Predictors of Recurrence of Syncope in Patients with Unexplained Syncope Undergoing Head Up Tilt Testing:

A Study Using Clinical, Hemodynamic and Echocardiographic Variables

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR DM (BRANCH ii, CARDIOLOGY) EXAMINATION, OF THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY TO BE HELD ON JULY/AUGUST 2011

BONAFIDE CERTIFICATE

This is to certify that the work presented in this dissertation titled "Predictors of Recurrence of Syncope in Patients with Unexplained Syncope Undergoing Head Up Tilt Testing: A Study Using Clinical, Hemodynamic and Echocardiographic Variables" done towards fulfillment of the requirements of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for the DM (Branch II) (Cardiology) examination to be conducted in July/August 2011, is a bonafide work of the candidate Dr. Anoop Mathew, post graduate student at the Department of Cardiology, Christian Medical College, Vellore, performed under my guidance and supervision. This dissertation has not submitted, fully or in part to any other Board or University.

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DECLARATION

I, Dr Anoop Mathew hereby declare that this dissertation entitled “Predictors of Recurrence of Syncope in Patients with Unexplained Syncope Undergoing Head Up Tilt Testing: A Study Using Clinical, Hemodynamic and Echocardiographic Variables” has been prepared by me under the direct supervision of Dr Bobby John, Professor, Department of Cardiology, Christian medical College, Vellore. This is being submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of regulations for the DM (Branch II) (Cardiology) examination to be conducted in July/August 2011.

This dissertation has not been submitted by me either in part or in full on any previous occasion to any University or Institution for the award of any degree or diploma

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Acknowledgement

This dissertation would not have been possible without the support and encouragement of many people.

I am indebted to Dr Bobby John, Professor, Department of Cardiology, Christian Medical College and Hospital (CMCH), Vellore for his valuable inputs and constant guidance throughout the study.

I thank Dr V Jacob Jose, Professor, Department of Cardiology, CMCH, Vellore, for help in conception of the research concept and for support throughout the study.

My special thanks to Dr Sunil Chandy, Profesor and Head, Department of Cardiology, CMCH, Vellore for the constant encouragement and support.

I thank Dr George Joseph, Professor, Department of Cardiology, CMCH, Vellore, for helping me recruit patients from his unit.

I thank Dr David Chase, Dr John Roshan and Dr John Jose for their valuable inputs.

I thank Dr Basu G for help with the statistics.

I thank Dr Sherin George for the constant support.

I am grateful to all the patients who gave consent to take part in this study, without whose cooperation this study would not have been possible.

CONTENTS

Predictors of Recurrence of Syncope in Patients with Unexplained Syncope Undergoing Head Up Tilt Testing:

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ABSTRACT

Background: Recurrent syncope is a common clinical problem. Head up tilt (HUT) testing reproduces reflex syncope in controlled laboratory settings. Echocardiographic monitoring of parameters including change in fractional shortening (FS) have been used to identify false positive responses on HUT testing. We assessed predictors of recurrent syncope in patients with unexplained recurrent syncope undergoing HUT testing.

Methods: This study is a prospective follow up of a cohort of patients undergoing HUT for unexplained recurrent syncope, with additional monitoring of echocardiographic left ventricular (LV) dimensions/ FS during HUT. The study was performed from Jan 2010 to Jan 2011.

Results: Sixty patients underwent HUT testing. Mean age was 46 ± 15 years and median duration of symptoms was 12 months (IQR 6 to 24 months). Thirty five (58.3%) patients had positive HUT response. Mean time to syncope was 31.5± 6.9 minutes. At the end of the tilt phase, FS in the HUT positive group increased significantly from baseline (32.4±0.68% to 37.5±0.64 %, $p< 0.001$), while FS did not change significantly in the HUT negative group. Ten (16.7%) patients had recurrent syncope on follow up. During HUT test, achieving a maximum heart rate of ≥ 108 beats per minute was predictive of recurrent syncope [OR 8.62 (1.002-73.84), p=0.049]

Conclusions: In patients with a positive response on HUT testing, there is a significant increase in LV FS during tilt as compared to those with a negative response. Patients who have recurrence of syncope on follow up tend to have higher peak heart rate attained during HUT. Hence peak heart rate attained during HUT testing can be used to identify patients at high risk of recurrence of syncope.

INTRODUCTION

Recurrent syncope is a commonly encountered clinical problem and remains a major diagnostic as well as therapeutic challenge in any clinical setting. Even though only a small percentage of patients experiencing syncope in the community present themselves to a hospital, syncope still accounts for 1% of presentations in European acute medical care settings (1)(2) (Figure 1). In order to prognosticate or risk-stratify patients presenting with syncope, two major aspects ought to be considered: first the risk of mortality and major adverse cardiac events and second the risk of recurrent syncope and consequent injury/ trauma. Syncope especially when recurrent has a pervasive detrimental influence on the quality of life of patients. Syncopal burden has inverse correlation with quality of life (3). Recurrent syncopal episodes are associated with orthopedic fractures and soft tissue injury in 12% of patients (4). While a detailed preliminary evaluation including an exhaustive clinical history, physical examination and a resting ECG will diagnose the underlying cause of syncope in 23 - 50% of subjects presenting with syncope, the remaining patients will require further risk stratification and additional diagnostic testing (5).

Reflex (neurally mediated) syncope, also known as neurocardiogenic syncope continues to be the leading cause of syncope in any clinical setting. In contemporary clinical practice, tilt testing remains the singular investigation that is available to the clinician, in order to demonstrate the propensity for reflex syncope in patients presenting with otherwise unexplained syncope. Head up tilt (HUT) testing reproduces reflex syncope in controlled laboratory settings and this has been shown to correlate with the patients original symptoms. Even in patients estimated to have a high risk of major adverse cardiac events and in those with clinical markers indicative of underlying brady-arrhythmia, HUT testing has been demonstrated to be of diagnostic value when

an underlying cardiovascular etiology has been ruled out by focused and comprehensive investigations. Since 1986 when HUT testing was originally introduced, the tilt protocol has evolved through multiple modifications. Studies have looked at the effect of modifying the duration of the pre-tilt supine phase, duration of tilt phase, angle to which the table is tilted and different drugs for pharmacological challenge, in order to arrive at an optimal protocol (6). HUT testing has become widely adopted in clinical practice, especially in the diagnostic evaluation of reflex syncope. However the absence of a "gold standard" for diagnosing syncope has made the sensitivity of HUT test questionable.

Apparently healthy subjects without any indication of cardiovascular disease may have a positive response to tilt testing (7) (8). The false positivity rate of HUT test is influenced both by patient characteristics including age and protocol characteristics including duration, angle of tilt and use of provocative drugs (9) (10). Studies comparing patho-physiological changes during positive tilt testing in healthy volunteers and patients presenting with reflex syncope have shown that the hemodynamic as well as humoral mechanisms leading to syncope are qualitatively and quantitatively different in these two groups. In patients presenting with reflex syncope, studies show that there is accelerated peripheral venous pooling as indicated by a more marked and exaggerated decline in left ventricular end diastolic dimension (LVEDD) as compared to HUT positive healthy volunteers. These patients were also demonstrated to have higher left ventricular (LV) contractility as indicated by values of fractional shortening (FS) associated with elevated levels of epinephrine (11).

Hence we postulated that, echocardiographic parameters such as FS and decrease in LVEDD may help in identifying a false positive group of patients in those undergoing HUT testing for unexplained syncope. We also postulated that these echocardiographic parameters

may help in assessing the risk of recurrence of syncope, thereby helping to direct therapy and interventions in order to limit recurrences and prevent physical injury and morbidity from syncope.

AIMS AND OBJECTIVES

Aim:

1. The aim of the study was to characterize the clinical, hemodynamic and echocardiographic variables including LV contractility during tilt in patients with unexplained recurrent syncope undergoing HUT test and to correlate the same with clinical outcomes

Objectives:

1. To determine the LV contractility during tilt in patients with unexplained recurrent syncope undergoing tilt testing by measuring the LV end diastolic and systolic dimensions and FS, using echocardiography during tilt
2. To determine if change in FS during HUT test will predict recurrence of syncope during follow up
3. To determine other significant predictors of recurrent syncope in the study population
4. To determine the predictors of positive response to HUT test

REVIEW OF LITERATURE

Syncope is a common presenting complaint in any clinical setting. Clinically syncope can be defined as a transient loss of consciousness caused by cerebral hypoperfusion, the hallmarks of which include abrupt onset, brief duration, and spontaneous complete recovery without residual neurological deficits. The term pre-syncope is commonly used in clinical parlance to depict a spell that is similar to the prodrome of syncope but which is not followed by loss of consciousness. Questions have been raised as to whether the patho-physiology of pre-syncope is same as that of syncope. In the 17th century John Hunter, a surgeon, documented the vasodepressor manifestations of syncope in a patient undergoing phlebotomy. In the 18th century, researchers including Foster identified vagally mediated cardio-inhibition as the causative mechanism underlying syncope. Lewis coined the term ´vasovagal syncope´ (12).

The prognosis of patients with syncope is not homogenous. Syncope can be benign. Less commonly syncope can be a harbinger of sudden cardiac death. Even though the cause is benign, recurrent syncopal episodes result in substantial morbidity. Syncope can cause injury. Syncope causes significant anxiety among subjects and their relatives, resulting in substantial functional limitation comparable to the level of debilitation seen in many chronic illnesses. Syncope poses a huge burden on the society in terms of medical, social, and economic impact. In the United States alone more than one million patients are worked up for syncope every year. Different registries have shown that that upto 5% of emergency department consultations and upto 6% of hospitalizations are for evaluation of syncope in Western populations (13). In a recent European series the median duration of hospitalization was 5.5 days (2). The most commonly encountered form of syncope is vasovagal syncope (common faint). It is also known as neurocardiogenic syncope or neurally mediated syncope. Proven as well as efficacious treatment for

neurocardiogenic syncope remains elusive. There are only limited therapeutic options for the management of recurrent of vasovagal syncope. Even though multiple drugs have been evaluated for preventing recurrence of syncope, the choice of therapy is mostly empirical and its efficacy is only suboptimal. Drug trials for this purpose have been mostly hampered by the sporadic as well as episodic nature of neurocardiogenic syncope and by the heterogeneity of the study population.

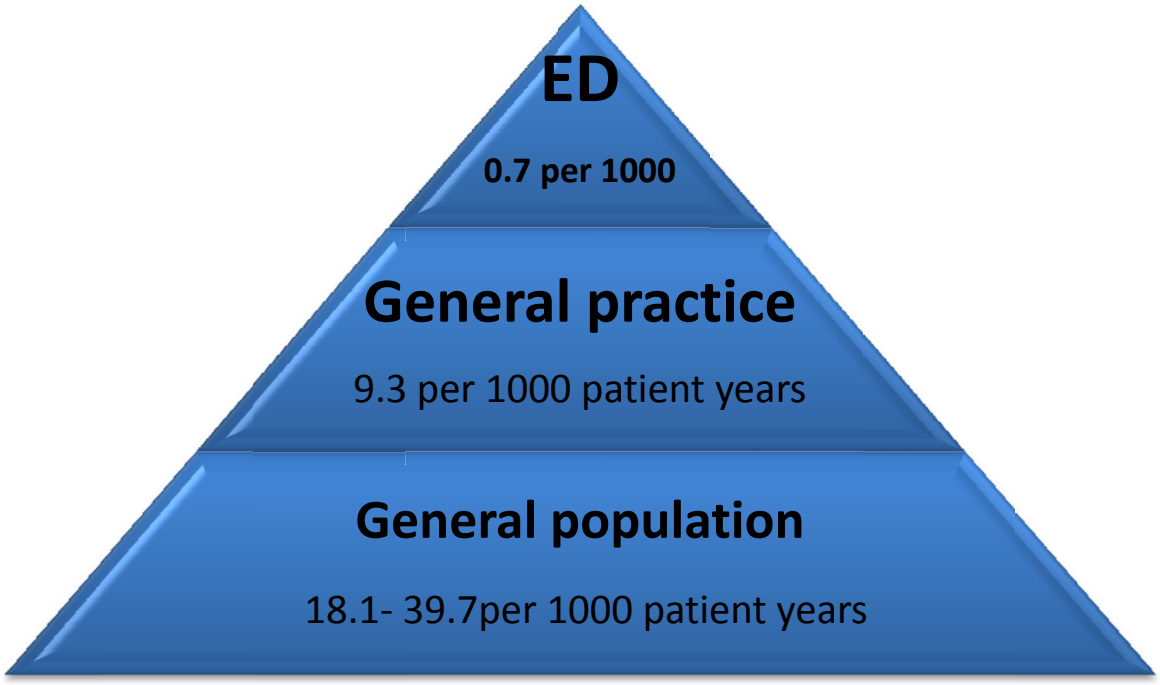


Figure 1.Syncope events/ visits per 1000 patient-years [data from (14)] ED Emergency department

An etiologic classification system for syncope has been proposed (15). Syncope is caused by global cerebral hypoperfusion. There are three main causes of transient low cardiac output which leads to syncope, as shown in table 1. The commonest underlying cause is a reflex mediated bradycardia which is also known as cardio-inhibitory type of reflex (neurally mediated) syncope. Secondly, cardiovascular causes also contribute to the etiology, including arrhythmia

and structural cardiac diseases. Yet another cause is inadequate venous return, which in turn may be due to dehydration or venous stagnation in the lower limbs.

Table 1. Etiological Classification of Syncope (15)
Reflex Syncope - Vasovagal or neurocardiogenic - Situational (e.g. cough, micturition) - Carotid Sinus Hypersensitivity - Syncope without obvious triggers or non-typical presentations
Syncope resulting from Orthostatic Hypotension - Medications causing Orthostatic Hypotension - Primary Autonomic Failure - Secondary Autonomic Failure - Volume Depletion
Cardiovascular Syncope - Arrhythmias: bradycardia and tachy-arrhythmias - Structural Cardiovascular Diseases: Valvular heart diseases, aortic dissection, acute myocardial infarction, hypertrophic cardiomyopathies, cardiac tumors, cardiac tamponade, coronary artery anomalies, pulmonary hypertension and pulmonary embolism

Pathophysiology of Neurocardiogenic Syncope

Blood pressure is regulated at the central nervous system (CNS) level by a complex mechanism. This includes CNS processing of both afferent and efferent signals. The

impulses relayed from the aortic and carotid sinus baroreceptors continually regulate the arterial blood pressure levels. These neural impulses are carried in the vagus nerve and glossopharyngeal nerve respectively and they relay to the CNS. These neural impulses are processed in the the nucleus tractus solitarius. This will result in inhibition of efferent sympathetic outflow and augmentation of the efferent vagal outflow (12). A fall in the arterial blood pressure leads to an augmentation of the sympathetic outflow and suppression of vagal output. Thus this neural reflex system modulates the balance between the sympathetic and parasympathetic system thereby regulating the blood pressure levels.

In addition to the above mentioned reflex system there are cardiopulmonary baroreceptors which are located in the ventricular walls and in the vasculature. These baroreceptors also regulate the sympathetic outflow (16). Ventricular stretch mediated by increased filling pressures is the main stimulus for these receptors. Based on the underlying pathogenesis, a classification system for neurocardiogenic syncope has been proposed. Neurocardiogenic syncope is classified as 'vasodepressor type' if hypotension occurs due to a loss of the vasoconstrictor sympathetic tone and 'Cardio-inhibitory' when bradycardia or asystole occurs. Neurocardiogenic syncope is classified as mixed if both these mechanisms contribute.

On assumption of erect posture a number of compensatory physiologic changes involving the sympathetic and parasympathetic reflex systems come into effect. When a patient assumes upright posture these is pooling of more than half a liter of blood in the lower extremities. Thus reduced venous return will decrease the filling pressure in the ventricle. Hence the mean arterial pressure as sensed by the aortic arch and carotid sinus drops (12). This would trigger a compensatory increase in the afferent neural activity; as a result there are increased sympathetic

impulses relayed to the cardiac and vascular structures. This cascade of events will ultimately result in peripheral vasoconstriction, increased inotropic and chronotropic response. As a result the mean arterial blood pressure is maintained and the subject does not faint.

While much research has gone into delineating the patho-physiology of neurally mediated syncope, not all aspects have been explained adequately thereby rendering the diagnosis and treatment of this common condition difficult. Hence therapies targeting different aspects of the purported patho-physiologic mechanism of syncope have not yielded optimal results. The most widely implicated patho-physiological mechanism causing neurally mediated syncope is the Bezold–Jarisch reflex (16,17). Bezold–Jarisch reflex is a neurocardiogenic reflex that is triggered by venous pooling in the lower limbs especially in dehydrated subjects. This results in decrease in ventricular volume and cardiac filling pressures. Compensatory baroreceptors reflex comes into play and resultant sympathetic discharge mediates increase in contractility of the left ventricle. Consequently mechanoreceptors that are activated by changes in wall tension located in the left ventricle wall are activated inappropriately. The C- fiber mediated afferent signals are relayed through the vagus nerve to the CNS. That mediates a reflex decrease in the sympathetic outflow to the vasculature and the cardiac structures, and inverse changes occur in parasympathetic activity. Marked vasodilatation occurs. This is accompanied by bradycardia and hypotension. These changes are the hallmarks of the loss of consciousness that characterize neurally mediated syncope. These changes are promptly reversed once the subject assumes supine posture. Thus we may conclude that neurally mediated syncope mainly takes place in predisposed patients due to excess lower limb venous pooling that leads to a sudden drop in venous return leading to inappropriate activation of LV mechanoreceptors thereby causing bradycardia and hypotension culminating in syncope.

Mechanoreceptors are present at multiple other sites including the urinary bladder, rectum, and respiratory organs. It has been postulated that abrupt activation of these receptors groups during micturition, defecation or coughing transmits afferent signals to the CNS thereby triggering a reflex mediated syncope. This is considered to be the maladaptive mechanism underlying situational syncope.

In humans Bezold–Jarisch reflex, has been implicated in the pathogenesis of reflex syncope largely based on indirect evidence. In patients with reflex syncope, during tilt, a surge in catecholamine levels has been demonstrated occurring just prior to loss of consciousness (18). However, exaggerated activation of the sympathetic system prior to syncope has not been demonstrated across studies in a consistent manner. In human studies, recording of neural impulses in peroneal nerve fibers using microelectrodes has demonstrated an increase in sympathetic neuronal traffic in the presyncopal phase, followed by a sudden withdrawal of sympathetic neuronal activity just prior to onset of syncope. Echocardiographic studies have demonstrated decrease in the LVEDD and end diastolic volume in syncopal subjects during tilt testing (19,20).

