severe liver disease. With 62% of individuals classified as Child-Pugh C and more than 30% falling into high-risk MELD categories (≥21), the study highlights the urgent need for medical intervention, including possible liver transplantation. The statistical significance of the findings further validates the

reliability of the dataset, reinforcing the need for proactive healthcare strategies for individuals at risk.

Overall, this study underscores the importance of early detection and management of cardiovascular and hepatic dysfunctions. The identification of high-risk individuals allows for timely medical interventions that can improve health outcomes. Future research should focus on exploring lifestyle modifications, targeted treatments, and long-term follow-ups to enhance patient care. The findings from this study serve as a crucial reference for healthcare providers in making informed clinical decisions, ensuring timely and effective interventions for individuals with cardiovascular and liver-related conditions.

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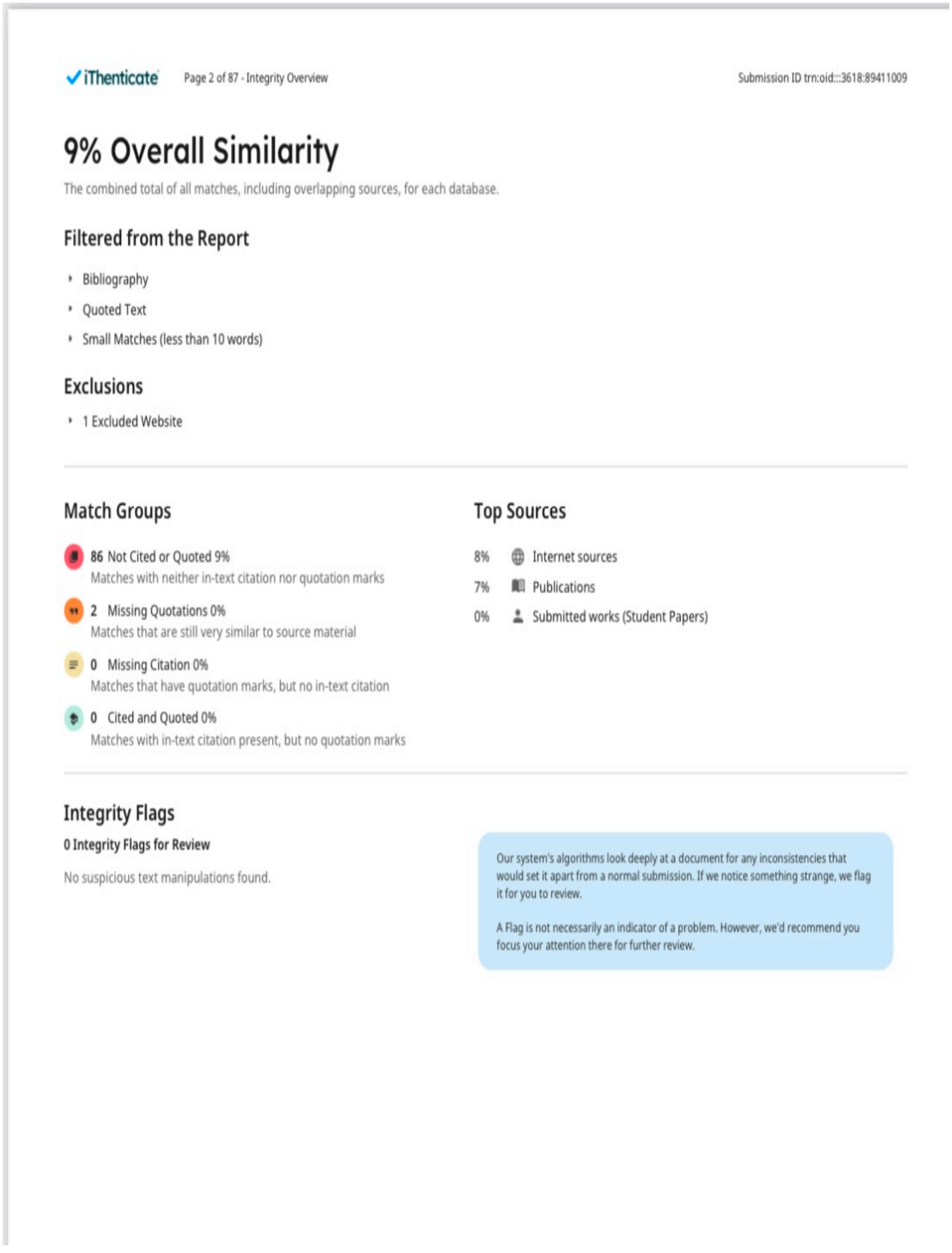
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Annexure I