However neurally mediated syncope in humans, cannot be entirely attributed to the Bezold–Jarisch reflex mechanism. This point has been illustrated by the fact that a pure vasodepressor response without a drop in heart rate has been demonstrated during tilt induced neurocardiogenic syncope in post heart transplant patients (12). Neural reflex mechanism is effectively ruled out in this case as cardiac afferent and efferent pathways are denervated in a transplanted heart. This partly explains the lack of bradycardia in these patients. Hence multiple triggers beyond those described in the Bezold–Jarisch reflex are likely to be operative in neurally mediated syncope. However skeptics have argued that response may be just a type 3 (as per

VASIS classification) also known as pure vasodepressor response occurring in cardiac transplant recipients and not due to the surgical cardiac denervation alone (21)(22,23). EEG monitoring during HUT has shown increased activity in the left cerebral hemisphere in subjects developing syncopal response on tilt test. This lateralization occurs prior to the onset of loss of consciousness and accompanies the drop in heart rate and blood pressure and onset of symptoms. Hence the left cerebral hemisphere may be a part of the neural circuit that mediates vasovagal syncope (24).

Studies have indicated that multiple neurotransmitters and neuro-modulators influence the pathogenesis of neurally mediated syncope. These neurotransmitters include serotonin (25), adenosine, and opioids (26). Emotional stress may precipitate reflex syncope. The neural reflex mechanism underlying vasovagal syncope may be modulated by impulses from the hypothalamus and forebrain. Prior to vasodepressor syncope a surge in the levels of beta endorphins have been demonstrated (27). Opioid μ receptor antagonists have been demonstrated to augment the cardiac baroreflex mediated stimulation of sympathetic pathway (28). The μ receptor antagonist naloxone has not been successful in aborting syncope during HUT (29).

Not only central neurotransmitters but also peripheral neuronal triggering mechanisms other than those typified by the Bezold–Jarisch reflex may play a role in the pathogenesis of reflex syncope. Intravenously administered adenosine has been shown to trigger sympathetic afferent activity (30). This fact underlies the use of adenosine as a pharmacological challenge during tilt testing. However adenosine induced tilt test response has shown no correlation with the recurrence rate of syncope or with the mechanism of spontaneous syncope as demonstrated by studies using implantable loop recorders (31). Pharmacological challenge with isoproterenol has been shown to counter the decrease in LVEDD that occurs in patients with reflex syncope

during tilt testing (32). Thus a decrease in ventricular volume leading to triggering of LV mechanoreceptors may not an imperative step in the pathogenesis of reflex syncope. Also the hemodynamic responses preceding syncope are distinctly different in those with passive tilt induced syncopal response as compared to those with a positive response to tilt testing with isoproterenol pharmacological challenge (33). Just prior to the onset of loss of consciousness, patients in the isoproterenol challenged tilt test group had a higher heart rate and cardiac output as compared to patients with passive tilt induced syncopal response. This heterogeneity in the hemodynamic response in these two groups of patients suggests differences in the syncopal triggering mechanisms in these groups.

The sudden fall in peripheral vascular resistance that is one of the final steps in the pathophysiology of reflex syncope, has been investigated by many groups. One postulate is that the peripheral vasodilatation is a passive mechanism mediated by a withdrawal of sympathetic activity. However multiple studies have demonstrated the presence of a sympathetic mediated active vasodilatation in humans. This active skeletal muscle vasodilatation may be initiated by changes in sympathetic tone mediated by cholinergic vasodilator nerves or by release of nitric oxide (NO). Patients with positive response to HUT test have been shown to have excessive metabolism of NO (34). Also the presence of NO as a peripheral neurotransmitter has been demonstrated in skeletal muscles.

Thus it can be concluded that the Bezold–Jarisch reflex is one of the many causative pathways of reflex syncope.

History and Physical Examination

Reflex syncope can be precipitated by a number of triggers including prolonged standing, strenuous exertion on a hot day, emotional stress, sight of blood and excessive physical pain. Patient may complaint of prodromal symptoms including lethargy, giddiness, sweating, dimness of vision, heaviness of head and nausea. Transient clinical signs that may be demonstrated at this stage include facial pallor, dilatation of pupils and flushing. This prodromal phase may be very brief or may last up to a few minutes prior to the actual occurrence of loss of consciousness. Some patients successfully abort the syncopal episode by recognizing this prodromal phase and assuming a supine position or by employing certain "physical counter pressure maneuvers". However, a minority of patients especially elderly subjects do not have a prodrome preceding the syncopal episode hence they are at risk of physical injury resulting from abrupt loss of postural tone (35).

Syncope is characterized by short duration of loss of consciousness, which may last for up to five minutes. Elderly patients have a number of atypical features including lack of prodromal symptoms, longer duration of syncope and post spell confusion which may last up to ten minutes or rarely longer. Syncope on occasions can have atypical features including jerky seizure-like movements. Some researchers refer to this as "convulsive syncope" (36). When the patient regains consciousness, he/ she may experience lethargy or fatigue. Patient may appear pale and at times can have profuse sweating after the spell. Post spell confusion is in favor of seizure disorder. Recovery from syncope is characteristically abrupt, and without any residual neurological deficits. A simple questionnaire has been used to differentiate syncope from seizures with a high degree of sensitivity as well as specificity (37). Questions enumerating clinical history from this point score system have been included in our questionnaire.

A focused and exhaustive clinical evaluation should also include an electrocardiogram, and a detailed drug history to identify the use of any proarrhythmic drugs including the use of Class IA and IC anti-arrhythmic medications. If available, bystanders who witnessed the syncopal spell should be interviewed to identify etiological markers. Jerky limb movements and tonic posturing may be associated with both cardiac and neurological causes of syncope. Absence of a prodrome is a clinical marker of cardiac arrhythmia. Rarely absence of prodromal symptoms may be indicative of dysautonomia (35). Recurrent syncope presents sporadically. It is not clear why syncopal spells occur in clusters, interspaced by relatively long asymptomatic periods. Hence compliance with drugs and other therapy becomes an issue. Family history of sudden cardiac death is important to elicit. History of exertional syncope points to structural heart disease especially those with fixed cardiac output.

A detailed clinical examination including postural blood pressure response should be documented in every patient. This includes a cardiovascular and neurologic examination in all patients. The clinical examination may provide clues to the etiological diagnosis including the presence of LV dysfunction, pulmonary arterial hypertension, valvular heart disease, hypertrophic cardiomyopathy and other structural cardiac diseases. Carotid bruits point to underlying carotid artery stenosis. In the majority of patients, a detailed clinical history and physical examination has high efficacy in identifying the etiology of syncope, though the underlying pathology of syncope remains unidentified in about 40% of patients (38).

Investigating Syncope

In cases where there is an underlying conduction disturbance or arrhythmia, ECG provides vital clues to the diagnosis. A number of conditions like sick node dysfunction,

atrioventricular conduction blocks, presence of an accessory pathway, channelopathies including Long QT Syndrome and Brugada syndrome, causing syncope, may be diagnosed on the ECG. The ECG may suggest a diagnosis of arrhythmogenic right ventricular dysplasia (ARVD). Frequent ventricular ectopics or a short run of nonsustained ventricular tachycardia especially in patients with underlying structural cardiac disease increases the likelihood of an underlying arrhythmic cause of syncope. However since the underlying arrhythmic episode is usually sporadic the diagnostic yield of a 12 lead surface ECG for this purpose may be low.

A variety of options for ambulatory ECG monitoring is available to the clinician. The "gold standard" for diagnosing cardiac arrhythmia as the cause of syncope is ECG documentation of the arrhythmia coinciding with the patients' symptoms. The singular factor that determines the device selection and duration of ambulatory monitoring is frequency of symptoms. Holter monitoring is widely available and is suitable for spells that recur on a daily basis. When the spells recur on a monthly basis an event monitor may be prefered. An implantable loop recorder is suitable for those with very infrequent symptoms. Implantable recorders can be used for up to 14 months and permits the clinician to correlate the patient's symptoms with the underlying cardiac rhythm. Implantable loop recorders have a high diagnostic yield. These devices are able to diagnose up to 90% of patients presenting with unexplained recurrent syncope (39). The use of implantable loop recorders has revolutionized the management of syncope by providing insights into the pathophysiology of syncope in an individual patient and it is shown to be cost-effective (40).

Cardiac imaging including echocardiogram is indicated only in select patients with syncope. Echocardiogram is a indicated if underlying structural heart disease is suspected clinically (41). It may also be indicated if the clinical history, physical examination and ECG

have not yielded a diagnosis. Echocardiogram is an appropriate imaging technique to diagnose many underlying causes of syncope including valvular lesions, hypertrophic obstructive cardiomyopathy (HCM), pulmonary embolism, LV dysfunction and ARVD. When patients known to have ischemic heart disease or those at high risk for ischemic heart disease, present with unexplained syncope, further evaluation is indicated. When there is history of exertional syncope, exercise stress testing is indicated. During exercise stress testing, a hypotensive response or a failure of blood pressure to increase with exercise is of particular significance. This may point towards a diagnosis of HCM, high risk ischemic heart disease and autonomic failure. Exercise stress testing may also suggest the presence of catecholaminergic polymorphic ventricular tachycardia. A diagnosis of neurocardiogenic syncope or reflex syncope can be made in a patient presenting with a typical history in the absence of other plausible explanations for the syncopal episode (42). Further evaluation is indicated if the clinical features are atypical.

Syncope in the Patient with an Apparently Normal Initial Evaluation

After a detailed clinical evaluation and focused comprehensive investigations, if no cardiac disease is detected, it is likely that the syncopal episodes are not associated with increased mortality. In such a scenario the main objective of further evaluation is to identify the risk of physical injury and occupational risks from recurrence of syncope. For this purposes the AHA/ACCF has defined a “malignant episode of syncope” as a syncopal spell that occurs without any prodromal warning symptoms resulting in physical injury to self or causing property damage (41). Hence occupational groups like drivers and pilots presenting with syncope may require further detailed evaluation for medico-legal purposes. However deciding on further investigations, after an apparently normal initial evaluation, can be a difficult task as the yield of

many of these investigations are low. Even after the above mentioned investigations do not yield a diagnosis one should still actively consider alternative diagnoses like reflex syncope, carotid sinus hypersensitivity especially in elderly patients, arrhythmias including atrial and ventricular tachycardia.

The Tilt Table Test

Ever since it was introduced into clinical practice in 1986 by Kenny, et al, tilt testing has become the investigation of choice for evaluating patients presenting with unexplained recurrent syncope (7). HUT testing is primarily used for arriving at a diagnosis of reflex syncope, in a patient with a compatible history. Even though HUT testing has become widely acceptable as a feasible test for diagnosing reflex syncope, the clinical implications of a positive test response is still uncertain. The sensitivity, positive yield, and reproducibility of HUT testing reported in literature, vary widely (43), (44). The estimated sensitivity and specificity of HUT testing depends on patient factors as well as multiple factors in the tilt protocol (45). The sensitivity of HUT test is reported to vary from 26% to 80%, however these estimates may be inaccurate as there is no established "gold standard" for diagnosing neurocardiogenic syncope. The estimated specificity is about 90%; however it is well known that many healthy volunteers have a positive response on HUT (46). Even when patients have a positive response on tilt testing, the hemodynamic changes and underlying pathophysiological mechanism may not be the same as those occurring during a spontaneous syncopal spell (as recorded by implantable loop monitors) (47).

Table 2 illustrates the positive and negative results of HUT testing in adults. Table 3 illustrates the reproducibility of tilt table testing. A positive response to initial HUT testing may

modify future responses to HUT testing. The reproducibility of HUT test (when a second tilt test is repeated after a variable time period ranging from hours to weeks) is 80% to 95% for an initially negative test response. However the reproducibility is much lower for an initially positive HUT response (30% to 90%). This has implications for the use of HUT testing in assessing the response to therapy. Indications for HUT testing are enumerated in Table 4. In subjects presenting with unexplained recurrent syncope, if the initial work up is negative and the clinically estimated likelihood of the diagnosis being reflex syncope is high, little incremental diagnostic or prognostic information is obtained by performing a HUT test. The negative predictive value of tilt table test is low in an individual patient with a high pretest probability for reflex syncope. In patients presenting with unexplained syncope and physical injury resulting from syncope and for those in high risk occupations, further investigations are warranted when the tilt response is negative. A positive HUT test response does not predict the risk for recurrence of syncope in these patients.

Table 2. Passive Head Up Tilt Testing in Subjects with Unexplained Syncope

Study	Total patients	Positive result on HUT	Controls	Controls with positive HUT	Tilt angle	Tilt duration
	n	n (%)	N	n (%)	degrees	minutes
Kerry et al(7)	15	10 (67)	10	1(10)	40	60

Fitzpatrick et al(48)	71	53 (75)	27	2 (7)	60	60
Strasberg et al(49)	40	15 (38)	10	0	60	60
Raviele et al(50)	30	15 (50)	8	0	60	60
Abi Samra et al(51)	151	63 (42)	15	0	60	20
Almquist et al (52)	15	4 (27)	18	0	80	10
Grubb et al(53)	25	6 (24)	6	0	80	30
Pongiglione et al(54)	20	4 (20)	0	-	90	15
Shen et al(55)	111	35(32)	23	2 (9)	70	45

Figure 2. Head Up Tilt table testing © N Engl J Med 2005;352:1004-10.

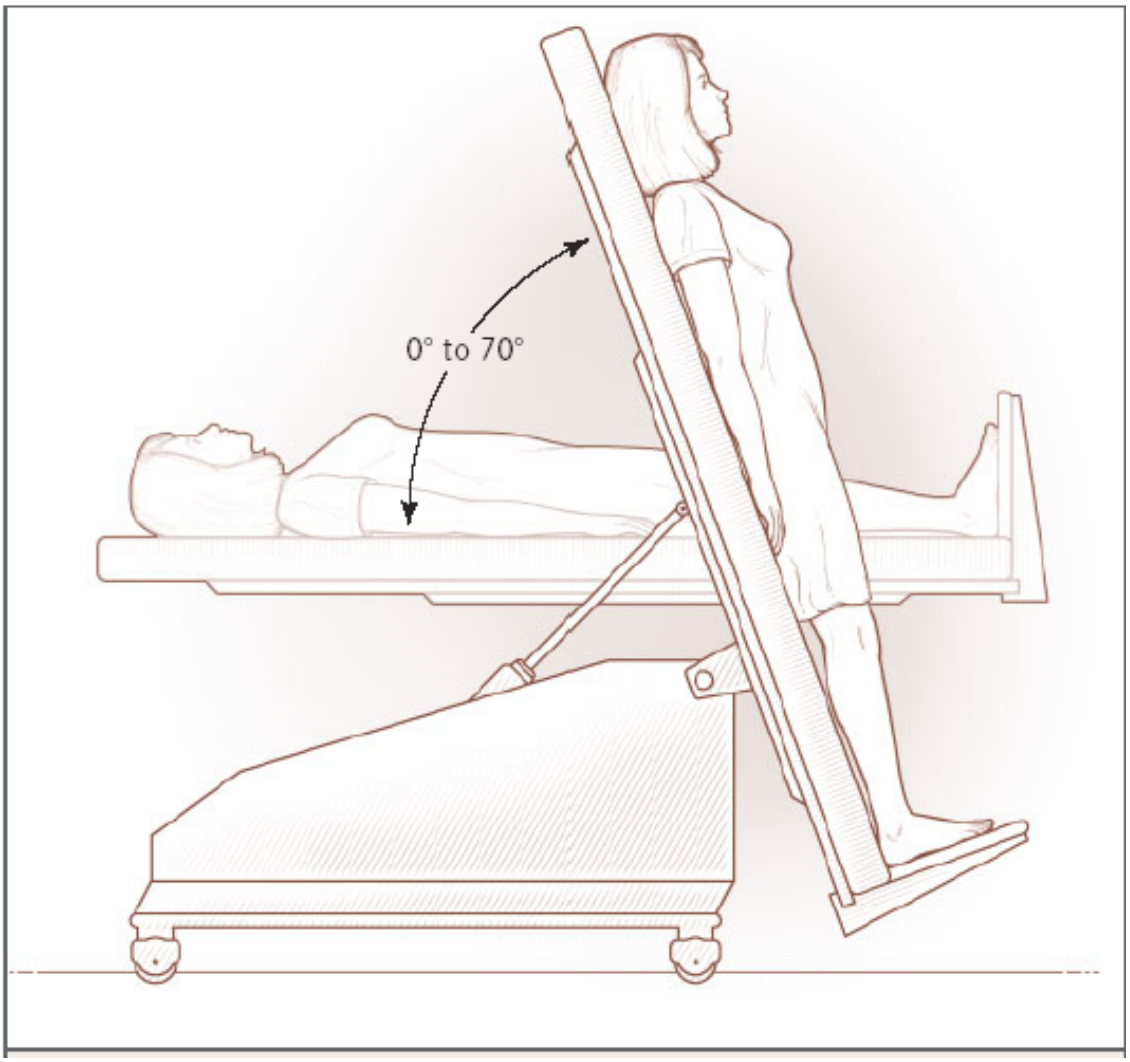


Table 3. **Reproducibility of the Initial Results of Tilt-Table Testing**

Study (Reference)	**Protocol for Tilt-Table Test**	**Time between Tests**	**Positive Reproducibility**	**Negative Reproducibility**
			n/n(%)	n/n(%)

de Buitleir et al (56)	80° tilt, 10-min duration	5 min	8/14 (57)	16/17 (94)
Brooks et al (57)	70° tilt, 25-min duration	1 d	11/30 (37)	45/56 (80)
Raviele et al (50)	60° tilt, 60-min duration	3 d	10/14 (71)	-
Fitzpatrick et al(48)	60° tilt, 60-min duration	-	24/31 (77)	-
Blanc et al(58)	60° tilt, 60-min duration	7 d	8/13 (62)	-
Fish et al(59)	Isoproterenol used	30 min	14–18/21 (67–86)	-
Chen et al (60)	Isoproterenol used	30 min	12/15 (80)	8/8 (100)
Grubb et al (61)	Isoproterenol used	5 d	13/14 (93)	6/7 (86)
Sheldon et al (62)	Isoproterenol used	2 wk	23/26 (88)	17/20 (85)
G. Foglia-Manzillo (63)	60º for up to 45 min. 400 µg sublingual spray nitroglycerin	13 days	12/33 (36)	-

Table 4. Indications for Head Up Tilt Testing (64)(15)
Definite indications Recurrent syncope in the absence of underlying cardiac disease or in the presence of organic cardiac disease, after cardiac causes of syncope has have been ruled out using appropriate investigations (Class I B) A single episode of unexplained syncope occurring in a high risk setting (occupational implications or trauma resulting from syncope) (Class I B) HUT testing is used for demonstrating the susceptibility to neurocardiogenic syncope to the patient (Class I C)
Possible indications Differentiation between reflex and orthostatic hypotension syncope (Class IIa C) Differentiation between syncope with jerking movements and seizure disorders (Class IIb C) Evaluation of repeated inexplicable falls (Class IIb C) Evaluation of patients with frequent syncope and psychiatric disease (Class IIb C) Evaluation of recurrent syncope in patients with neurological disorders like dysautonomia or peripheral neuropathy Evaluation of exertional syncope especially exercise stress testing does not reproduce the spell

Not indicated in

HUT testing is not recommended for evaluation of response to treatment (Class III B)

Isoproterenol HUT testing is contraindicated in patients with ischemic heart disease (Class III C)

Head-up tilt testing using sublingual nitroglycerin (NTG) challenge

HUT testing augmented by sublingual nitroglycerin was advocated by an Italian group (65) in 1995 by addition to the standard Westminster protocol (48)(7) of a NTG provocation phase. The nitroglycerin-head-up tilt is more sensitive than the non-medicated passive tilt test (65), and it is simpler and is increasingly used across the world. Thus, the test consisted of the 45 minute passive phase directly followed, if negative, by nitroglycerin administration with the test continuing for further 20 min. This protocol, although twice as sensitive as the passive tilt alone, is time-consuming and not well accepted by many cardiologists. From the available evidence, shortening the passive phase from 45 minutes to 20 minutes results in a significant reduction in the positive response rate. On the other hand, this was balanced by an increase in the positive responses during the provocation phase (Tables 5, 6). Thus, the final positivity rate of the test remained unchanged (6). A possible explanation is that nitroglycerin ensures the positive responses of the late passive phase of the test, besides including an additional number of drug-induced positive responses. In fact, assuming similar conditions of tilt angle, drug and its dose, the number of positive responses during the provocation phase of the test is always greater after a shortened rather than after a conventional passive phase (Tables 5, 6). Furthermore, the reduction of the passive phase to 20 minutes was accompanied by a decrease in the so-called ‘exaggerated

responses'. Finally, it was found that the specificity of the test was not affected by the duration of the passive phase (Table 7).

Stabilization phase

If invasive maneuvers are avoided before the test, 5 minutes are sufficient for the patient to achieve a stable physical condition.

Passive phase

Tilt angle

As previously shown (66), the best tilt angle is 60º

Passive phase duration

For all the above-mentioned reasons that the optimal duration of the passive phase is considered to be 20 minutes.

Drug Challenge/ provocation phase

Multiple studies have evaluated testing protocols (table 6) using sublingual crushed nitroglycerin tablets at a dosage of 300 µg dosage. However significant individual variation in pharmacokinetics resulted, including differences in the mucosal absorption rate depending on the amount of saliva available. This was an important consideration in older patients. In many subsequent studies (table 5), a protocol using 400 µg nitroglycerine oral spray has been reported. This protocol has many advantages including a higher dose of nitroglycerine used, better pharmacokinetics, ease of administration and reduced time to syncope. This protocol imparts better sensitivity to the tilt testing while maintaining comparable levels of specificity. Using sublingual nitroglycerin tablets, the time to syncope was demonstrated to be 7±8 minutes (65).

This interval was reduced to 5 ± 4 minutes using nitroglycerin in the spray form (67) ((68). This finding underlies a recommendation to shorten the duration of test by cutting down on the duration of the provocation phase from 20 to 15 minutes. This reduction in the duration of the provocation phase does not seem to affect the sensitivity of the test (6).

Interruption of tilt

The tilt phase is terminated and the patient made supine when the following end points occur: First, at the end of the drug challenge stage if the patient remains asymptomatic (test negative). Second, if there is loss of consciousness (test positive). The test is deemed positive whenever loss of consciousness (that is, the reproduction of the patients' presenting symptom of syncope) occurs in association with hypotension and/ or bradycardia, with sudden (<5 minutes) onset. This positive HUT response is considered to be analogous to the prototypical vasovagal syncope. Recommendations suggest that the tilt phase should be terminated just when syncope is initiated with loss of postural tone, rather than at the onset of hypotension. The response can be classified according to the VASIS recommendations. However for classification purposes the hemodynamic responses until the onset of syncope has to be taken into account. The subsequent bradycardia should not be used for classifying the response. The tilt may also be terminated if there is a tardy onset, prolonged (>5 minutes) hypotension. This is an indication of orthostatic intolerance and is associated with minor symptoms usually. The clinical significance of this particular response is still debated. This response at times can be documented in healthy subjects also. However in patients with orthostatic intolerance without syncope, autonomic nervous system dysfunction cannot be ruled out (21).

Diagnostic Criteria for Tilt Testing (15)

- In patients presenting with unexplained syncope, but without organic cardiac disease the induction of hypotension and/ or bradycardia with reproduction of syncope is diagnostic of reflex syncope
- In patients without organic cardiac disease the induction of progressive hypotension is diagnostic of orthostatic hypotension
- In patients without organic cardiac disease, the induction of reflex hypotension and/ or bradycardia without reproduction of syncope *may be* diagnostic of neurocardiogenic syncope
- In patients with structural heart disease, first tachy/bradyarrhythmias and alternative cardiovascular causes of syncope should be reasonably ruled out before interpreting the positive tilt test result as diagnostic
- If the subject has loss of consciousness without accompanying hypotension and/ or bradycardia, a diagnosis of psychogenic pseudosyncope may be considered

Table 5. ***Results of tilt table testing potentiated by nitroglycerin sublingual spray 400 µg***

Study, year	Patient nos	Passive phase Duration (min)	Passive phase positivity (%)	NTG phase positivity (%)	Total Positive response	Exaggerated responses
Natale, et al 1998(69)	33	20	4(12)	22(67)	26(78)	
Del Rosso, et al. 1998(70)	202	20	22(11)	119(59)	141 (70)	8 (4)
Del Rosso,	69	20	7 (10)	36 (52)	43 (62)	3 (4)

et al. 1999(71)						
Total passive phase 20 min	304	20	33 (11)	177(58)	210(69)	23 (8)
Bartoletti, 1999(67)	84	45	15(18)	28(33)	43 (51)	18(21)
Fogila Manzillo, et al. 1999(63)	48	45	9 (19)	25 (52)	34 (71)	2 (4)
Del Rosso, et al. 2000(68)	31	45	3 (10)	21 (68)	24(77)	2(6)
Total passive phase 45 min	163	45	27 (17)	74 (45)	101 (62)	22(13)

Table 6. Results of head-up tilt + use of nitroglycerin sublingual tablets 300 µg

Study, year	Patient number	Duration of passive phase	Passive phase positivity (%)	NTG phase positivity (%)	Total positive responses	Exaggerated responses
Raviele, et al. 2000(72)	71	20	9 (13)	26 (36)	35(51)	3(4)
Raviele, et al. 1995(65)	235	45	59 (25)	60 (26)	119(51)	33 (14)
Kurbaan, et al. 1999(73)	102	45	35 (34)	38 (37)	73 (72)	-
Total (only 55, 65)	337	45	94 (28)	98(29)	192 (57)	33 (14)

Table 7. Response to sublingual nitroglycerin challenge tilt table test in healthy controls

Study, year	Number of patients	Age (Mean± SD)	HUT protocol	Positive response (%)
Raviele, et al. 1995 (65)	35	54± 19	60º×45 + 20 min NTG 0.3 mg	2(6)
Aerts, et al. 1997 (74)	20	27± 4	70º×45 + 15 min ISDN 5 mg	6 (30)
Natale, et al. 1998 (69)	16	67± 9	70º× 20 + 15 min NTG 0.4 mg	2 (12)
Del Rosso, et al. 1998(70)	34	45±17	60º×20 + 25 min NTG spray 0.4 mg	2(6)
Ammirati, et al. 1998(75)	23	36 ± 12	60º×30 + 15 min ISDN 1.25 mg	0 (0)
Bartoletti, et al. 1999(67)	25	49 ± 17	60º×45 + 20 min NTG spray 0.4 mg	1(4)
Del rosso, et al. 2000(68)	47	52± 20	60º×20 + 20 min NTG spray 0.4 mg	2(4)
Raviele, et al. 2000(72)	30	44± 10	60º×20 + 20 min NTG 0.3 mg	3 (10)
Total	230			18(8)

Classification of vasovagal syncope by the Vasovagal Syncope International Study (VASIS) in 1992 provided insight into the different types of vasovagal responses observed during tilt-induced syncope (76). This classification system has been extended to tilt testing with pharmacological challenge (70) (73). Based on the hemodynamic changes preceding syncope the positive response to HUT testing can be categorized into different subgroups. This classification system is useful both as a research tool and for directing medical therapy and interventions. This

system may be of use in making the choice of permanent pacing versus drug therapy for the treatment of recurrent reflex syncope.

Table 8: Classification of reflex syncope induced by HUT table testing- the modified VASIS Classification (73) (76)	
Type	Classification
Type 1 or mixed	Heart rate during syncope ≥ 40 beats per minute (bpm) or falls to < 40 bpm for < 10 seconds ± asystole for < 3 seconds. BP always falls prior to heart rate.
Type 2A or Cardioinhibitory	Heart rate during syncope < 40 bpm for >10 seconds but asystole for > 3 seconds does not occur. BP falls prior to heart rate.
Type 2B or Cardioinhibitory with asystole	Asystole for > 3 seconds occurs. Systolic BP falls to < 80 mm Hg at or after rapid fall in heart rate.
Type 3 or Pure vasodepressor	Heart rate does not fall > 10% from its peak at syncope. Fall in BP alone precipitates syncopal response.
First exception — chronotropic incompetence. This subgroup of patients shows no tachycardia response during the tilt (i.e. maximum heart rate during tilt < 10% from the pre-tilt rate). However there is no hypotensive response. *Second exception — excessive heart rate increase*. These patients show an excessive heart rate rise both at the onset of the upright posture and during the entire duration of tilt, till prior to onset of syncope (i.e. heart rate greater than 130 bpm).	

Subsequently a classification system based on interpretation of the hemodynamic patterns during the pre-syncopal phase of the HUT test with and without drug challenge using sublingual nitroglycerin, has been proposed (21). First type is the classic vasovagal syncope pattern: in the

pre-syncopal period, subjects had a rapid and full compensatory reflex adaptation to the upright posture. This lead to a stabilization of the blood pressure levels until abrupt onset of the reflex syncope took place at the end of tilt. Second type is the dysautonomic (vasovagal) syncope pattern in which steady-state adaptation to upright posture does not take place. This mal-adaptation leads to a steady and progressive fall in the blood pressure levels till the occurrence of a typical vasovagal reaction. Third is the orthostatic intolerance pattern in which there was a progressive fall in the blood pressure levels, similar to that of the dysautonomic group, but this hypotensive response was not followed by vasovagal syncope.

Complications and contraindications of tilt table testing

Tilt table testing is generally considered to be safe. There has been no report of mortality during tilt testing. Few cases of ventricular tachycardia with the use of isoproterenol in patients with ischemic heart disease or sick sinus syndrome have been reported in literature (77). However, no life threatening adverse effects from the use of nitroglycerine have been reported. Minor adverse reactions are common with both these drugs. These include palpitations with the use of isoproterenol and headache with the use of nitroglycerine. Rarely arrhythmias including atrial fibrillation can be triggered during or following a positive HUT test. This usually is transient (78). Despite the low risk involved in tilt testing, resuscitation equipment including defibrillators should be readily available in the HUT room. Caution should be exercised in patients with known arrhythmias, HCM, aortic stenosis and LV systolic dysfunction.

False Positive HUT Test and Use of Echocardiography during HUT test

Echocardiographic studies have provided insight into the pathogenesis of syncope during tilt testing. The loss of consciousness due to reflex syncope has been by convention attributed to the activation of LV mechanoreceptors, which in turn trigger the Bezold-Jarisch reflex. Peripheral venous pooling, sympathetic nervous system activation, as well as hypercontractility of a relatively empty left ventricle are the other potential triggers involved in this reflex pathway that mediates reflex syncope. Indirect evidence for the role of LV mechanoreceptors in pathogenesis of reflex syncope in humans is largely derived from studies demonstrating increased LV FS and decreased LV volumes in subjects with syncope and positive tilt testing (19). Studies using echocardiography during tilt testing show an accelerated rate of reduction of end-diastolic volume index as well as significant decrease in stroke volume index and ejection fraction in subjects presenting with reflex syncope as compared to healthy volunteers. This phenomenon is due to redistribution of blood to the peripheral venous system during tilt and an early parasympathetic effect on LV contractility (79).

The incidence of false positive responses has been demonstrated to be a problem inherent in HUT test. This has led many investigators to opine that tilt testing should only be used to confirm a clinically based diagnosis (10).

Even though tilt induced syncopal response can occur in healthy volunteers (false positive), the hemodynamic as well as humoral changes that accompany this response are qualitatively distinct from those of HUT positive patients who complaint of syncopal spells during normal daily activities (true positive). These distinct hemodynamic and humoral alterations are enumerated below (11).

a) Time to Syncope:

This was about twice as long in HUT positive healthy volunteers as compared to HUT positive patients with neurocardiogenic syncope.

b) Time to drop in blood pressure:

The true positives had a drop in blood pressure as early as two minutes into the test accompanied, at the same time, by tachycardia, while in HUT positive healthy controls (false positive cases), these alterations were absent till about two minutes before the onset of symptoms. The normal controls did not demonstrate either of these characteristics.

c) LV end-diastolic dimension:

The pattern of peripheral venous pooling in the three groups (true positives, false positives and true negatives) was indirectly estimated by analyzing changes in LVEDD. During tilt, both HUT positive groups (true positives and false positives) exhibited a progressive decline in LVEDD, significantly different from pre-tilt LVEDD value, while the HUT negative subjects (true negatives) demonstrated an initial decline that promptly stabilized (11)(19). There is more rapid and exaggerated peripheral pooling of intravascular volume in patients presenting with neurocardiogenic syncope. The hypotensive response and the abrupt reduction in peripheral venous return during HUT are indicative of an abnormality in vascular control. Studies have demonstrated impaired vasoconstrictor response in patients with neurally mediated syncope during HUT (80) and during dynamic leg exercise (81)(82).

d) Fractional shortening:

In patients with true positive reflex syncope, it has been demonstrated that FS assessed by echocardiography, significantly increases throughout the HUT test to become, at 2 min before the end of HUT, statistically different from the false positive and true negative groups (11).

e) Epinephrine levels:

During HUT, patients with reflex syncope had a sixfold increase in the epinephrine levels as compared to the baseline value, accompanying the bradycardia and hypotension. This increase in epinephrine level was significantly more than the level of increase seen in patients with false positive HUT results (11).

Monitoring of select echocardiographic parameters like LVEDD and FS to enhance the specificity and sensitivity of HUT testing for the diagnosis of neurally mediated syncope has been proposed (11). However this awaits controlled trials with larger numbers of patients. The population of HUT positive patients is heterogenous when classified in terms of LV contractility change during HUT test. It has been shown that LV hyper-contractility preceding syncope exists in about 50% of HUT positive patients (83). In the remaining patients without increase in LV contractility prior to the syncope, it could be that a mechanism other than Bezold Jarisch reflex may be underlying the syncope. Or else it could be that this group of patients may be false positive cases, behaving like the HUT positive normal volunteers in whom LV contractility does not increase before syncope.

No correlation has been made between monitoring parameters like LVEDD and FS during HUT test and clinical outcomes. Moreover the findings of increased FS during HUT have

been questioned in some earlier studies (84). The use of echocardiography during HUT testing has been largely limited to research settings.

We may conclude that the use of echocardiographic monitoring of select parameters during HUT provides valuable insight into the pathogenesis of neurally mediated syncope and these findings may have major clinical and prognostic implications.

Electrophysiological study in Unexplained Syncope

In patients presenting with syncope of unknown cause, recent studies show that positive yield with electrophysiology study (EPS) occur mainly in the subgroup of patients with structural cardiac disease (85). The ability of EPS to arrive at an etiological diagnosis for syncope is determined by the pre-test probability of the disease as well as the protocol used for the study. The advent of efficacious non-invasive electrocardiographic monitoring including prolonged monitoring has increased the diagnostic yield and in turn has diminished the prominence of EPS as a diagnostic test. In those presenting with syncope and bi-fascicular block, the high degree AV block may be transient. Hence it may not be picked up on routine 24 hour ambulatory Holter monitoring and, hence prolonged period of monitoring may be required to document it by ECG (86). In those subjects presenting with syncope associated with bi-fascicular block, an EPS has high sensitivity for diagnosing intermittent high degree AV block. However the negative predictive value of EPS is limited while evaluating intermittent AV block as the etiology of loss of consciousness.

EPS may help in risk stratifying syncope. The induction of a sustained ventricular tachycardia and presence of severe LV systolic dysfunction can predict a life-threatening syncopal episode. The nonexistence of these adverse prognostic indicators in turn predicts a

more favorable outcome. It can be concluded that EPS with programmed electrical stimulation is a test with high diagnostic and prognosticating value in those presenting with unexplained recurrent syncope in the presence of coronary artery disease and markedly depressed cardiac function. However the diagnostic efficacy of EPS is questionable in patients with dilated cardiomyopathy (4).

The current ESC guidelines suggest the following: In patients presenting with syncope in the presence of concomitant ischemic heart disease, EPS is warranted when the initial work up indicates an underlying arrhythmia (15). In patients presenting with syncope and underlying bundle branch block, EPS is to be performed when noninvasive investigations have not yielded an etiological diagnosis. In patients with syncope preceded by abrupt onset short duration palpitations, EPS can be considered when noninvasive tests have not yielded the correct diagnosis. In patients with high risk occupations and in patients with a diagnosis of Brugada syndrome, ARVD or HCM presenting with syncope, EPS may be performed in a select subgroup of cases (15).

Therapeutic Options in Reflex Syncope:

Counter-pressure maneuvers

With the paucity of data on the effectiveness of pharmacological data, non-pharmacological therapy, including physical counter-pressure maneuvers are emerging as the preferred treatment of reflex syncope. Recent studies have shown that isometric physical counter-pressure maneuvers involving the legs including leg crossing, or of the arms including hand grip and arm tensing, may successfully counter the hypotensive phase of an impending neurally mediated syncope by inducing a significant BP blood pressure rise. This would allow

the subject to abort or delay the frank syncopal spell in many cases, thereby preventing injury (87) (88).

Tilt training exercises

Tilt training involves the patient assuming an enforced period of upright posture, at a prescribed angle. The duration of tilt is progressively increased till the prescribed time period is reached. In highly motivated young patients with recurrent syncope and/ or presyncope triggered by orthostatic challenge, tilt training may reduce or prevent the recurrence of syncope (89). The main drawback of tilt training is the poor long term compliance as this requires high patient motivation. Many studies including randomized controlled trials have shown that tilt training may not be efficacious in preventing recurrence of syncope in patients undergoing repeat tilt table testing (90).

Drug therapy

Since Bezold- Jarisch reflex is known to be the major underlying pathophysiological mechanism of reflex syncope, the use of beta blockers may decrease the ventricular mechanoreceptor activation, by virtue of their negative inotropic effect in neurally mediated syncope. However the outcomes of multiple randomized control trials have not lent credence to this plausible hypothesis. The use of beta blockers may not be optimal in subtypes of reflex syncope in which mechanisms other than increased sympathetic activation are operational. Beta blockers may aggravate the bradycardia in carotid hypersensitivity syndrome. Majority of randomized trials with long term follow up have failed to prove the effectiveness of beta blockers in preventing recurrent syncope (91)(92).