Ethical Clearance Certificate

Azadi Ka Amrit Mahotsav

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 906/2023-24 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "A PROSPECTIVE OBSERVATIONAL STUDY OF AUTONOMIC DYSFUNCTION IN CIRRHOSIS OF LIVER AND ITS CORRELATION WITH ELECTROCARDIOGRAPHY AND ECHOCARDIOGRAPHY".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.MUDIREDDY BINDU BHAVANI,

NAME OF THE GUIDE: DR.R.M.HONNUTAGI, PROFESSOR, DEPT. OF GENERAL MEDICINE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

Annexure II

SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR-586 103

RESEARCH INFORMED CONSENT FORM

TITLE OF THE PROJECT : A PROSPECTIVE OBSERVATIONAL STUDY OF AUTONOMIC DYSFUNCTION IN CIRRHOSIS OF LIVER AND ITS CORRELATION WITH ELECTROCARDIOGRAPHY AND ECHOCARDIOGRAPHY

PG GUIDE : Dr. R. M. HONNUTAGI

PG STUDENT : DR.MUDIREDDY BINDU BHAVANI

PURPOSE OF RESEARCH: -

BENEFITS: -

I understand that my participation in this study will help the investigator diagnose the disease better and help manage the disease.

PROCEDURE: -

I understand that relevant history will be taken, and I will undergo a detailed clinical examination, after which necessary investigations and treatment will be given.

RISK AND DISCOMFORTS: -

I understand that no risk is involved, and I will experience no pain during the procedures.

CONFIDENTIALITY: -

I understand that medical information produced by this study will become a part of my hospital records and will be subjected to the hospital's confidentiality and privacy regulations. Information of a sensitive personal nature will not be a part of the medical records but will be stored in the investigator's research file.

Suppose the data are used for publication in the medical literature or for teaching purposes. No names will be used in that case, and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand I may see the pictures and videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION: -

I understand that I may ask more questions about the study at any time Concerned. The researcher is available to answer my questions or

concerns. I will be informed of any significant new findings discovered during this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION: -

I understand that my participation is voluntary, and I may refuse to participate or withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that the researcher may terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange my continued care by my physician, if appropriate.

INJURY STATEMENT:-

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, and if such injury were reported promptly, medical treatment would be available, but no further compensation would be provided. I understand that my agreement to participate in this study does not waive my legal rights.

I have explained to (patient's / relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in the patient's language.

_____ _____

Investigator / P. G. Guide Date

I confirm that …….(Name of the PG guide / chief researcher) has explained the research, my study procedures, the possible risks and discomforts, and the benefits I may experience. I have read and understand this consent form. Therefore, I agree to consent to my participation as a subject in this research project.