Alpha receptor agonists including etilefrine and midodrine have been used in the management of neurally mediated syncope. Peripheral blood pooling due to failure to achieve adequate vasoconstriction of the peripheral blood vessels underlie many cases of neurally mediated syncope. Etilefrine, an alpha agonist vasoconstrictor, at a dose of 25 mg twice daily failed to reduce the frequency or time to recurrent syncope in a randomized clinical trial as compared to placebo (93). The major drawback with midodrine is the mulpile and frequent dosing. This limits long-term compliance in most patients (94). Alpha agonists may cause urinary retention, hence caution to be exercised in elderly male patients. Based on available data it may be concluded that long term drug treatment with alpha-agonists alone may not be of benefit in neurally mediated syncope. Also long-term becomes a problem in patients with sporadic symptoms.

Paroxetine has been shown to be efficacious in the management of frequent recurrent syncopal episodes (95). Tough fludrocortisone is widely used there are no randomized trials in adults which show fludrocortisone to be efficacious.

Dual Chamber pacing with rate drop algorithm

Studies have yielded conflicting results on the efficacy of cardiac pacing in patients with recurrent syncope (96) (97). A recent meta-analysis has shown a non-significant 17% reduction in the recurrence of syncope by pooling data from multiple double-blind trials. There was an 84% reduction in those trials where the control group did not have a pacemaker implantation, probably contributed to by a placebo effect (98). Different explanations for the suboptimal results with pacing have been offered. The most plausible one is that cardiac pacing may affect the cardio-inhibitory component of the vasovagal reflex; however pacing has no impact on the vasodepressor element of syncope.

MATERIALS AND METHODS

Study design:

This study is a prospective follow up of a cohort of patients undergoing tilt table testing for unexplained recurrent syncope in the absence of structural heart disease, with additional monitoring of echocardiographic LV dimensions/ FS during tilt table testing. The study was performed during a 13 month period from Jan 2010 to Jan 2011.

Setting:

The study was conducted at the department of Cardiology, Christian Medical College Hospital, Vellore. Patients were recruited from the Cardiology outpatient department as well as the Electrophysiology outpatient department. The patients were followed up till the end of the study period. Consecutive patients referred for HUT test were enrolled provided they met the inclusion and exclusion criteria. Oral questionnaire was administered by one of the investigators prior to the tilt test in all the cases. Patients were followed up during review consults. If the patient was not able to return for a review consult, then telephonic interview and follow up was performed.

Subjects:

Inclusion Criteria:

1. At least two syncopal episodes, with minimum one episode in the last one year that remained unexplained in spite of a detailed history, comprehensive physical examination, 12-lead ECG and echocardiography. 24 hour ambulatory Holter recording and electrophysiology study were performed if indicated. Neurology consultation was obtained if indicated.

2. Normal heart structure and function by echocardiographic criteria.

Exclusion Criteria:

1. Technically inadequate echocardiographic images/ window
2. History of usage of drugs known to cause orthostatic hypotension at the time of tilt testing. For those patients on beta blockers, beta blockers needed to be interrupted two days prior to tilt table testing.

Tilt Table Testing Protocol:

After obtaining documentary informed consent from all participants in the study, the tilt table testing was performed in a quite dedicated room. The patient was fasting for 4 hours prior to the test. All medications that could interfere with tilt table testing including diuretics, vasodilators and beta blockers were withheld for at least 48 hours prior to the test. The HUT table testing was performed by using an electrically controlled tilt table with a foot board for weight bearing (Ausmedic supplies, Australia) and three safety straps across the bed to hold the patient in case of loss of consciousness. The heart rate was continuously monitored using a 3-lead ECG monitor. Radial intra-arterial blood pressure monitoring was performed during the test. Electrocardiographic and BP data were continuously displayed on a monitor (Hewlett Packard).

We used a modification of the "Italian Protocol" with a longer stabilization phase and longer provocative phase.

Stabilization phase: The tilt was performed only after an initial observation period in the supine posture for 20 minutes.

Passive phase: 20 minutes of passive tilt at 60 degree tilt

Provocation phase: If there were no hypotension and/or bradycardia in the passive phase, sublingual spray of nitroglycerin 400 µg was administered at 60 degree tilt and patient was monitored for further 20 minutes

The test was interrupted, and the patient was brought down to the supine position in the following situations:

(1) Completion of the schedule in the absence of symptoms (test negative).

(2) Syncope (test positive).

(3) Progressive, prolonged (>5 minutes) orthostatic hypotension associated with minor symptoms (exaggerated response)

The test was considered positive whenever syncope (that is, the reproduction of the patient's original symptoms) occurred in association with hypotension, bradycardia or both, with rapid (<5 minutes) onset. For the categorization of the type of response the patterns of hemodynamic changes until that moment (but not bradycardia occurring after the onset of syncope (71)) were considered.

The patients were observed for a minimum period of 20 minutes after the test and longer if they continued to be symptomatic. All positive responses were classified according to the modified VASIS classification (21). An "exaggerated response" to nitroglycerine spray was interpreted as a negative response. This response was considered to be due to the pharmacological effects of nitrates. This response was identified by the progressive and slow (> 5 minutes) development of decrease in systolic blood pressure with associated compensatory tachycardia or only minimal bradycardia.

Echocardiographic analysis:

Two-dimensional echocardiography was performed using an Acuson ultrasonography system. An M mode image from a standard para-sternal short-axis view at the level of the LV papillary muscles was recorded in the supine resting stage, 1 minute after initiation of HUT, at 5,10, 20 minutes, at 1 minute post nitrate administration, 25,30, 35 and 40 minutes and once at the onset of symptoms if any. LVEDD and left ventricular end-systolic dimension (LVESD) were determined using M-mode echocardiography. Each value obtained by averaging two consecutive heart beats. The corresponding blood pressure and heart rate during these echocardiographic recording were also recorded. FS was later calculated using the formula:

$$\text{Fractional Shortening, FS} = \frac{[\text{LVEDD-LVESD}]}{\text{LVEDD}} \times 100$$

FS Change = FS at end tilt phase of HUT test- Baseline FS

FS Slope = FS Change/ Time duration from start of tilt to last recording of FS at the end of the tilt phase

One investigator and another health professional were present during the HUT test.

Follow up:

Follow up was done during review consults. If the patient is not able to return for a review consult, then telephonic interview and follow up was performed. Data regarding recurrent of syncope was collected during the follow up interview. Data was also collected detailing the medications taken and if tilt training was performed as advised. Compliance with exercise and drugs were also noted.

Sample Size Calculation:

Assuming a syncope recurrence rate of 40% in patients with HUT positivity and increased FS and 10% recurrence rate in patients with HUT positivity and no increase in FS we needed to recruit 32 patients in each sample to demonstrate a 30% difference in proportion of patients with recurrent syncope between the two groups. α error was taken as 5%. Power (1-β) was taken as 80%.

Statistical Analysis:

Data was stored and analyzed using SPSS version 17 (SPSS Inc. Chicago, IL, USA). Continuous variables are expressed as mean ± SD for normally distributed variables and median (inter-quartile range) for not normally distributed variables. Categorical variables expressed as number (percentage). Continuous variables were examined for normality of distribution using the Kolmogorov-Smirnov test when sample size was greater than 50 and Shapiro-Wilk test for less than 50. Differences in frequency of continuous variable were analyzed using independent sample student's *t*-test for normally distributed variables. For not normally distributed continuous variables a non-parametric Mann Whitney test was used. For discrete variables, Chi square statistics (or Fisher`s exact test if applicable for a cell count less than 5) was used. Paired sample *t*-test was used for comparing the change from baseline in parameters monitored during HUT test at different time points. We used binary logistic regression analysis to identify potential predictors of positive response on HUT test and predictors of recurrence of syncope on follow up. All parameters which showed a p value < 0.1 during the initial analysis were included in the binary logistic regression analysis. A p- value of < 0.05 was considered statistically significant.

RESULTS

Between January 2010 and January 2011, a total of 116 patients, presenting with unexplained syncope and undergoing tilt table testing, at our institution were evaluated for the study. Of these, 45 patients were not enrolled based on inclusion/ exclusion criteria. The major reasons for non-inclusion were presence of only one episode of syncope prior to tilt table testing and poor echocardiographic window. Eleven patients did not give consent to take part in the study. Sixty patients underwent tilt table testing with additional monitoring of select echocardiographic parameters (Figure 3).

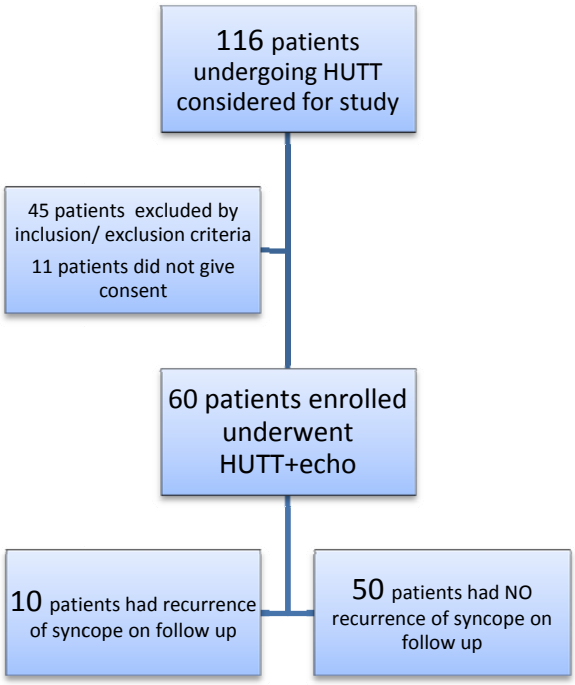


Figure 3. Diagram of patient flow through the study

Baseline Characteristics

The baseline characteristics of the cohort of patients are mentioned in tables 9.1 and 9.2. The mean age was 46 ± 15 years with a majority being males (63.3%). The baseline rhythm was

sinus in all the patients with a baseline heart rate of 79±14 beats per minute. The median number of episodes of syncope prior to HUT testing was 2 (inter-quartile range 2 to 5 episodes). The median duration of symptoms was 12 months (inter-quartile range 6 to 24 months). The findings regarding clinical history are also enumerated in table 9.2. Few features atypical of reflex syncope were found in the cohort, including history of exertional syncope in 8.3%, convulsive syncope in 5%, post spell confusion in 3.3% and palpitations preceding the syncopal episode in 15% of patients.

Table 9.1 Baseline Characteristics (Continuous Variables)	
Variable	**Median (inter-quartile range)**
Number of episodes of syncope prior to HUT test	2 (2-5)
Duration of symptoms (months)	12 (6-24)
Duration between last syncopal episode and HUT test (weeks)	4.14 (1.17-8.92)

Table 9.2 Baseline Characteristics (Categorical Variables)	
Variable	**Number (%) n=60**
Sex (male)	38 (63.3)
History of exertional syncope ever present: yes	5 (8.3)
At times wake up with a cut tongue after syncope: yes	0 (0)
Ever had a sense of aura preceding spells : yes	0 (0)
Emotional stress ever associated with spells: yes	13 (21.7)
Ever noted to have head turning during spells: yes	0 (0)
Tonic posturing or jerking movements during spells: yes	3 (5)

Ever been confused after a spell: yes	2 (3.3)
Ever had lightheaded spells: yes	38 (63.3)
Ever sweat before spells: yes	11 (18.3)
Prolonged standing associated with spells: yes	15 (25)
Posture at onset of syncope: Sitting	22 (36.7)
Posture at onset of syncope: Standing	38 (63.3)
Palpitations preceding syncope: yes	9 (15)

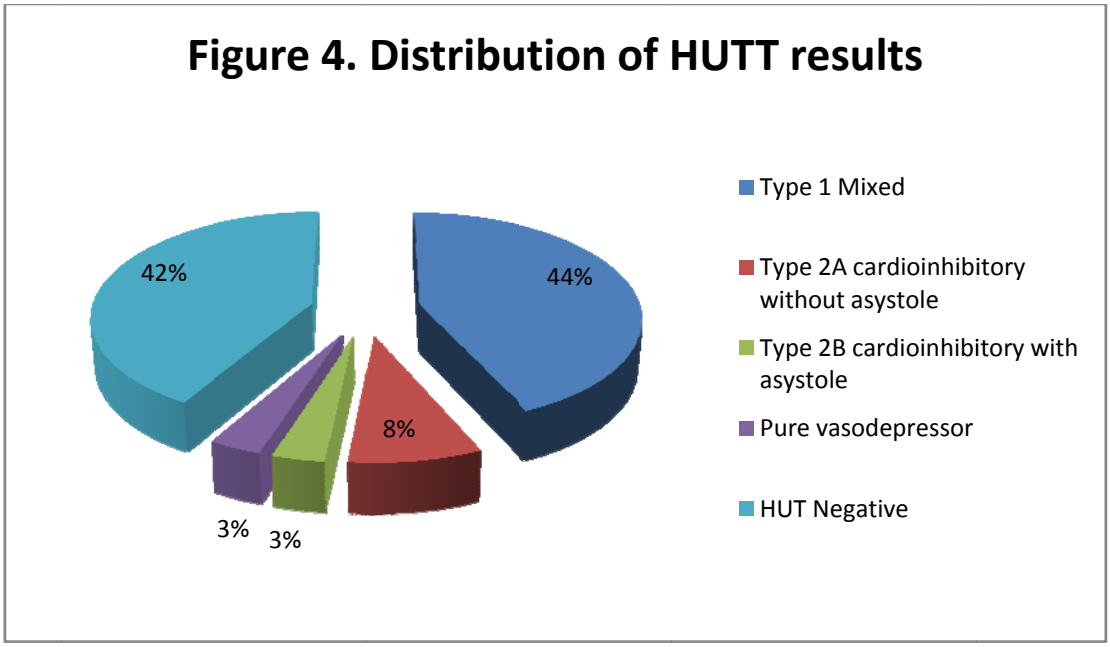
Response to HUT and hemodynamic variables during HUT test

Thirty five (58.3%) patients had positive HUT test response. Of those with a HUT positive response only one patient had a positive response during the drug free tilt (1.7% of the total population). The mean time to syncope was 31.5± 6.9 minutes. Distribution of HUT test responses is illustrated in Figure 4. Type 1 or mixed response was commonest (43.3%). This was followed by type 2A or cardio-inhibitory without asystole (8.3%). Both type 2B and type 3 responses were rare (3.3% each). Eight patients older than 60 years had a positive HUT test response (72.7% of those older than 60 years).

Figures 5 and 6 illustrate the changes in heart rate and mean arterial pressure (MAP) during HUT respectively. The data in figures 5-9, and in table 10, represent the measurements at five time-points; baseline supine state, at 5 and 10 minutes after initiation of drug free tilt, at 5 minutes prior to end of HUT and just prior to end of HUT (if HUT test negative) or at onset of symptoms (if HUT test positive). The initial response to HUT was an increase in heart rate, which started as early as five minutes of HUT. The mean heart rate progressively increased till the end of tilt in HUT test negative patients. Whereas, in HUT test positive patients, heart rate

progressively increased only to abruptly drop at the onset of symptoms. The difference in heart rate between the two groups became significant only at the end of HUT test.

There was no significant intergroup difference in the MAP between the HUT positive and HUT negative groups during the drug free tilt phase. There was no significant hypotension after nitrate administration in HUT negative patients. In HUT positive patients, the decrease in MAP preceded the bradycardia response. This was consistent with a diagnosis of type 1 or mixed response in 74.3% of HUT positive patients. Most patients had symptoms of cerebral hypo-perfusion/ syncope at a MAP of less than 60 mm Hg.



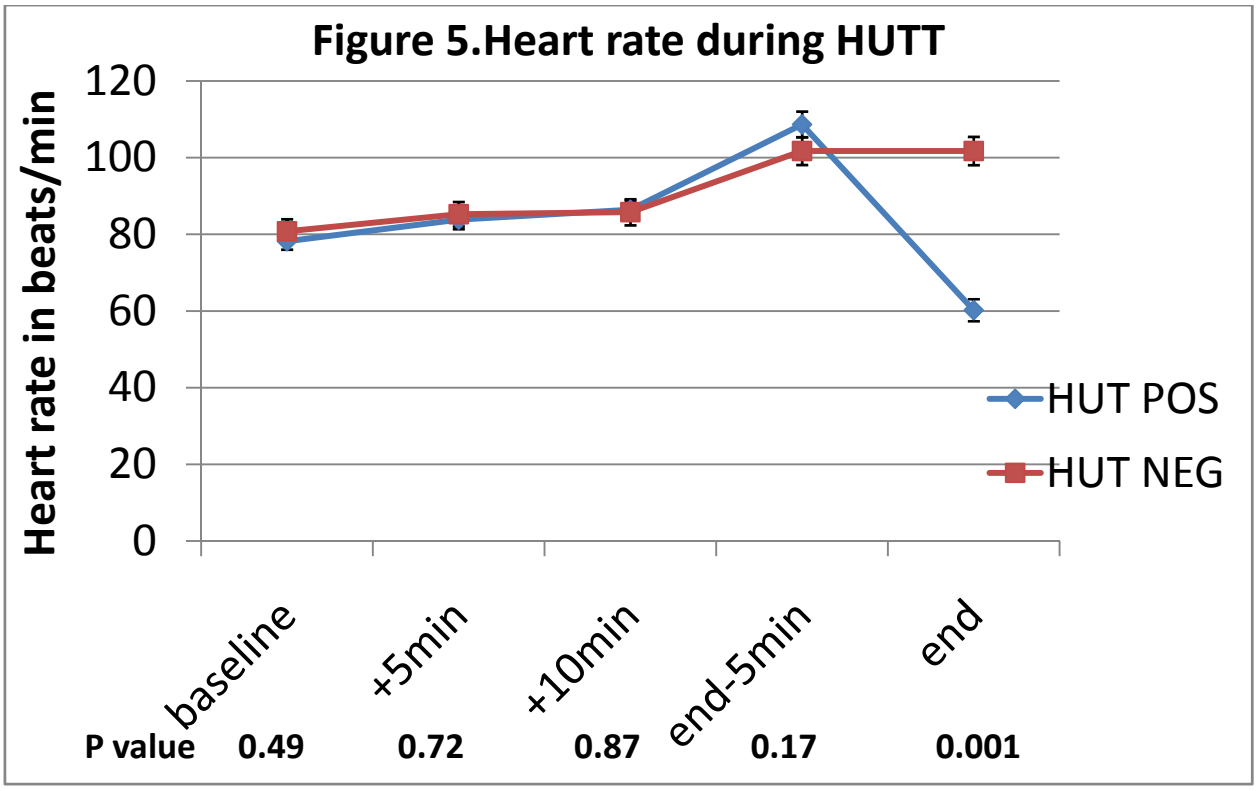


Figure 5.Heart rate during HUTT
Heart rate in beats/min
120
100
80
60
40
20
0
HUT POS
HUT NEG
baseline
+5min
+10min
end-5min
end
P value
0.49
0.72
0.87
0.17
0.001

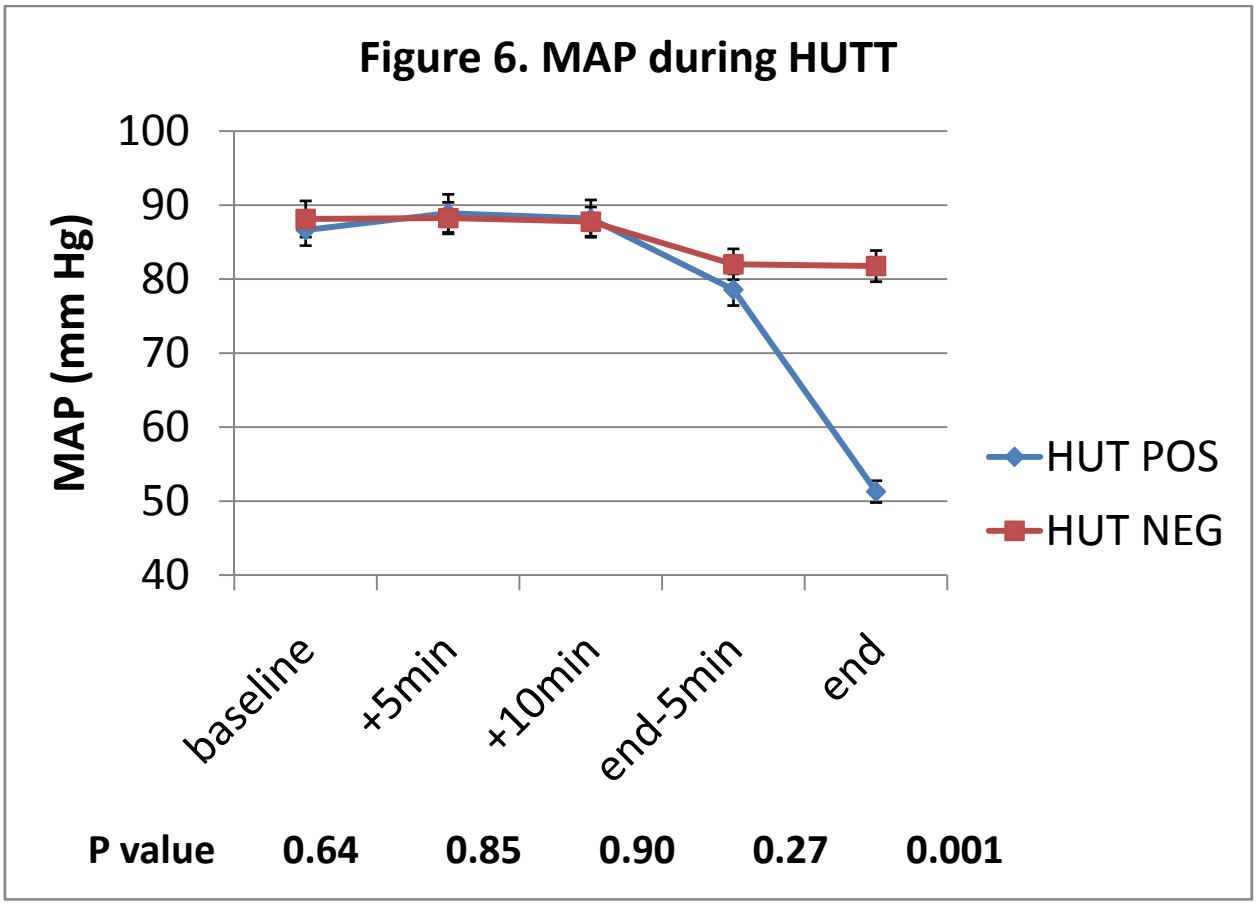


Figure 6. MAP during HUTT
MAP (mm Hg)
100
90
80
70
60
50
40
HUT POS
HUT NEG
baseline
+5min
+10min
end-5min
end
P value
0.64
0.85
0.90
0.27
0.001

Echocardiographic variables monitored during HUT

The left ventricular internal dimensions during diastole and systole (LVIDD/LVIDS) were recorded during the supine resting stage, 1 min after initiation of HUT, at 5,10, 20 minutes, at 1 minute post nitrate administration, 25,30, 35 and 40 minutes and once at the onset of symptoms if any. The measurements at the first three and last two time points are illustrated in figures 7-9 and in table 10.1-10.3. The LVIDD and LVIDS decreased significantly from baseline in both HUT positive and negative groups during tilt. LVIDD decline started early in the HUT positive group. The reduction in LVIDD in HUT positive group as compared to HUT negative group was greater, though it was not statistically significant. There was an early onset and rapid decline in LVIDS in HUT positive patients as compared to HUT negative patients.

At the end of the tilt phase, FS in the HUT positive group increased significantly from baseline (32.4±0.68% to 37.5±0.64 %), while FS did not change significantly in the HUT negative patients. This increase in FS in the HUT test positive group was apparent as early as 10 minutes after initiation of tilt (Figure 9). The rate of change of FS as depicted by the FS slope was significantly more rapid in the HUT test positive group: +0.23±0.34 %/min as compared to -0.002±0.6 %/min in the HUT test negative groups. These changes are depicted in table 11.

Table 10.1. Echocardiographic and Hemodynamic Parameters at Specified Time Periods During HUT test: Time Trend for **HUT Positive Patients**

	Baseline supine	Baseline+ 5 min	Baseline+10 min	End - 5 min	End
HR(beats/min)	78±2	83±2 *	86±2 ‡	109±3 ‡	60±3 ‡
LVIDD mm	42.1±0.55	40.6±0.58 ‡	40.4±0.64 ‡	39.4±0.76 ‡	39.0±0.74 ‡
LVIDS mm	28.4±0.43	27.0±0.42 ‡	26.5±0.49 ‡	25.5±0.56 ‡	24.4±0.58 ‡
MAP mm Hg	86±2	89±3	88±2	79±2 ‡	51±1 ‡
FS %	32.4±0.68	33.5±0.56 *	34.4±0.57 ‡	35.1±0.65 ‡	37.5±0.64 ‡

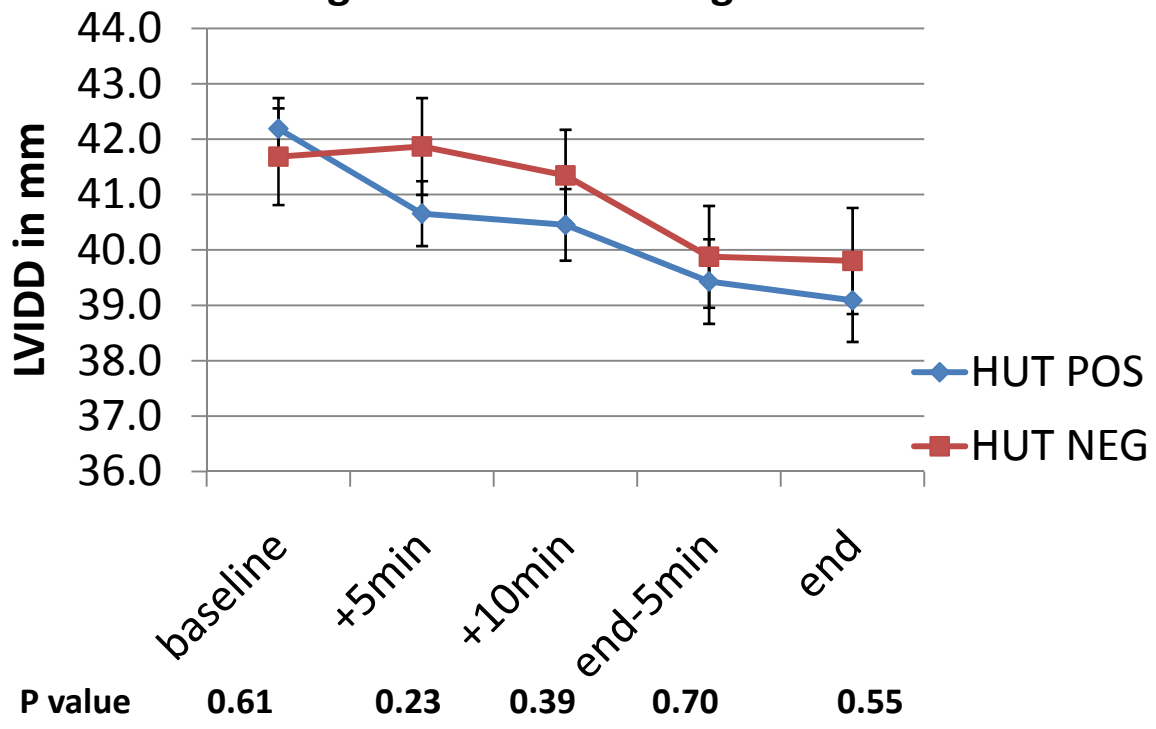
*P value <0.01, ‡ P value < 0.001 on paired samples t test compared to baseline. The time points showed in this table: Supine baseline, 5 minutes after start of 60 ° HUT (Baseline+5min), 10 minutes after HUT (Baseline + 10min), 5 minutes prior to end of HUT (End- 5 min) and at onset of symptoms or just prior to end of HUT (End). HR: Heart rate. MAP: Mean arterial pressure. FS: Fractional Shortening.

Table 10.2. Echocardiographic and Hemodynamic Parameters at Specified Time Periods During HUT test: Time Trend : HUT Negative Patients

	Baseline supine	Baseline+ 5 min	Baseline+10 min	End - 5 min	End
HR(beats/min)	81±3	85±3 *	85±3 *	102±4 ‡	102±4 ‡
LVIDD mm	41.6±0.87	41.8±0.87	41.3±0.82	39.8±0.91 ‡	39.8±0.95 ‡
LVIDS mm	28.0±0.69	28.1±0.74	28.0±0.69	26.9±0.75 *	26.8±0.72 ‡
MAP mm Hg	88±2	88±2	88±2	82±2 ‡	82±2 *
FS %	32.6±0.88	32.7±0.95	32.1±0.92	32.4±0.87	32.5±0.79

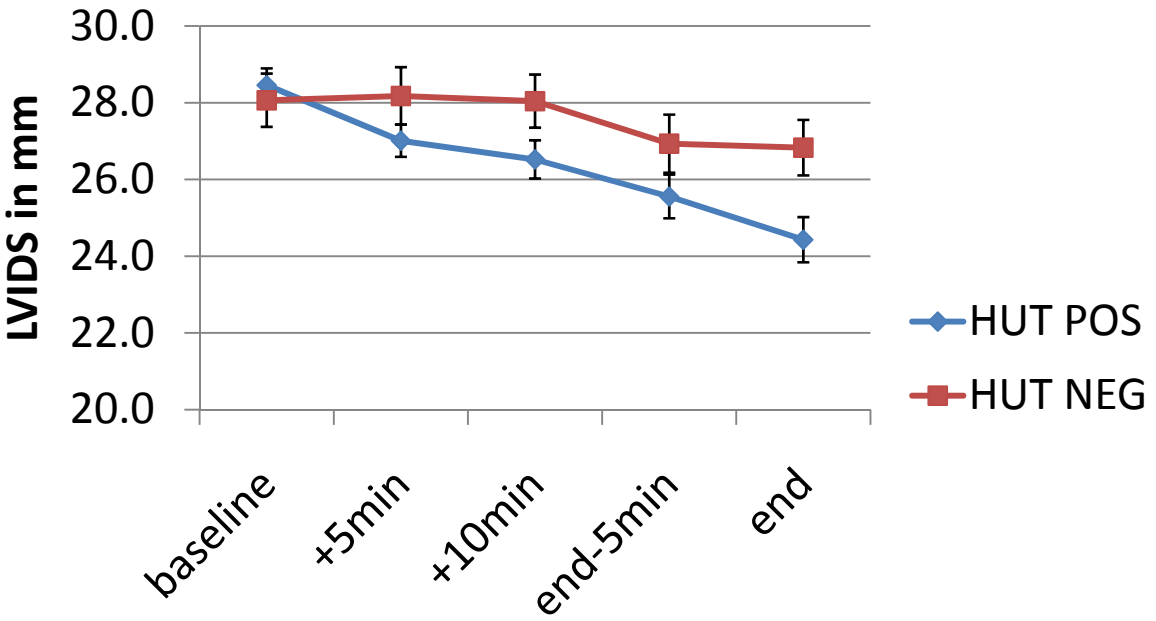
*P value <0.01, ‡ P value < 0.001 on paired samples t test compared to baseline value in each group. The time points showed in this table: Supine baseline, 5 minutes after start of 60 ° HUT (Baseline+5min), 10 minutes after HUT (Baseline + 10min), 5 minutes prior to end of HUT (End- 5 min) and at onset of symptoms or just prior to end of HUT (End). HR: Heart rate. MAP: Mean arterial pressure. FS: Fractional Shortening.

Figure 7. LVIDD during HUTT



P value	0.61	0.23	0.39	0.70	0.55

Figure 8. LVIDS during HUTT



P value	0.54	0.15	0.07	0.14	0.01

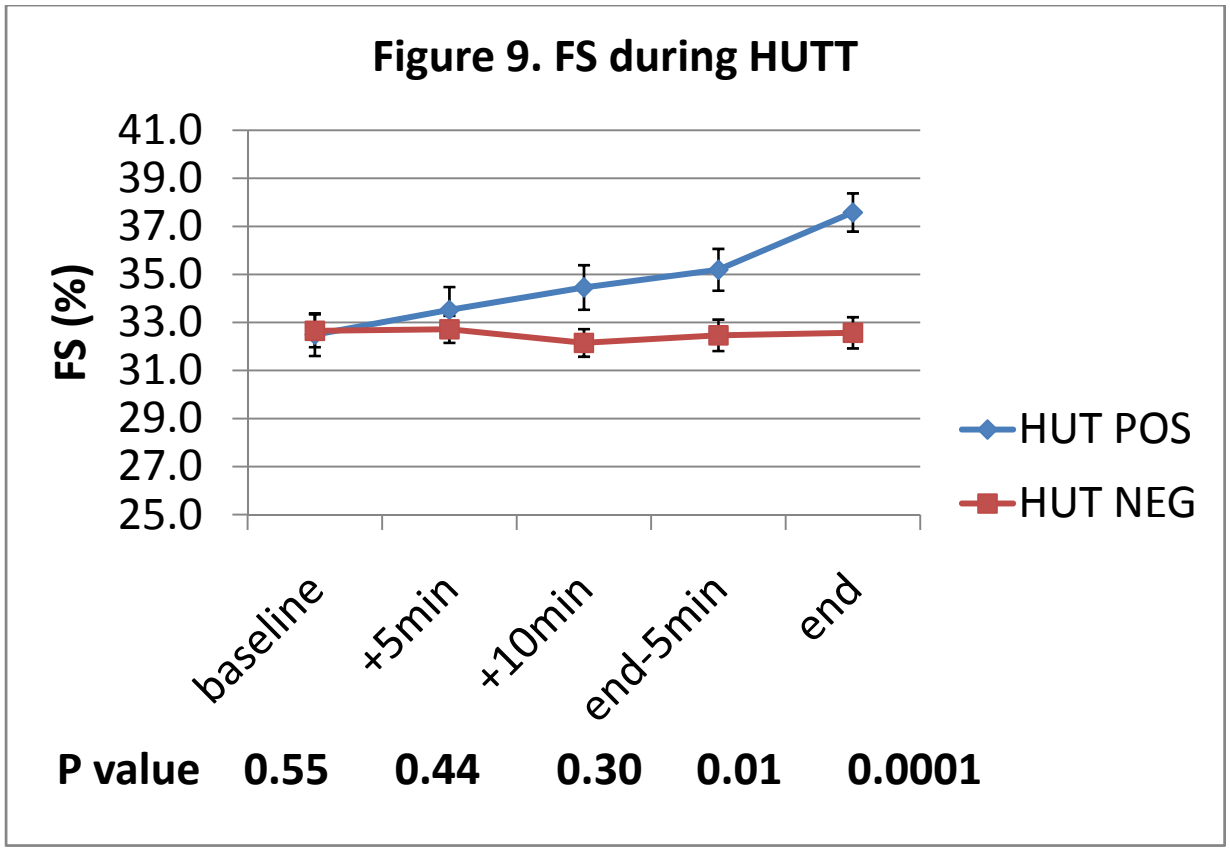
Table 10.3. Echocardiographic and Hemodynamic Parameters at Specified time Periods during HUT test: Time Trend: **Comparison HUT+ and HUT- groups**

	Baseline supine	Baseline+ 5 min	Baseline+10 min	End - 5 min	End
HR(beats/min)					
HUT+	78±2	83±2	86±2	109±3	60±3 ‡
HUT -	81±3	85±3	85±3	102±4	102±4 ‡
LVIDD mm					
HUT +	42.1±0.55	40.6±0.58	40.4±0.64	39.4±0.76	39.0±0.74
HUT -	41.6±0.87	41.8±0.87	41.3±0.82	39.8±0.91	39.8±0.95
LVIDS mm					
HUT +	28.4±0.43	27.0±0.42	26.5±0.49	25.5±0.56	24.4±0.58 *
HUT -	28.0±0.69	28.1±0.74	28.0±0.69	26.9±0.75	26.8±0.72 *
MAP mm Hg					
HUT +	86±2	89±3	88±2	79±2	51±1 ‡
HUT -	88±2	88±2	88±2	82±2	82±2 ‡
FS %					
HUT +	32.4±0.68	33.5±0.56	34.4±0.57	35.1±0.65 *	37.5±0.64 ‡
HUT -	32.6±0.88	32.7±0.95	32.1±0.92	32.4±0.87 *	32.5±0.79 ‡

*P value <0.01, ‡ P value < 0.001. The time points showed in this table: Supine baseline, 5 minutes after start of 60 ° HUT (Baseline+5min), 10 minutes after HUT (Baseline + 10min), 5 minutes prior to end of HUT (End- 5 min) and at onset of symptoms or just prior to end of HUT (End). HR: Heart rate. MAP: Mean arterial pressure. FS: Fractional Shortening. HUT+: HUT Positive. HUT-: HUT negative.

Table 10.4. Echocardiographic and hemodynamic variables classified according to response to HUT test

Variable	**HUT result** Number (%)		**P value**
	Positive Mean ± SD	**Negative** Mean±SD	
Heart rate (baseline) bpm	78±13	81±16	0.497
Heart rate (peak) bpm	110±19	105±18	0.350
Heart rate (minimum) bpm	59±14	80±14	0.0001
Difference between maximum and minimum heart rate during HUT bpm	52±21	25±12	0.0001
FS (baseline) %	32.4±4.0	32.6±4.4	0.885
FS (onset of symptoms/end of tilt) %	37.5±3.8	32.5±3.9	0.0001
FS change(absolute value) %	5.0±2.9	- 0.08±2.5	0.001
FS slope %/min	0.23±0.34	-0.002±0.6	0.001



Predictors of positive response to HUT

We assessed if the baseline patient characteristics or hemodynamic/ echocardiographic variables measured during HUT test would predict positive response to head up tilting in this cohort of patients. Both HUT positive and negative groups seemed to have a similar syncopal burden in terms of number of syncopal episodes prior to HUT and duration of symptoms prior to HUT. Clinical historical features typical of reflex syncope were also similarly distributed between the two groups. The baseline hemodynamic parameters were also similar. In conclusion none of the baseline clinical variables or hemodynamic parameters was predictive of a positive response to HUT testing (tables 11.1 and 11.2).