Participant/guardian	Date
Witness to signature	Date

ANNEXURE III

OPC POISOINING CASE PROFORMA

PROFORMA

Name IP number

Age: Sex

Address: Occupation :

Date of Admission: Date of discharge:

Chief Complaints :

History of present illness:

Past history:

Personal History:

Physical Examination:

On Examination :

VITALS:

Temperature:

Pulse:

Respiratory rate:

Blood pressure:

GENERAL CONDITION:

Pallor:	Yes/ No
Icterus:	Yes/ No
Cyanosis:	Yes/No
Clubbing:	Yes/No
Lymphadenopathy:	Yes/No
Edema:	Yes/No

SYSTEMIC EXAMINATION:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

CENTRAL NERVOUS SYSTEM:

PER ABDOMEN EXAMINATION:

INVESTIGATIONS:

COMPLETE BLOOD COUNT:

Total count	CELLS/CMM
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Basophils	%
Monocytes	%
Haemoglobin	GM/Dl
Platelet count	LAKHS/CMM

PT with INR:

PT	
INR	

RBS:

RBS	MG/Dl

RENAL FUNCTION TEST:

BLOOD UREA	MG/Dl
SERUM CREATININE	MG/Dl
SERUM SODIUM	MEQ/Lt

SERUM POTASSIUM	MEQ/Lt

LIVER FUNCTION TEST :

TOTAL BILIRUBIN	MG/Dl
CONJUGATED BILIRUBIN	MG/Dl
UNCONJUGATED BILIRUBIN	MG/Dl
SGOT	UNITS/Lt
SGPT	UNITS/Lt
ALBUMIN	MG/Dl
ALP	UNITS/Lt

ECG :

ECHO:

Provisional Diagnosis:

ANNEXURE IV

MASTER CHART

S.NO	PATIENT NAME	IPNO	AGE	SEX	SUPINE PR	STANDING PR	VALSALVA	SUPINE BP	STANDING BP	HAND GRIP	ECG	ECHO	INTERPRETATION	CHILD PUGH	MELD
1	REVANASIDDA	98781	45	MALE	80 BPM	76 BPM	80 BPM	130/80 MMHG	110/80 MMHG	130/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 50%,MILD PAH,RA/RV DILATED,TYPE1 DDF	SUGG OF AUTO DYSF	B	14
2	VILAS	2E+05	30	MALE	86 BPM	90 BPM	86 BPM	110/70	90/60	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 50%,NO PAH.	SUGG OF AUTO DYSF	C	34
3	RAFEEQ	2E+05	36	MALE	98 BPM	100 BPM	96 BPM	140/80	140/80	130/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 60%,NO PAH.	NO EVIDENCE	B	9
4	LAKKAMMA	4E+05	55	FEMA	120	120 BPM	110 BPM	110/70	100/70	110/70 MMHG	SINUS TACHYCARDIA	CONC LVH,NORMAL LVEF 60%,TYPE 1	TYPE 1 DDF SUGGES	B	8
5	KRISHNAPPA	1E+05	58	MALE	82 BPM	86 BPM	80 BPM	110/80	100/70	100/70 MMHG	LOW VOLTAGE QRS CON	NORMAL LVEF 60%,TYPE 1 DDF,DEGEN	TYPE 1 DDF SUGGES	B	7
6	SHIVAJI	1E+05	45	MALE	120 BPM	110 BPM	120 BPM	110/70 MMHG	120 /70 MMHG	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	C	13
7	MADIWAL	1E+05	33	MALE	82 BPM	86 BPM	80 BPM	100/70	110/70	100/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH	NO EVIDENCE OF	B	12
8	ANNARAYA	99785	45	MALE	80 BPM	84 BPM	80 BPM	90/60	94/60	90/60 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA,NO PAH	NO EVIDENCE OF	C	26
9	SANTHOSH	964	18	MALE	110	114 BPM	110 BPM	90/60	100/70	90/60 MMHG	SINUS TACHYCARDIA	NORMAL LV EF 55%,NO RWMA,NO PAH	NO EVIDENCE OF	C	24
10	MAHANTAGOUDA	1E+05	49	MALE	70 BPM	74 BPM	70 BPM	110/70 MMHG	120/70 MMHG	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	19
11	SANTHOSH	3926	36	MALE	92 BPM	94 BPM	90 BPM	110/60	120/70	110/60 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 60%,NO RWMA,MILD	NO EVIDENCE OF	B	11
12	ABHIJIT	4555	39	MALE	110 BPM	100 BPM	110 BPM	100/70 MMHG	80/60 MMHG	100/70 MMHG	SINUS TACHYCARDIA	TACHYCARDIA,NORMAL LV EF 60%,NO RWMA,MILD PAH	POSTURAL DROP IN BP SUGG OF AUTO DYSF	B	29
13	MALLANNA	5338	28	MALE	78 BPM	80 BPM	78 BPM	110/70 MMHG	120/70 MMHG	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	12
14	VITTAL	6526	42	MALE	64 BPM	68 BPM	64 BPM	130/80 MMHG	140/80 MMHG	130/80MMHG	SINUS BRADYCARDIA	NORMAL LV EF 55%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	9
15	RAMAGOND	6828	60	MALE	88 BPM	80BPM	86 BPM	110/70	100/70	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL RESTING LVEF 60%,NO	DIAST DYFUN SUGG OF	C	26
16	RAJKUMAR	1E+05	43	MALE	82 BPM	76 BPM	82BPM	110/70	100/70	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA,NO	DIAST DYFUN SUGG OF	B	18
17	SHAWALI	2E+05	65	MALE	84 BPM	86 BPM	84 BPM	140/90 MMHG	150/90 MMHG	140/90 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	20
18	KAMAGONDA	3E+05	55	MALE	110 BPM	100 BPM	110 BPM	200/100MMH	180/100 MMHG	200/100MMHG	NORMAL SINUS RHYTHM	CONC LVH,NORMAL LVEF 55%,NO RWMA,NO PAH,TYPE1 DDF	DIAST DYFUN SUGG OF AUTO DYSF	C	14
19	SHRISHAIL	2E+05	51	MALE	76 BPM	70 BPM	76 BPM	140/90	120 /70	140/90 MMHG	T WAVE INVERSIONS IN	ISCHEMIC HEART DISEASE(INF WALL),EF	NO EVIDENCE OF	B	17
20	ANIL KUMAR	2E+05	35	MALE	90 BPM	84 BPM	90 BPM	90/60MMHG	80/60MMHG	90/60MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	39
21	SURESH	2E+05	45	MALE	96 BPM	98BPM	96BPM	100/70 MMHG	110/70M MHG	100/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	18
22	SHIVANGOUD	2E+05	45	MALE	110	100BPM	110 BPM	90/60	94/60MM	90/60 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO PAH,NO RWMA	NO EVIDENCE OF	C	34
23	MALLAPPA	5471	40	MALE	84BPM	86 BPM	84BPM	130/90 MMHG	134/90M MHG	130/90MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	28
24	AKSHAY	6010	28	MALE	110	120BPM	110BPM	120/80	130/90M	120/80MMHG	P MITRALE ?LT ATRIAL EN	LVEF 45-50%,NO RWMA,NO RA/RV	NO EVIDENCE OF	B	17
25	SABU	5353	56	MALE	80 BPM	74BPM	80BPM	90/60M MHG	84/60MM HG	90/60MMHG	LEFT BUNDLE BRANCH BL	NORMAL LVEF 55%,NO RWMA,DEGEN CHANGES,MILD PAH,TYPE1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	35
26	RAJASHEKAR	4633	52	MALE	86BPM	80 BPM	86BPM	90/60M	70/50MM	90/60MMHG	SINUS RHYTHM,LONG Q	NORMAL LVEF 60%,NO RWMA,NO	DIASTOLIC DYSF	C	44
27	KASHINATH	6032	56	MALE	84 BPM	80 BPM	84 BPM	110/60 MMHG	120/70 MMHG	110/70 MMHG	SINUS BRADYCARDIA	NORMAL LVEF 60%,NO RWMA,NO PAH,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	29
28	DYMAWWA	2E+05	65	FEMA	86 BPM	90BPM	86BPM	110/60	90/60MM	110/60MMHG	ATRIAL FLUTTER WITH 2	SEVERE PAH,RA/RV DILATED,LVEF	NO EVIDENCE OF	C	14
29	SIDDU	2E+05	38	MALE	130BPM	126BPM	130BPM	130/70	120/80M	130/70MMHG	SINUS TACHYCARDIA	TACHYCARDIA NOTED,NORMAL LVEF	NO EVIDENCE OF	B	9
30	KALAVATHI	3E+05	63	FEMALE	74 BPM	70BPM	76BPM	140/80 MMHG	130/80M MHG	140/80MMHG	LOW VOL COMPLEXES,Q	NORMAL LVEF 60%,NO RWMA,DEGEN CHANGES,TYPE 1DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	B	13
31	MAHADEVAPPA	50224	45	MALE	82BPM	84 BPM	82BPM	100/70 MMHG	90/60MM HG	100/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,GRADE 1 MR,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	18
32	ASHOK	4E+05	44	MALE	100BPM	104BPM	98BPM	100/70 MMHG	94/60MM HG	100/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO CLOT,NO PAH	NO EVIDENCE OF AUTO DYSF	C	10
33	SANJU	1E+05	38	MALE	72BPM	76BPM	72 BPM	110/70 MMHG	100/70M MHG	110/70MMHG	SINUS TACHYCARDIA	NORMAL LVEF 60%,NO RWMA,NO PAH,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	27
34	PRAKASH	84916	35	MALE	78BPM	80BPM	78BPM	100/60	90/60MM	100/60MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO	NO EVIDENCE OF	C	26