Table 11.1. Predictors of response to Head Up Tilt Testing: Categorical Variables				
Variable	**HUT result** Number (%)		**p-value**	**Odds ratio** (95% CI)
	Positive 35(58.3)	**Negative** 25(41.7)		
Sex (male)	22(62.9)	16(64)	0.928	0.95(0.32-2.76)
Age more than 60 years	8 (22.0)	3 (12.0)	0.284	1.64 (0.59-4.53)
More than 5 syncopal episodes prior to HUT test	12 (34.3)	6(24)	0.39	1.65 (0.52-5.23)
History of exertional syncope	2 (5.7)	3 (12)	0.64	0.44 (0.69-2.88)
Emotional stress associated with spells	8 (22.9)	5 (20)	0.79	1.18 (0.33-4.17)
History of lightheaded spells present	24 (68.6)	14 (56)	0.31	1.71 (0.59-4.97)
Sweating preceding spells	6 (17.1)	5 (20)	1.00	0.82 (0.22-3.08)
Prolonged sitting/ standing associated with spells	10 (28.6)	5 (20)	0.45	1.60 (0.47-5.44)
Palpitations preceding spells	6 (17.1)	3 (12)	0.72	1.51 (0.34-6.75)

Table 11.2. Predictors of Response to Head Up Tilt: Continuous Variables			
Variable	**HUT result** Number (%)		**P value**
	Positive Mean ± SD	**Negative** Mean ± SD	
Age (years)	46.00±16.32	46.1±13.89	0.98
Heart rate (baseline) bpm	78±13	81±16	0.497
MAP (baseline) mm Hg	87±12	88±12	0.646
FS (baseline) %	32.4±4.0	32.6±4.4	0.885
	Median (IQR)	Median (IQR)	
Number of prior syncopal episodes	2 (2-7)	3 (2-4)	0.795
Duration of symptoms (months)	12 (6-24)	10 (4-15)	0.105
Duration between last syncope and HUT test (weeks)	4.25 (1.00-8.71)	3.71 (1.92-11.0)	0.799

Follow up and predictors of recurrence of syncope

We could follow up all the enrolled patients till the end of the study period. Patients were followed up for 6.3± 2.5 months. Ten (16.7%) patients had recurrent syncope during the follow up period, while 50 (83.3%) patients remained symptom free with no recurrence of syncope. There was no mortality reported. We evaluated the baseline clinical variables, the hemodynamic

and echocardiographic variables recorded during HUT test, the response to HUT test and the treatment received to identify potential predictors of recurrence of syncope. None of the baseline echocardiographic parameters, the type of HUT test response or the treatment received influenced the outcome. FS change during HUT test was not predictive of recurrent syncope (Figure 10). Those patients who had a recurrence of syncope on follow up seemed to have a non significant trend for shorter duration of symptoms [median (IQR) 5 (2.75-17.25) v s 12 (6-24) p=0.09] but similar number of syncopal episodes prior to HUT testing [median (IQR) 2 (2-10) v s 2 (2-5), p=0.660]. Patients who had recurrence of syncope on follow up had significantly higher maximum heart rate during tilt (115±7 v s 106±20, p=0.018). On multivariate analysis the only factor that was predictive of recurrent syncope was the maximum heart rate achieved during HUT test. During HUT test, achieving a maximum heart rate of ≥ 108 beats per minute was predictive of recurrent syncope with a sensitivity of 80% and specificity of 50% based on receiver operating characteristic (ROC) curve analysis (Figure 11). Subgroup analysis including only HUT test positive patients did not identify any predictor of recurrent syncope.

Table 12.1. Predictors of recurrent syncope on follow up: Categorical Variable

Variable	**Syncope recurrence** Number (%)		**p-value**	**Odds ratio** (95% CI)
	Present 10 (16.7)	**Absent** 50 (83.3)		
Sex (male)	8 (80)	30(60)	0.299	2.66 (0.512-13.87)
More than 5 syncopal episodes prior to HUT test	4 (40)	14(28)	0.468	1.71 (0.41-7.00)

Peak HR achieved during HUT ≥ 108	26 (52)	9 (90)	0.035	8.30 (0.97-70.55)
HUT Positive	6 (60)	29 (58)	1.00	1.08 (0.27-4.33)
Type 1 Positive	5 (50)	21 (42)	0.64	1.38 (0.35-5.38)
Type 2A Positive	1 (10)	4 (8)	1.00	1.27 (0.12-12.80)
Type 2B Positive	0 (0)	2 (4)	1.00	1.20 (1.07-1.35)
Type 3 Positive	0 (0)	2 (4)	1.00	1.20 (1.07-1.35)
HUT Negative	4(40)	21 (42)	1.00	0.92 (0.23-3.67)
Use of beta-blockers on follow up	6(60)	22(44)	0.49	1.90 (0.47-7.61)
Use of Metoprolol on follow up	3 (30)	9(18)	0.40	1.95 (0.42-9.04)
Use of Atenolol on follow up	3 (30)	8 (16)	0.37	2.25 (0.47-10.59)
Use of Bisoprolol on follow up	0 (0)	5 (10)	0.67	1.22 (1.07-1.38)
Tilt training done	4 (40)	19 (38)	1.00	1.08 (0.27-4.35)

Table 12.2. Predictors of recurrent syncope on follow up: Continuous Variables

Variable	**Syncope Recurrence** Mean ± SD		p- value
	Present (N=10)	Absent (N=50)	
Age (years)	45.08±13.79	46.23±15.62	0.829

Heart rate (baseline) bpm	84±10	78±15	0.284
Heart rate (peak) bpm	115±7	106±20	0.018
Heart rate (minimum) bpm	72±17	67±17	0.364
MAP (baseline) mm Hg	90±16	87±11	0.394
MAP (end of tilt) mm Hg	65±19	63±17	0.694
FS (baseline) %	34.5±4.0	32.1±4.1	0.16
FS change(absolute value) %	3.2±2.88	2.8±3.9	0.795
Follow up duration (months)	5.85±3.70	6.39±2.30	0.668
	Median (IQR)	Median (IQR)	
Number of prior syncopal episodes	2 (2-10)	2 (2-5)	0.660
Duration of symptoms (months)	5 (2.75-17.25)	12 (6-24)	0.099
FS slope %/min	0.11(0.02-0.15)	0.09 (0.01-0.19)	0.937

Following parameters for HUT positive patients alone: N=35	**Syncope Recurrence** N=6	**Syncope Recurrence** N=29	P value
Time of onset of syncope during HUT minutes Mean ± SD	32.83±3.32	31.24±7.49	0.616
FS change (absolute value) % Mean ± SD	4.9±2.4	5.1±3.1	0.88
Tilt training done on follow up: Number (%)	3 (50)	17(58.6)	1.00

Two patients underwent pacemaker implantation on follow up. One HUT negative patient underwent VDDR pacemaker implantation for significant AV conduction disease. Another HUT negative patient had recurrent syncope and underwent pacemaker implantation at another hospital. Another patient with type 2B response underwent EP study and was found to have sinus node dysfunction and hence was advised pacemaker implantation.

Therapy did not alter the recurrence of syncope. We analyzed the use of atenolol, metoprolol and bisoprolol separately. None of these drugs were associated with a decrease in the incidence of recurrent syncope. Fludrocortisone was not prescribed to any of these patients. Tilt training was advocated to all HUT positive patients, however only 57.1% of patients with a positive HUT test response were performing tilt training at the end of the follow up period. Despite performing tilt training with adequate compliance, it was not associated with a significant decline in the incidence of recurrent syncope.

Table 13. Logistic Regression: Predictors of Recurrent Syncope Multivariate Analysis

	OR (95% CI)	P value
Duration of symptoms in months	0.99 (0.96-1.02)	0.702
Peak HR achieved during HUT ≥ 108	8.62 (1.002-73.84)	0.049

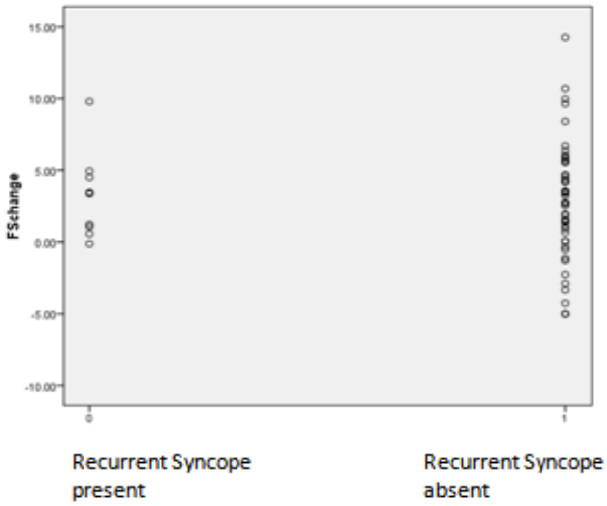


Figure 10. Distribution of FS change during HUT in patients with and without recurrence of syncope

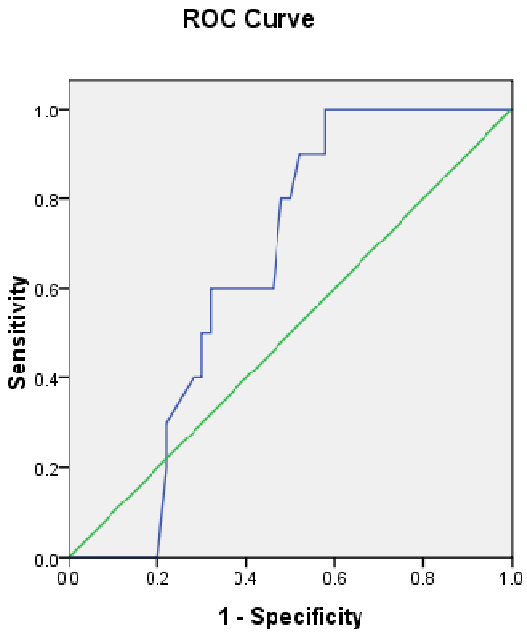


Figure 11. Use of maximum HR achieved during HUT for predicting recurrent syncope: ROC analysis

DISCUSSION

Baseline Characteristics:

The mean age of the present study cohort was 46 ±15 years, with a demographic profile similar to a series of 640 patients reported by Kazemi, et al (99). The median duration of symptoms prior to HUT test was 12 months and the onset of symptoms was in the fourth decade of life, which appear to be older than that reported in a community survey done by Colman, et al (100). The majority of subjects in this community survey had experienced neurocardiogenic syncope as teenagers and adolescents while only 5% of patients had the first episode after the age of 40 years (100). This difference in the age of onset of symptoms can be explained by the recall bias that patients may have. Patients may not recall the initial episodes of syncope which occur in adolescence, thus affecting the accuracy of the estimated duration of symptoms. An alternative explanation is referral bias. Colman's review was a community based study and our data was obtained from a tertiary referral centre with a dedicated electrophysiology unit. Only a small fraction of patients in the community experiencing syncope, seek medical consultation. Younger patients are less likely to seek medical care as compared to older patients for complaints of syncope (14). The initial episode of syncope has a bimodal distribution, with a high prevalence of first faints in patients in second and third decades of life, with a peak of about 47% in females and 31% in males occurring in the second decade of life (101). The second peak occurs after the age of 65 for both males and females (102). However we could not demonstrate such a pattern, probably because we excluded patients presenting with a single episode of syncope.

The effect of age on the results of HUT test has been studied. Kochiadakis, et al, demonstrated that age predicted a different type of syncopal response to HUT testing (103). The response is predominantly cardio-inhibitory or mixed in young patients and vasodepressor in elderly patients. Similarly in our cohort, all the patients with pure vasodepressor response to

HUT were more than 65 years of age (25% of patients older than 65). A number of explanations for this phenomenon have been proposed. Sympathetic withdrawal in response to tilt is more pronounced in older subjects (103). Also in elderly subjects, the autonomic nervous system is unable to make the appropriate compensatory heart rate changes in response to the orthostatic stress (104). In our cohort 22% of HUT positive patients were older than 60 years. It has been shown that NTG may have different pharmacodynamic properties in the older patients. NTG causes greater vasodilatation in older patients probably due to the decreased intravascular volume in this population (105).

Echocardiography during HUT test:

The use of HUT testing has become widespread, especially for unmasking susceptibility to vasovagal faint in subjects with presenting with syncope of unknown cause, despite the lack of an accepted diagnostic "gold standard" for reflex syncope. The high unexplained syncope rate in both general and specialist referral settings justify new strategies for evaluation and diagnosis of syncope.

Studies have analyzed the utility of echocardiography during HUT testing for providing additional diagnostic information (11,19,20,79,83). Even though tilt induced syncopal response can occur in healthy volunteers (false positive), the hemodynamic as well as humoral changes that accompany this response are qualitatively distinct from those of HUT positive patients who complaint of syncopal spells during normal daily activities (true positive) (11). In patients with neurally mediated syncope it has been shown that there is more rapid peripheral blood pooling as shown by a more marked and rapid decline in LVIDD as compared to healthy HUT positive volunteers. These patients are also shown to have higher LV contractility as indicated by values

of FS as compared to healthy HUT positive healthy volunteers (11). This is mediated by higher levels of epinephrine in the first group (11).

FM Leonelli, et al suggested that the false positive group of patients in those undergoing HUT testing for unexplained syncope, may be identified by using echocardiographic parameters like FS and LVIDD (11). An increase in FS from baseline (FS change) during HUT test may be used as a surrogate marker for identifying true positive patients. Similar to the findings in Leonelli's study we found that in HUT positive patients, there is an early onset significant increase in LV FS during tilt as compared to HUT negative patients. The decrease in LVIDD during HUT is a marker of peripheral venous pooling. In our cohort there was a non significant trend for greater decrease of LVIDD in HUT positive patients as compared to HUT negative patients (Figure 6). To the best of our knowledge no study has earlier analyzed the utility of echocardiographic parameters measured during HUT for predicting clinical outcomes.

Predictors of a positive HUT response:

Our study sought to determine the predictors of a positive HUT response, including the clinical history, baseline hemodynamic and echocardiographic parameters recorded during HUT test in patients with unexplained syncope. Oh, et al, undertook a similar study in 711 patients. In their series vasodepressive type was the predominant pattern of positive response (76.6%), compared to only 3.3% in our series. They found the occurrence of junctional rhythm to be a predictor of an impending positive response to HUT. The shorter time interval between the last episode and HUT test, younger age and a history of physical injury during a syncopal episode were found to be associated with a positive response to HUT (106). In our cohort, which was much smaller, compared to the above mentioned series, we found that none of the clinical history

or baseline hemodynamic/echocardiographic parameters other than FS change were predictive of a positive response to HUT. FS change can be considered to be an early marker for identifying HUT positive response (Figure 9). We could demonstrate a significant increase in FS from baseline, with a mean increase of 5±2.9 % in patients with a positive HUT response.

Of the 35 patients with a HUT positive response only one patient had a positive response during the drug free tilt (1.7% of patients undergoing HUT testing); the remaining had a positive response after administration of nitroglycerine. This is in contrast to data from table 5, which shows that studies report 10-12% passive stage positivity, for the same passive phase duration. In patients with unexplained syncope and a positive HUT response, the autonomic imbalance causing syncope begins immediately after tilt, but syncope develops much later (107). Hence early sympathovagal imbalance in our cohort cannot be ruled out in spite of the late occurrence of syncope. Our overall HUT positivity of 58.3% is slightly less than the 62-78% positivity reported in table 5 with a similar duration of passive tilt and a similar dose of NTG.

Predictors of Recurrence of Syncope:

Risk stratification is the cornerstone of management of syncope. The risk of sudden cardiac death as well as the risk of recurrence of syncope and physical injury should be assessed. Several risk scoring systems including the OESIL score, EGSYS score and the S. Francisco Syncope rule are available for predicting mortality and serious adverse effects (108)(109)(110). Most of the serious adverse outcomes are related to the underlying cardiac disease. We did not include patients with structural heart disease in our study. In addition, only 8 (13.3%) patients were older than 65 years, making the risk profile of our cohort low. Therefore the absence of life threatening events or mortality at the end of 6.3± 2.5 months of follow up was not surprising.

Assessment of the risk of recurrence of syncope is important; recurrent syncope especially without prodrome is associated with increased risk of physical injury including orthopedic fractures and soft tissue injury in up to 12% of patients (4). A number of studies have looked at predictors of recurrent syncope in order to identify a patient subgroup with high risk of recurrence of syncope (1,111-113). The number of episodes of syncope during the patient's life term is the strongest predictor of recurrence (111). In our study none of the baseline clinical variables, the echocardiographic parameters, the type of HUT test response or the treatment received influenced the outcome.

We could identify only one hemodynamic variable: the maximum heart rate achieved during HUT test as the significant predictor of recurrent syncope. We observed that patients who achieved heart rate greater than 108 bpm were more likely to develop recurrence of symptoms (sensitivity of 80% and specificity of 50%). Tachycardia during tilt testing may be a surrogate marker of the sympathetic surge and epinephrine levels. Studies have shown a significantly higher peak epinephrine level in patients presenting with neurally mediated syncope with HUT positive response as compared to healthy volunteers with a HUT positive response (11). Epinephrine dilates skeletal muscle and splanchnic resistance vessels at concentrations measured in humans under stress thereby contributing to inappropriate vasodilatation (114). Epinephrine peaked in patients with neurocardiogenic syncope just before the occurrence of symptoms. It is not clear if epinephrine surge is the cause for the syncope or is just a failing compensatory mechanism to maintain cardiac output. Our findings suggest that the higher peak heart rate achieved during HUT test, may identify a subgroup of patients with increased risk of recurrence of syncope. In this patient subgroup further studies are required to identify the contribution of excess sympathetic activation to the pathogenesis of recurrent syncope. Further we need to study

if aggressive beta blockage in this subgroup of patients will be beneficial in preventing recurrent syncope.

An increase in FS from baseline (FS change) during HUT test may be used as a surrogate marker for identifying true positive patients (11). True positives and false positives may have differences in terms of recurrence rate of syncope, the main outcome of interest in syncope management. Hence we generated a null hypothesis that there is no difference in FS change in those patients who had recurrence of syncope on follow up as compared to those who had no recurrence. However in our study we could not demonstrate any difference in the FS change during HUT test between these two groups. There was no significant difference irrespective of whether the whole cohort was analyzed or just the HUT test positive cohort was analyzed.

Sheldon, et al, demonstrated that the risk of a recurrent syncope following a positive response on isoproterenol challenge HUT test can be estimated from two simple clinical variables—the number of episodes of syncope prior to testing and the duration of symptoms prior to testing (113). They added that this estimate can be modified further based on whether patients faint or just have presyncopal symptoms during the HUT test and based on the minimum heart rate recorded during symptoms. There may have been a recall bias in our group of patients as compared to the more literate patients that were interviewed in the above mentioned study. Another study demonstrated that female gender, the presence of ≥ 3 syncope events before HUT, and arterial baroreceptor sensitivity below median value after the start of HUT or after the administration of NTG were significantly and independently associated with recurrence of syncope (115). However we did not find a significant association between any of these factors and recurrence of syncope. Multiple studies have shown that the type of response to HUT testing does not predict future recurrence (112). Our results confirm the same.