35	VIJAYAKUMAR	4E+05	36	MALE	82 BPM	84BPM	82BPM	100/70	90/60MM	100/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF55%,NO PAH	NO EVIDENCE OF	C	44
36	GOURISHANKAR	3E+05	24	MALE	80 BPM	84BPM	80BPM	130/80 MMHG	134/80M MHG	130/80MMHG	SINUS TACHYCARDIA	NORMAL LVEF 60%,NO PAH	NO EVIDENCE OF AUTO DYSF	C	30
37	BHAGESH	2E+05	35	MALE	98 BPM	94BPM	98BPM	90/60M	94/60MM	90/60MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO	NO EVIDENCE OF	C	23
38	MARUTI	90408	45	MALE	68BPM	72BPM	68 BPM	100/60	90/60	100/60MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO	NO EVIDENCE OF	C	26
39	VINOD	3E+05	30	MALE	90BPM	98BPM	90BPM	120/70 MMHG	126/80M MHG	120/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	13
40	MADIWAL	1E+05	33	MALE	82 BPM	86 BPM	80 BPM	100/70	110/70	100/70MMHG	NORMAL SINUS RHYTHM	NORMAL SYSTOLIC LVEF 60%,NO	NO EVIDENCE OF	C	12
41	DATTA	4E+05	42	MALE	100BPM	110 BPM	100 BPM	100/60	94/60	100/60 MMHG	NORMAL SINUS RHYTHM	NORMAL LV SYSTOLIC FUNCTION WITH	NO EVIDENCE OF	C	17
42	SHREEMANT	11929	45	MALE	96BPM	90BPM	98 BPM	110/70 MMHG	100/70 MMHG	110/70MMHG	NORMAL SINUS RHYTHM	NORMAL LV SYSTOLIC FUNCTTION WITH EF 55%,NO RWMA,NO PAH,TYPE	DIASTOLIC DYS SUGGESTIVE OF AUTO	B	10
43	PRAKASH	15421	44	MALE	86 BPM	80 BPM	86 BPM	120/70 MMHG	130/80 MMHG	120/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LV SYSTOLIC FUNCTION WITH EF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	24
44	SIDDARUDA	15654	50	MALE	92 BPM	88 BPM	92 BPM	110/80 MMHG	120 /70 MMHG	110/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA,RA/RV DILATED	NO EVIDENCE OF AUTO DYSF	B	9
45	AMASIDDA	16400	30	MALE	96 BPM	90 BPM	96 BPM	100/60 MMHG	110/70M MHG	100/60MMHG	NORMAL SINUS RHYTHM	NORMAL LV SYSTOLIC FUNCTION WITH EF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	17
46	SAGAR	16015	30	MALE	86BPM	80BPM	86BPM	110/60 MMHG	100/60M MHG	110/60 MMHG	NORMAL SINUS RHYTHM	NORMAL LV SYSTOLIC FUNCTTION WITH EF 55%,NO RWMA,NO PAH,TYPE	DIASTOLIC DYSF SUGGES OF AUTO	C	21
47	GIREMALLA	16401	48	MALE	78 BPM	74BPM	78BPM	120/80 MMHG	130/80M MHG	120/80MMHG	NORMAL SINUS RHYTHM	NORMAL LV SYSTOLIC FUNCTTION WITH EF 60%,NO RWMA,NO PAH,TYPE	DIASTOLIC DYS SUGGESTIVE OF AUTO	B	11
48	LAXMIBAI	17610	80	MALE	84BPM	80BPM	84BPM	110/80 MMHG	100/80M MHG	110/80MMHG	NORMAL SINUS RHYTHM	NORMAL LV SYSTOLIC FUNCTTION WITH EF 55%,NO RWMA,NO PAH,TYPE	DIASTOLIC DYS SUGGESTIVE OF AUTO	B	18
49	MITUNAKUMAR	16985	34	MALE	110 BPM	100BPM	110BPM	100/60 MMHG	80/60 MMHG	90/60 MMHG	? AV DISSOCIATION	SEVERE PAH,SEVERE TR,MILD MR EF 45%,NO RWMA	POSTURAL DROP IN BP SUGG OF AUTO DYSF	B	34
50	MAHESH	17620	32	MALE	94BPM	90BPM	94BPM	110/70 MMHG	120/70M MHG	110/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	C	14

51	SRINATH	17423	45	MALE	86 BPM	80BPM	86 BPM	120/80 MMHG	130/80M MHG	120/80MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	18
52	LAKSHMAN	17622	47	MALE	86 BPM	80 BPM	86 BPM	130/70 MMHG	140/80 MMHG	130/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	B	10
53	ABHIMANYU	18514	50	MALE	80 BPM	76 BPM	80 BPM	110/80 MMHG	120/80M MHG	110/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	B	11
54	SIDDARAM	18433	32	MALE	110BPM	100 BPM	110 BPM	120/80 MMHG	110/80 MMHG	120/80MMHG	SINUS TACHYCARDIA	TACHYCARDIA NOTED,NORMAL LVEF 55%,NO CLOT,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	C	9
55	SANGAPPA	18231	60	MALE	90 BPM	86BPM	90 BPM	160/90 MMHG	150/90 MMHG	160/90MMHG	LEFT VENTRICULAR HYPE	CONC LVH,NORMAL LVEF 55%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	17
56	SADASHIV	17987	45	MALE	78 BPM	80 BPM	78 BPM	120/70 MMHG	130/70 MMHG	120/70 MMHG	NORMAL SINUS RHYTHM	NORMAL SYSTOLIC LVEF 60%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	12
57	MAHESH	15984	49	MALE	70 BPM	68 BPM	70 BPM	100/70 MMHG	90/60 MMHG	100/70 MMHG	SINUS TACHYCARDIA	TACHYCARDIA,NORMAL LV EF 60%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	C	18
58	LAXMAN	1E+05	58	MALE	78 BPM	74BPM	78 BPM	130/80 MMHG	140/80 MMHG	130/80MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO PAH,TYPE1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	32
59	KISHAN	19366	57	MALE	94 BPM	92BPM	90 BPM	120/70 MMHG	110/80M MHG	120/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO CLOT,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	28
60	IRAPPA	95680	60	MALE	78 BPM	74BPM	78BPM	110/70 MMHG	100/70M MHG	110/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA,DEGEN CHANGES,MILD PAH,TYPE1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	30
61	GURURAJ	1E+05	35	MALE	90 BPM	86BPM	90BPM	100/60	94/60	100/60MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	12
62	CHANDAPPA	1E+05	45	MALE	86 BPM	80BPM	86BPM	110/80 MMHG	100/70 MMHG	110/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	B	15
63	SHRISHAIL	2E+05	35	MALE	80 BPM	74BPM	80BPM	100/70 MMHG	94/60 MMHG	100/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	14
64	YALLAPPA	2E+05	50	MALE	90BPM	86BPM	90BPM	110/70 MMHG	1O0/70 MMHG	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO CLOT,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	17
65	SANTHOSH	1E+05	42	MALE	86 BPM	80 BPM	86 BPM	100/60 MMHG	90/60 MMHG	100/60 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	9
66	UMAKANTH	3E+05	48	MALE	84 BPM	80 BPM	84 BPM	110/70 MMHG	100/70 MMHG	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO PAH,TYPE1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	11
67	CHANDRASHEKAR	3E+05	52	MALE	86 BPM	82BPM	86 BPM	120/70 MMHG	130/70 MMHG	120/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO CLOT,NO PAH	NO EVIDENCE OF AUTO DYSF	B	8
68	ASHOK	79217	41	MALE	90 BPM	86 BPM	90 BPM	100/60 MMHG	90/60 MMHG	100/60 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO PAH,TYPE1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	25
69	TUKARAM	1E+05	53	MALE	86 BPM	80 BPM	86 BPM	90/60 MMHG	80/60 MMHG	90/60 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO PAH,TYPE1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	B	30
70	SHANKAR	2E+05	45	MALE	88BPM	84BPM	88BPM	110/70 MMHG	120/70 MMHG	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	10
71	MALLAPPA	2E+05	48	MALE	70 BPM	74BPM	70 BPM	120/70 MMHG	130/80 MMHG	120/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	C	8
72	SADASHIV	3E+05	53	MALE	80 BPM	76 BPM	80 BPM	110/60 MMHG	90/60MM HG	110/60 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	21
73	PRABHUGOUDA	3E+05	45	MALE	100BPM	96BPM	100 BPM	150/90 MMHG	140/90M MHG	150/90MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	14
74	SUMITRA	2E+05	58	FEMALE	80BPM	74 BPM	80 BPM	110/70 MMHG	120 /70 MMHG	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA,NO PAH,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	12
75	NAGESH	3E+05	35	MALE	86 BPM	84BPM	86BPM	130/80 MMHG	120/80M MHG	130/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH,B/L PLEURAL EFFUSION	NO EVIDENCE OF AUTO DYSF	B	9