We also evaluated if the treatment received influenced the recurrence of syncope. Trials have shown that in patients with recurrent vasovagal symptoms triggered by stress caused by prolonged upright posture, the prescription of progressively longer periods of enforced upright posture (also known as 'tilt training') may decrease the recurrence of syncope (89). In our study cohort, all patients with a positive response on HUT were provided with oral and written instructions to perform tilt training exercises. However the only 57.1% of patients were compliant with the tilt training protocol at the end of the follow up period. In the compliant patients, we did not find any significant effect of tilt training on the recurrence rate of syncope. This may be due to the small size of the study.

We also analyzed the pattern of beta blocker usage and its influence on the recurrence of syncope. Though widely used in the management of reflex syncope, multiple studies have failed to prove the efficacy of beta blockers in preventing recurrent syncope (91, 92). Our findings were consistent with these studies. Beta blockers including atenolol and metoprolol failed to prevent recurrent syncope. However these agents still continue to be widely used.

LIMITATIONS

1. We recruited our patients from the outpatient department of cardiology units and the electrophysiology units. This may have caused a referral bias. Hence the pretest probability of having underlying arrhythmia as the cause of syncope is higher as compared to the general population. This may have affected the response to tilt testing.
2. The sample size that we have used is not adequately powered to detect small differences in outcome. The study was designed to detect a 30% difference in outcome with 80% power.
3. Inter-observer variability was not accounted for. In the measurement of echocardiographic parameters assessment of kappa statistic should have been ideal.
4. FS is preload dependent. Use of preload independent variables like strain measurements of the LV long-axis function would have provided better insight into LV contractility changes (116).
5. Intravenous cannulation decreases the specificity of tilt testing especially in elderly and children (117). Vascular cannulation may have decreased the specificity of HUT test in our study.
6. Non invasive continuous finger blood pressure using digital photoplethysmography measurement has become the standard of care for monitoring blood pressure during tilt testing (118). However this was not available at our institution, thus limiting the generalizability of the study findings.
7. Short follow up time is yet another limitation of our study. About one third of patients with syncope have a recurrence when followed up for 3 years (15). Hence longer follow up may have altered our results.

8. Many patients with positive tilt test have been instructed on specific physical maneuvers to abort the progression of presyncope to syncope. Hence many patients may just have presyncope on follow up and experience no syncopal episodes. This has not been accurately documented in the study.
9. We used a 20 minute post nitrate tilt protocol as compared to the 15 minute post drug tilt in the Italian protocol. This may decrease the specificity of tilt testing.
10. Smoking status significantly influences the response to HUT test during the post nitrate phase (119). We did not document the smoking status of our study patients.
11. Monitoring of echocardiographic variables at regular intervals during HUT test may not be cost effective in terms of the additional diagnostic or prognostic information obtained.

CONCLUSIONS

1. In patients with a positive response on HUT testing, there is an early onset of significant increase in LV fractional shortening during tilt as compared to those with a negative HUT response

2. Change in LV fractional shortening during HUT is not predictive of recurrence of syncope on follow up

3. Patients who have recurrence of syncope on follow up tend to have higher peak heart rate attained during HUT test. Hence peak heart rate attained during HUT test can be used to predict the risk of recurrence of syncope on follow up in patients with unexplained syncope.

H.U.T TEST STUDY PROFOMA

Name:

Hosp no:

Address:

Mobile number 1: 2:

Enrollment number:

1. Date of enrollment (dd/mm/yyyy):
2. Date of birth(dd/mm/yyyy):
3. Sex (1. Male 2. Female) :
4. Number of episodes of syncope:
5. Date of last episode (dd/mm/yyyy):
6. Duration of symptoms in months:
7. Duration of LOC during last episode of syncope in minutes:
8. History of exertional syncope 1. Yes 2. No
9. At times do you wake with a cut tongue after your spells? 1. Yes 2. No
10. At times do you have a sense of aura (deja vu or jamais vu) before your spells? 1. Yes 2.No
11. At times is emotional stress associated with losing consciousness? 1. Yes 2.No
12. Has anyone ever noted your head turning during a spell? 1. Yes 2.No

13. Has anyone ever noted that you are unresponsive or have no memory of your spells afterwards? 1. Yes 2.No
(Score as yes for any positive response)

14. Has anyone ever noted that you are confused after a spell? 1. Yes 2.No

15. Have you ever had lightheaded spells? 1. Yes 2.No

16. At times do you sweat before your spells? 1. Yes 2.No

17. Is prolonged sitting or standing associated with your spells? 1. Yes 2.No

18. Posture at onset of syncope 1. Sitting 2. Standing 3. Supine 4. Nonspecific

19. Recovery 1. Gradual 2. Sudden

20. Palpitations preceding the spell 1. Yes 2. No

Baseline	BP	HR	LVIDd	LVIDs
Supine				
Tilt start				
5 min				
10 min				
15 min				
20 min				
Post nitrate start				
5 min				
10 min				
15 min				
20 min				

Min HR……. Max HR…….

21. Result:

1. Type 1 Mixed
2. Type 2A cardioinhibitory without asystole
3. Type 2B cardioinhibitory with asystole
4. Type 3 pure vasodepressor
5. Orthostatic hypotension
6. HUT negative

Follow up:

1. Follow up date (dd/mm/yyyy)
2. Medication: Betablocker 1. YES 2. NO
3. In Yes for above 1. Metoprolol 2. Atenolol 3. Not applicable
4. Fludrocortisone 1. YES 2 NO
5. Postrural training exercises done with > 80% compliance 1. YES 2. NO
6. Reccurence of syncope 1. YES 2. NO
7. Duration of LOC on reccurence

Informed Consent form to participate in a research study

Study Title: Predictors of Recurrence of Syncope in Patients with Unexplained Syncope Undergoing Head Up Tilt Testing: A Study Using Clinical, Hemodynamic and Echocardiographic Variables Study Number:

Subject's Initials: _____ Subject's Name: _____

Date of Birth / Age:_____

Please initial box

(Subject)

(i) I confirm that I have read and understood the information sheet for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ]

(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s) [ ]

(v) I agree to take part in the above study. [ ]

Signature (or Thumb impression) of the Subject/Legally Acceptable Representative:_____

Date: _____/_____/_____

Signatory's Name: _____

Signature of the Investigator: _____

Date: _____/_____/_____

Study Investigator's Name: Dr Anoop Mathew, MD

Signature of the Witness: _____

Date:_____/_____/_____

Name of the Witness: _____

Patient Information Sheet

You are enrolled in a research study to evaluate the additional use of echocardiographic monitoring during HUT test. This study will take approximately one hour. Initially to monitor the blood pressure in your arteries a small catheter will be introduced into an artery in your wrist. You will be made to sleep on an electronically controlled tilt table and the table will be tilted up to an angle of 70 degrees. During this time your vital parameters will be monitored. An echo evaluation of your heart by keeping an ultrasound probe on your chest will be done every 5 minutes during the test. The participation in this modification of the Head up tilt test that you are undergoing is purely voluntary. You have the option of undergoing the HUT test without using echo monitoring. You will not be penalized in any form for not participating in this study or for withdrawing from this study at any stage, if you desire so. In the event of any injury arising as a result of participation in this study treatment for the same at our institution will be provided.

GLOSSARY FOR THE MASTER DATA SHEET

Recsyncope : Recurrence of syncope on follow up

0= No recurrence of syncope on follow up

1= Syncope recurred on follow up

Sex= Sex of the subject

0= Male, 1= Female

Syncopemorethan5: syncopal episodes prior to HUTT more than 5

HUTpositive: Positive response on HUT test

0= negative response on HUT testing 1= positive response on HUT testing

Type1HUTTpositive: HUT test type 1 positive response

0= Type 1 HUT positive 1= All else

HUTT2Apositive: HUT test type 2A positive

0= Type 2A HUT positive 1= All else

HUTT2Bpositive: HUT test type 2A positive

0= Type 2B HUT positive 1= All else

Type3positive: HUT test type 3 positive

0= Type 3 HUT positive 1= All else

HUTnegative: HUT test negative response

0= HUT negative 1= All else

Bblockeruse: Use of beta blockers on follow up

0 = "Patient using beta blockers on follow up" 1 = "No beta blocker use on follow up"

Metoprololuse: Metoprolol used on follow up

- 0 = "Metoprolol used on follow up"
- 1 = "Metoprolol NOT used on follow up"

Atenololuse: Atenolol used on follow up

- 0 = "Atenolol used on follow up"
- 1 = "Atenolol NOT use on follow up"

Bisoprololuse: Bisoprolol used on follow up

- 0 = "Bisoprolol used on follow up"
- 1 = "Bisoprolol NOT used on follow up"

Tilttrainin: Tilt training done on follow up with adequate compliance

- 0 = "Tilt training done on follow up"
- 1 = "Tilt training NOT done on follow up"

Age: Age of the patinent in years

Nosyncope: Number of episodes of syncope prior to HUT

Duratsymptoms: Duration of symptoms in month prior to HUT

Followmonth: duration of follow up in months

SyncopetoHUTT: Duration between last syncopal episode and HUT

HRbaseline: Heart rate at baseline

HRpeak: Maximum heart rate recorded during HUT

HRmin: Minimium HR recorded during HUT test

MAPbaseline: Mean arterial pressure (MAP) at baseline

MAPmin: MAP minimum recorded

FSchange: Fractional Shortenting (FS) change during HUT test

Syncopetime: Time in minutes at onset of syncope during HUT

FSslope: Change in FS during HUTT %/min

Dateenroll: Date of enrollment

Dob: Date of birth

Datelastsyn: Date of last episode of syncope

Exertsyncop: History of exertrional syncope 1= Yes 2= No

Cuttongue: Do you wake with a cut tongue after spells 1= Yes 2= No

Dejavu: Do you have sense of aura before the LOC 1= Yes 2= No

Emotionstress: Is emotional stress associated with LOC 1= Yes 2= No

Headturnin: Has anyone noted your head turning during a spell 1= Yes 2= No

Unresponse: Ever been unresponsive, no memory of spells 1= Yes 2= No

Confusion: Ever noted you are confused after a spell 1= Yes 2= No

Lightheaded: Ever had lightheaded spells 1= Yes 2= No

Sweating: Do you sweat before your spells 1= Yes 2= No

Prolongedsitting: Is prolonged sitting or stanging associated with spells 1= Yes 2= No

Postureonset: Posture at onset of syncope

1= Sitting 2= Standing 3= Supine 4= Not able to specify

Recovery: Recovery from spells 1= Gradual 2= Sudden

Palpitations: Palpitations preceeding syncope 1= Yes 2= No

HRTILT: Heartrate tilt start

HR5MIN: Heartrate 5 min

HR10MIN: Heartrate 10 min

HR15MIN: Heartrate 15 min

HR20MIN: Heartrate 20min

HRNITRATE: Heartrate postnitrate start

HR25MIN: Heartrate 25 min

HR30MIN: Heartrate 30 min

HR35MIN: Heartrate 35 min

HR40MIN: Heartrate 40 min

HREND: HR at end of HUT test

LVIDDbase: LVIDD Baseline

LVIDDTILT: LVIDD tilt start

LVIDD5MIN: LVIDD 5min

LVIDD10MIN: LVIDD 10 min

LVIDD15MIN: LVIDD 15min

LVIDD20MIN: LVIDD 20 min

LVIDDNITRATE: LVIDD post nitrate

LVIDD25MIN: LVIDD 25 min

LVIDD30MIN: LVIDD 30 min

LVIDD35MIN: LVIDD 35min

LVIDD40MIN: LVIDD 40 min

LVIDDEND: LVIDD at end of HUTT

LVIDSbase: LVIDS Baseline

LVIDStilt: LVIDS tilt start

LVIDS5MIN: LVIDS 5min

LVIDS10MIN: LVIDS 10min

LVIDS15MIN: LVIDS 15min

LVIDS20MIN: LVIDS 20 min

LVIDSNITRATE: LVIDS post nitrate

LVIDS25MIN: LVIDS 25 min

LVIDS30MIN: LVIDS 30 min

LVIDS35MIN: LVIDS 35min

LVIDS40MIN: LVIDS 40 min

LVIDSEND: LVIDS at end of HUTT

EFENDTIME: Time of last recording of LVIDD/LVIDS

MAPbase: MAP at baseline

MAPtilt: MAP at tilt start

MAP5MIN: MAP 5 minutes

MAP10MIN: MAP 10 minutes

MAP15MIN: MAP 15 minutes

MAP20MIN: MAP 20 minutes

MAPNITRATE: MAP post nitrate administration

MAP25MIN: MAP 25 minutes

MAP30MIN: MAP 30 minutes

MAP35MIN: MAP 35 minutes

MAP40MIN: MAP 40 minutes

MAPEND: MAP at end of tilt test

FSbaseline: Fractional Shortening at baseline

FStilt: FS at tilt start

FS5min: FS at 5 minutes of tilt

FS10min: FS at 10 minutes of tilt

FS15min: FS at 15minutes of tilt

FS20min: FS at 20 minutes of tilt

FSnitrate: FS post nitrate administration

FS25min: FS at 25 minutes of tilt

FS30min: FS at 30 minutes of tilt

FS35min: FS at 35 minutes of tilt

FS40min: FS at 40 minutes of tilt

FSend: FS at just prior to syncope or at end of tilt

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MASTER DATA SHEET

name	Recsyncope	Sex	syncopemorethan5	HUTpositive	Type1HUTTpositive	HUTT2Apositive	HUTT2Bpositive	Type3positive	HUTnegative	Bblockeruse	Metoprololuse	Atenololuse	Bisoprololuse	Tiltrainin	Age	Nosyncope	Duratsymptoms	followmonth	SyncopetoHUTT
MOHAMMED TOHID	0	0	0	0	1	1	1	1	0	1	1	1	1	1	49.02	5	6	8.51	2.00
SANTIRAM BANERJEE	0	0	1	1	1	1	1	0	1	0	0	1	1	0	68.94	2	12	11.27	4.29
SHYAM PRASAD SINGH	0	0	0	1	1	0	1	1	1	0	0	1	1	0	50.98	7	72	6.80	3.71
SRIHARI DAS	0	0	1	1	1	0	1	1	1	1	1	1	1	1	46.49	2	12	5.36	4.00
RAJAVELU P	0	0	1	1	1	0	1	1	1	0	0	1	1	1	51.78	2	3	4.93	3.43
KSHUDIRAN JANA	0	0	1	0	1	1	1	1	0	0	0	1	1	1	41.54	2	6	5.65	4.43
BALAIKUMAR SAHA	0	0	1	1	0	1	1	1	1	0	1	1	0	0	53.37	2	6	4.76	1.14
PRADIP SARKAR	0	0	1	1	0	1	1	1	1	0	1	0	1	1	36.75	2	60	7.79	1.29
KIRAN DEVI	0	1	1	1	0	1	1	1	1	1	1	1	1	0	23.61	2	24	7.39	2.29
NITAI CHANDRA MAJI	0	0	1	0	1	1	1	1	0	1	1	1	1	1	45.50	2	4	7.39	1.14
MOSTAQUE AHMED	0	0	1	0	1	1	1	1	0	0	1	1	0	1	56.09	2	10	4.17	2.00
JAGADISH CHANDRA SAHA	0	0	1	1	1	1	0	1	1	1	1	1	1	0	50.45	2	24	7.43	25.86
SHAIK MAHABOOB SAHEB	0	0	1	0	1	1	1	1	0	0	0	1	1	0	53.54	2	1	7.62	0.57
TULSI DE	0	1	0	1	0	1	1	1	1	1	1	1	1	0	67.61	15	120	7.13	0.86
NANU TABAURI	0	1	1	0	1	1	1	1	0	0	1	0	1	1	55.61	2	12	7.85	0.86
RUMIKA BIBI	0	1	0	0	1	1	1	1	0	1	1	1	1	1	16.72	5	24	7.00	19.86
LATA KAHAR	0	1	0	0	1	1	1	1	0	1	1	1	1	1	34.79	8	12	4.11	0.71
KARUNA SAHA	0	1	1	1	0	1	1	1	1	0	0	1	1	1	54.77	3	24	5.85	0.86
SIVAPRAKASAM R	0	0	1	0	1	1	1	1	0	1	1	1	1	1	19.90	2	1	7.82	2.29