76	REVANASIDDA	3E+05	27	MALE	70 BPM	72BPM	70 BPM	100/60 MMHG	90/60 MMHG	100/60 MMHG	SINUS TACHYCARDIA	NORMAL LVEF 60%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	9
77	NAGAMMA	138	60	FEMALE	60BPM	64BPM	70 BPM	80/60M MHG	74/50MM HG	80/60MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	8
78	SANGAYYA	385	58	MALE	100BPM	96BPM	100 BPM	100/60 MMHG	80/60MM HG	100/60 MMHG	PREMATURE VENTRICUL	NORMAL LVEF 55%,NO RWMA,NO PAH,TYPE1 DDF	SUGG OF AUTO DYSF	C	11
79	DASTAGIRSAB	249	56	MALE	84 BPM	80 BPM	84 BPM	110/60 MMHG	100/60M MHG	110/60MMHG	SINUS TACHYCARDIA	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	9
80	LAXMAN	2E+05	75	MALE	90 BPM	84BPM	90 BPM	110/70 MMHG	100/60M MHG	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	9
81	PRAKASH	16799	31	MALE	98 BPM	94 BPM	98 BPM	120/70 MMHG	130/80 MMHG	120/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	B	14
82	TANAJI	3E+05	75	MALE	80 BPM	74 BPM	80 BPM	100/60 MMHG	90/60 MMHG	100/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA,DEGEN CHANGES,MILD PAH,TYPE1 DDF	SUGG OF AUTO DYSF	C	25
83	ABHIMANYU	14004	56	MALE	78 BPM	70BPM	78 BPM	120/70 MMHG	110/70 MMHG	120/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA,TYPE 1 DDF	SUGG OF AUTO DYSF	C	18
84	NARSAPPA	11192	38	MALE	90 BPM	86 BPM	90 BPM	110/80 MMHG	100/70M MHG	110/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	B	6
85	BASAVARAJ	3E+05	50	MALE	84 BPM	80 BPM	84 BPM	100/70 MMHG	90/60MM HG	100/70MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	C	18
86	SOMANNA	10465	40	MALE	78BPM	70 BPM	78 BPM	100/70 MMHG	94/60 MMHG	100/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	B	12
87	SIDDAPPA	9495	60	MALE	100BPM	96BPM	100 BPM	100/70 MMHG	110/70M MHG	100/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO PAH,TYPE1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	17
88	MOUNESH	8761	28	MALE	120 BPM	110 BPM	120 BPM	130/90 MMHG	140/80 MMHG	130/90MMHG	SINUS TACHYCARDIA	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	B	8
89	MUTTAPPA	20745	33	MALE	94BPM	90BPM	94BPM	110/80 MMHG	120/80M MHG	110/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO RWMA,NO PAH,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	11
90	JATEPPA	10139	70	MALE	100 BPM	96BPM	100BPM	140/100MMH	130/90M MHG	140/100MMHG	LOW VOLTAGE QRS CON	NORMAL LVEF 60%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	C	10
91	SANJAY	20156	43	MALE	80 BPM	74BPM	80 BPM	94/60 MMHG	90/60MM HG	94/60MMHG	LOW VOLTAGE QRS CON	NORMAL LVEF 55%,LA,LV DIALTED WITH PERICARDIAL EFFUSION	NO EVIDENCE OF AUTO DYSF	C	18
92	MOTILAL	71334	32	MALE	90 BPM	86 BPM	90BPM	120/70 MMHG	110/80 MMHG	120/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	29
93	SACHIN	3E+05	30	MALE	120 BPM	124BPM	120BPM	120/70 MMHG	130/80M MHG	120/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO RWMA,NO PAH,NO CLOT	NO EVIDENCE OF AUTO DYSF	C	24
94	RAJKUMAR	3E+05	35	MALE	80 BPM	84 BPM	80BPM	120/70 MMHG	110/80M MHG	120/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 55%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	B	9
95	CHANDRAKANTH	3E+05	61	MALE	72BPM	74BPM	72BPM	120/70 MMHG	130/80M MHG	120/70MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA,TYPE 1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	12
96	ASHOK	1E+05	29	MALE	72BPM	76 BPM	72BPM	130/70 MMHG	120/70M MHG	130/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	14
97	MAREPPA	2E+05	65	MALE	80 BPM	82BPM	80BPM	120/80 MMHG	130/70M MHG	120/80MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO PAH,TYPE1 DDF	DIASTOLIC DYS SUGGESTIVE OF AUTO	C	28
98	CHANNAPPA	2E+05	48	MALE	88BPM	84BPM	88BPM	130/80 MMHG	120/80M MHG	130/80 MMHG	NORMAL SINUS RHYTHM	NORMAL LVEF 60%,NO PAH,NO RWMA	NO EVIDENCE OF AUTO DYSF	C	30
99	SANJIV	24634	32	MALE	84BPM	80BPM	84BPM	110/70 MMHG	100/60M MHG	110/70 MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO RWMA,NO PAH	NO EVIDENCE OF AUTO DYSF	B	18
100	BASAVARAJ	3E+05	37	MALE	78BPM	74BPM	78BPM	100/60 MMHG	94/60MM HG	100/60MMHG	NORMAL SINUS RHYTHM	NORMAL LV EF 55%,NO PAH,TYPE1 DDF	DIAST DYFUN SUGG OF AUTO DYSF	C	32