Name	Recsyncope	Sex	syncopemorethan5	HUTpositive	Type1HUTTpositive	HUTT2Apositive	HUTT2Bpositive	Type3positive	HUTnegative	Bblockeruse	Metoprololuse	Atenololuse	Bisoprololuse	Tiltrainin	Age	Nosyncope	Duratsymptoms	followmonth	SyncopetoHUTT
NIBARAN CHANDRA DAS	0	0	1	0	1	1	1	1	0	0	1	0	1	1	61.03	2	36	3.71	2.57
AKHIL CHANDRA DEBNATH	0	0	0	1	0	1	1	1	1	1	1	1	1	1	72.95	5	36	3.55	4.29
ANANDA PAUL	0	1	1	0	1	1	1	1	0	1	1	1	1	1	58.52	2	4	7.56	0.29
ASHUTOSH PAUL	0	0	1	0	1	1	1	1	0	0	1	1	0	1	63.61	3	24	8.51	17.14
UMASRI ARCHANA	0	1	1	1	0	1	1	1	1	1	1	1	1	1	23.14	3	6	6.57	0.57
MAYA SAHA	0	1	1	0	1	1	1	1	0	1	1	1	1	1	53.37	3	36	8.41	20.86
RAMITA ROY	0	1	1	0	1	1	1	1	0	1	1	1	1	1	21.37	3	18	8.38	20.43
PABITRA KUMAR GHOSH	0	0	1	1	0	1	1	1	1	1	1	1	1	1	67.95	2	6	2.56	0.14
PADMANABHAN N	0	0	1	1	0	1	1	1	1	0	0	1	1	0	41.64	2	24	7.43	0.14
EDYFAIRLAND K	0	0	0	1	0	1	1	1	1	0	1	0	1	0	25.36	20	24	5.52	1.00
ASHIS MANDAL	0	0	0	1	0	1	1	1	1	0	0	1	1	1	37.92	5	24	7.52	17.71
SWAPAN BASU	0	0	1	1	0	1	1	1	1	0	1	0	1	0	50.99	2	8	5.32	0.29
PAVITHRA N	0	1	0	1	0	1	1	1	1	1	1	1	1	0	17.98	20	12	6.97	8.71
BIMAN KUMAR BANERJEE	0	0	1	1	1	1	1	0	1	0	1	0	1	1	65.55	4	12	5.42	4.43
SUBRAMANIAM NAIDU L	0	0	1	1	0	1	1	1	1	0	1	0	1	1	49.60	2	1	3.42	4.86
MAMTA SHARMA	0	1	0	0	1	1	1	1	0	0	1	0	1	1	27.77	10	12	4.37	4.14
SARBANI BANDYAPADHYDAY	0	1	0	1	0	1	1	1	1	1	1	1	1	0	44.61	24	24	3.65	4.29
PUNITHA KASI	0	1	1	1	0	1	1	1	1	1	1	1	1	0	24.78	2	6	3.29	5.57
MUTHURAMU P	0	1	1	1	1	1	0	1	1	1	1	1	1	1	65.12	2	3	8.64	4.14

name	Recsyncope	Sex	syncopemorethan5	HUTpositive	Type1HUTTpositive	HUTT2Apositive	HUTT2Bpositive	Type3positive	HUTnegative	Bblockeruse	Metoprololuse	Atenololuse	Bisoprololuse	Tilttrainin	Age	Nosyncope	Duratsymptoms	followmonth	SyncopetoHUTT
LAXMIDEVI	0	1	1	0	1	1	1	1	0	1	1	1	1	1	35.98	3	7	5.59	1.86
VENKATARAMAN G	0	0	1	0	1	1	1	1	0	1	1	1	1	1	59.74	3	6	5.62	8.71
KALYANI DAS	0	1	0	0	1	1	1	1	0	1	1	1	1	0	43.04	10	60	3.61	4.29
MOHAMMED VR	0	0	1	0	1	1	1	1	0	0	1	1	0	1	54.87	3	12	0.62	2.43
BHASWARTI SADHUKHAN	0	1	1	1	0	1	1	1	1	1	1	1	1	0	36.12	2	8	12.62	25.43
MANJU BOWRI	0	1	1	1	0	1	1	1	1	0	0	1	1	0	70.36	2	12	5.59	0.14
KAMAL CHAKRABORTHY	0	0	1	0	1	1	1	1	0	1	1	1	1	1	55.49	2	12	12.39	22.71
SAURAV VERMA	0	0	1	1	0	1	1	1	1	1	1	1	1	0	20.02	2	6	6.74	16.86
UJJAL KUMAR GHOSAL	0	0	1	1	0	1	1	1	1	0	1	1	0	0	50.62	3	60	6.90	11.86
GOPAN NANDY	0	0	1	0	1	1	1	1	0	1	1	1	1	1	61.18	2	2	6.74	3.57
DEBASIS PAL	0	0	0	1	1	0	1	1	1	1	1	1	1	0	32.87	10	60	6.74	12.43
SUNIDRA SARKA	0	1	0	1	0	1	1	1	1	1	1	1	1	1	40.86	7	24	7.03	2.71
AJAYKUMAR KHAMRUI	1	0	1	0	1	1	1	1	0	1	1	1	1	1	48.16	2	10	11.76	3.71
SANDIP NATH	1	0	1	1	0	1	1	1	1	0	0	1	1	0	29.11	2	120	8.57	13.29
SNEHASISH MUKHERJEE	1	1	1	1	0	1	1	1	1	0	0	1	1	0	45.80	2	11	9.89	8.29
GNANASEKARN	1	0	0	0	1	1	1	1	0	0	1	0	1	1	47.68	10	3	8.34	9.00
MOHANA SUNDARAM	1	0	0	1	0	1	1	1	1	1	1	1	1	0	52.86	5	36	3.48	4.43
N MANAVALAN	1	0	0	1	0	1	1	1	1	0	0	1	1	1	71.17	10	1	1.77	0.29
SK MOBARAK	1	0	1	0	1	1	1	1	0	0	1	0	1	1	32.03	3	6	4.53	4.43
ASHOK PRASAD TARUN	1	0	1	1	1	0	1	1	1	1	1	1	1	1	43.03	2	2	2.60	4.43
LYDIA	1	1	0	1	0	1	1	1	1	1	1	1	1	1	24.94	20	4	0.99	10.00
JITENDRA TALUKDAR	1	0	1	0	1	1	1	1	0	0	1	0	1	0	56.03	2	4	6.64	13.00

name	HR baseline	HR peak	HR min	MAP baseline	MAP min	FSchange	Syncopetime	FSslope	dateenroll	dob	datelastsyn	Exertsyncop	Cuttongue	Dejavu	Emotionstress	Headturnin	Unresponse	Confusion	Lightheaded
MOHAMMED TOHID	84	96	84	95	95	-4.99		-0.12	06-May-2010	06-Jan-1962	22-Apr-2010	2	2	2	2	2	2	2	1
SANTIRAM BANERJEE	76	98	82	86	53	4.39	35	0.13	12-Feb-2010	03-Feb-1942	13-Jan-2010	2	2	2	2	2	2	2	1
SHYAM PRASAD SINGH	84	112	45	97	43	10.69	31	0.34	28-Jun-2010	23-Jan-1960	02-Jun-2010	2	2	2	1	2	2	2	1
SRIHARI DAS	50	80	45	70	40	1.88	39	0.05	12-Aug-2010	17-Jul-1964	15-Jul-2010	2	2	2	2	2	2	2	1
RAJAVELU P	80	111	54	76	43	5.54	33	0.17	23-Aug-2010	03-Apr-1959	30-Jul-2010	2	2	2	2	2	1	2	2
KSHUDIRAN JANA	54	59	58	62	62	-0.53		-0.01	01-Aug-2010	01-Jul-1969	01-Jul-2010	2	2	2	2	2	2	2	2
BALAIKUMAR SAHA	72	102	53	89	45	3.55	25	0.18	28-Aug-2010	01-Sep-1957	20-Aug-2010	2	2	2	2	2	2	2	2
PRADIP SARKAR	64	105	52	82	49	0.05	39	0.00	29-May-2010	15-Apr-1974	20-May-2010	1	2	2	2	2	2	2	1
KIRAN DEVI	82	96	75	91	40	8.40	38	0.22	10-Jun-2010	06-Jun-1987	25-May-2010	2	2	2	1	2	2	2	1
NITAI CHANDRA MAJI	71	92	70	85	73	-2.27		-0.06	09-Jun-2010	15-Jul-1965	01-Jun-2010	1	2	2	2	2	2	2	2
MOSTAQUE AHMED	52	70	52	105	97	-3.34		-0.08	15-Sep-2010	12-Dec-1954	01-Sep-2010	2	2	2	2	2	2	2	1
JAGADISH CHANDRA SAHA	80	110	54	99	59	3.41	27	0.14	08-Jun-2010	01-Aug-1960	09-Dec-2009	2	2	2	2	2	2	2	2
SHAIK MAHABOOB SAHEB	74	108	74	97	80	1.59		0.04	01-Jun-2010	01-Jul-1957	28-May-2010	2	2	2	2	2	2	2	1
TULSI DE	73	123	73	92	45	4.19	38	0.11	16-Jun-2010	06-Jun-1943	10-Jun-2010	2	2	2	1	2	2	2	1
NANU TABAURI	116	112	101	101	91	0.65		0.02	26-May-2010	07-Jun-1955	20-May-2010	2	2	2	2	2	2	2	2
RUMIKA BIBI	95	120	95	83	76	3.55		0.09	20-Jun-2010	26-Apr-1994	01-Feb-2010	2	2	2	1	2	2	2	1
LATA KAHAR	76	112	82	71	64	1.12		0.03	17-Sep-2010	01-Apr-1976	12-Sep-2010	1	2	2	2	2	1	2	1
KARUNA SAHA	81	84	64	94	57	3.47	28	0.14	26-Jul-2010	07-Apr-1956	20-Jul-2010	2	2	2	1	2	2	2	1
SIVAPRAKASAMR	86	128	86	73	67	-4.27		-0.11	26-May-2010	18-Feb-1991	10-May-2010	2	2	2	2	2	2	1	2

name	HR baseline	HR peak	HR min	MAP baseline	MAP min	FSchange	Syncope time	FS slope	dateenroll	dob	datelastsyn	Exert syncop	Cut to ngue	Deja vu	Emotionstress	Headturnin	Unresponse	Confusion	Lightheaded
NIBARAN CHANDRA DAS	104	112	103	81	70	-2.90		-0.07	28-Sep-2010	01-Jan-1950	10-Sep-2010	2	2	2	2	2	2	1	1
AKHIL CHANDRA DEBNATH	59	76	42	82	56	9.97	35	0.33	03-Oct-2010	02-Feb-1938	03-Sep-2010	2	2	2	2	2	2	2	2
ANANDA PAUL	66	78	64	74	65	4.60		0.12	03-Jun-2010	06-Jul-1952	01-Jun-2010	2	2	2	2	2	2	2	2
ASHUTOSH PAUL	82	112	82	98	93	1.96		0.05	04-May-2010	06-Jun-1947	04-Jan-2010	2	2	2	1	2	2	2	1
UMASRI ARCHANA	98	120	46	70	53	1.41	30	0.06	04-Jul-2010	25-Nov-1987	30-Jun-2010	2	2	2	2	2	1	2	1
MAYA SAHA	80	92	80	99	79	-1.29		-0.03	09-May-2010	02-Sep-1957	14-Dec-2009	2	2	2	1	2	2	2	2
RAMITA ROY	90	133	90	72	70	-5.02		-0.13	10-May-2010	02-Sep-1989	18-Dec-2009	2	2	2	2	2	2	2	1
PABITRA KUMAR GHOSH	68	105	68	79	48	14.25	32	0.57	02-Nov-2010	01-Feb-1943	01-Nov-2010	2	2	2	2	2	2	2	2
PADMANABHAN N	83	122	75	100	59	5.77	27	0.23	07-Jun-2010	26-May-1969	06-Jun-2010	2	2	2	2	2	2	2	1
EDYFAIRLAND K	86	90	46	92	54	4.18	24	0.21	05-Aug-2010	05-Sep-1985	29-Jul-2010	2	2	2	2	2	2	2	2
ASHIS MANDAL	88	102	42	92	51	4.22	2	2.11	05-Jun-2010	10-Feb-1973	01-Feb-2010	2	2	2	2	2	2	2	1
SWAPAN BASU	101	116	60	112	64	2.53	35	0.08	10-Aug-2010	19-Jan-1960	08-Aug-2010	2	2	2	2	2	2	2	2
PAVITHRA N	76	136	70	75	54	9.64	32	0.32	22-Jun-2010	20-Jan-1993	22-Apr-2010	2	2	2	1	2	2	2	1
BIMAN KUMAR BANERJEE	65	78	60	71	54	1.41	40	0.04	08-Aug-2010	25-Jun-1945	08-Jul-2010	2	2	2	2	2	2	2	2
SUBRAMANIAM NAIDU L	91	106	46	100	64	2.74	24	0.13	08-Oct-2010	07-Jun-1961	04-Sep-2010	2	2	2	2	2	2	2	1
MAMTA SHARMA	96	118	96	95	93	0.92		0.02	10-Sep-2010	06-Apr-1983	12-Aug-2010	2	2	2	2	2	2	2	1
SARBANI BANDYAPADHYDAY	84	103	47	82	52	5.90	28	0.24	01-Oct-2010	05-Jun-1966	01-Sep-2010	1	2	2	2	2	2	2	1
PUNITHA KASI	81	140	60	64	61	4.69	37	0.13	11-Oct-2010	03-Apr-1986	02-Sep-2010	2	2	2	2	2	2	2	1
MUTHURAMU P	48	62		74	56	6.35	34	0.21	02-May-2010	02-Dec-1945	03-Apr-2010	2	2	2	2	2	2	2	1

name	HR baseline	HR peak	HR min	MAP baseline	MAP min	FSchange	Syncopetime	FSslope	dateenroll	dob	datelastsyn	Exertsyncop	Cuttongue	Dejavu	Emotionstress	Headturnin	Unresponse	Confusion	Lightheaded
LAXMIDEVI	90	109	90	96	83	2.70		0.07	02-Aug-2010	20-Jan-1975	20-Jul-2010	1	2	2	2	2	2	2	2
VENKATARAMANG	61	100	61	94	86	1.44		0.04	02-Aug-2010	17-Apr-1951	02-Jun-2010	2	2	2	2	2	2	2	1
KALYANI DAS	94	137	93	84	77	2.73		0.07	01-Oct-2010	01-Jan-1968	01-Sep-2010	2	2	2	1	2	2	2	2
MOHAMMED VR	70	90	70	80	80	-1.14		-0.03	01-Jan-2011	02-Mar-1956	15-Dec-2010	2	2	2	2	2	2	2	1
BHASWARTI SADHUKHAN	72	124	60	81	35	2.69	28	0.11	01-Jan-2010	01-Dec-1974	07-Jul-2009	2	2	2	2	2	2	2	1
MANJU BOWRI	72	120	58	84	40	5.58	38	0.16	03-Aug-2010	05-Sep-1940	02-Aug-2010	2	2	2	1	2	2	2	1
KAMAL CHAKRABORTHY	56	94	56	86	71	0.05		0.00	07-Jan-2010	18-Jul-1955	01-Aug-2009	2	2	2	1	2	2	2	1
SAURAV VERMA	92	129	63	93	52	6.05	28	0.24	29-Jun-2010	06-Jan-1991	03-Mar-2010	2	2	2	1	2	2	2	1
UJJAL KUMAR GHOSAL	71	138	64	101	43	6.69	38	0.19	24-Jun-2010	02-Jun-1960	02-Apr-2010	2	2	2	2	2	2	2	1
GOPAN NANDY	100	112	95	93	86	-0.33		-0.01	29-Jun-2010	07-Nov-1949	04-Jun-2010	2	2	2	2	2	2	2	1
DEBASIS PAL	60	145	40	87	56	3.23	34	0.11	29-Jun-2010	02-Mar-1978	03-Apr-2010	2	2	2	2	2	2	2	2
SUNIDRA SARKA	85	93	60	93	49	5.60	27	0.22	21-Jun-2010	06-Mar-1970	02-Jun-2010	2	2	2	2	2	2	2	1
AJAYKUMAR KHAMRUI	81	117	81	76	69	1.22		0.03	29-Jan-2010	16-Nov-1962	03-Jan-2010	2	2	2	2	2	2	2	2
SANDIP NATH	78	121	70	75	48	3.46	33	0.12	05-May-2010	05-Dec-1981	01-Feb-2010	2	2	2	1	2	2	2	1
SNEHASISH MUKHERJEE	99	122	99	83	56	3.41	35	0.11	24-Mar-2010	26-Mar-1965	25-Jan-2010	2	2	2	2	2	2	2	1
GNANASEKAR N	82	104	82	108	96	1.06		0.03	10-May-2010	10-May-1963	08-Mar-2010	2	2	2	2	2	2	2	1
MOHANA SUNDARAM	93	114	64	123	77	3.38	30	0.14	05-Oct-2010	05-Mar-1958	04-Sep-2010	2	2	2	2	2	2	2	1
N MANAVALAN	77	110	72	83	56	9.79	32	0.33	28-Nov-2010	12-Nov-1939	26-Nov-2010	2	2	2	2	2	2	2	2
SK MOBARAK	83	110	83	92	87	0.56		0.01	04-Sep-2010	03-Jan-1979	04-Aug-2010	2	2	2	2	2	2	2	2
ASHOK PRASAD TARUN	70	122	32	87	45	4.50	38	0.13	02-Nov-2010	02-Jan-1968	02-Oct-2010	2	2	2	2	2	2	2	2
LYDIA	98	120	70	73	38	4.94	29	0.20	20-Dec-2010	04-Feb-1986	11-Oct-2010	2	2	2	2	2	2	2	1
JITENDRA TALUKDAR	76	108	76	103	78	-0.12		0.00	02-Jul-2010	01-Jan-1955	02-Apr-2010	2	2	2	2	2	2	2	2

name	Sweating	Prolongedsitting	Postureonset	Recovery	Palpitations	HRTILT	HR5MIN	HR10MIN	HR15MIN	HR20MIN	HRNITRATE	HR25MIN	HR30MIN	HR35MIN	HR40MIN	HREND	LVIDDbase	LVIDDTILT	LVIDD5MIN
MOHAMMED TOHID	2	2	1	2	2	80	90	92	91	87	88	86	90	94	96	96	43.20	43.20	44.20
SANTIRAM BANERJEE	2	2	1	2	2	82	84	80	82	82	83	90	96	98		98	44.10	35.50	34.10
SHYAM PRASAD SINGH	1	1	1	2	2	76	76	81	78	83	82	93	112			45	34.10	32.10	31.10
SRIHARI DAS	2	2	1	2	2	57	61	61	62	63	63	62	71	80		45	44.60	41.40	41.40
RAJAVELU P	2	2	2	2	1	80	82	78	83	87	82	108	111			54	35.90	35.90	35.80
KSHUDIRAN JANA	2	2	1	2	1	53	51	52	52	51	52	52	54	59	58	58	43.30	43.30	43.30
BALAIKUMAR SAHA	2	1	2	1	2	74	74	75	77	78	102					53	44.10	38.20	38.20
PRADIP SARKAR	2	2	2	2	2	63	66	72	71	75	96	105	99	95		52	41.00	39.90	39.30
KIRAN DEVI	2	2	2	2	2	94	90	91	89	92	95	92	95	96		75	40.10	40.40	39.30
NITAI CHANDRA MAJI	2	2	2	2	2	70	72	80	75	75	83	92	89	92	87	87	44.70	46.40	44.70
MOSTAQUE AHMED	1	2	2	2	2	55	54	53	51	58	61	63	57	70	60	60	42.30	41.80	42.20
JAGADISH CHANDRA SAHA	1	1	2	2	2	84	86	85	86	90	90	110				54	46.90	46.00	45.00
SHAIK MAHABOOB SAHEB	2	2	2	2	2	88	83	85	88	87	86	95	104	108	112	112	51.80	51.80	54.20
TULSI DE	2	2	2	2	1	103	92	93	98	93	99	110	121	123		93	40.00	40.20	39.00
NANU TABAURI	2	2	2	2	2	110	108	104	101	109	101	108	104	108	112	112	41.20	41.20	40.10
RUMIKA BIBI	2	1	2	2	2	89	96	97	94	97	99	107	109	120	120	120	39.90	39.90	39.90
LATA KAHAR	2	1	2	2	2	86	92	82	88	95	83	106	118	112	107	107	37.10	37.20	37.80
KARUNA SAHA	1	1	2	2	2	80	80	79	80	79	84	82				64	42.40	43.00	44.10
SIVAPRAKASAMR	1	2	1	1	2	88	107	106	103	112	103	120	104	112	128	128	36.70	38.50	40.10

name	Sweating	Prolongedsitting	Postureonset	Recovery	Palpitations	HRTILT	HR5MIN	HR10MIN	HR15MIN	HR20MIN	HRNITRATE	HR25MIN	HR30MIN	HR35MIN	HR40MIN	HREND	LVIDDbase	LVIDDTILT	LVIDD5MIN
NIBARAN CHANDRA DAS	1	2	1	2	2	103	105	106	108	105	103	102	106	108	112	112	32.00	32.10	33.90
AKHIL CHANDRA DEBNATH	1	2	1	2	1	58	63	64	62	67	66	67	76			42	37.10	37.10	35.00
ANANDA PAUL	1	1	2	2	1	64	67	64	66	64	64	62	64	67	78	78	40.70	38.90	37.20
ASHUTOSH PAUL	2	2	1	2	1	82	84	84	92	91	96	120	120	112	111	111	38.40	38.60	37.80
UMASRI ARCHANA	2	1	2	1	2	94	101	102	108	105	111	120				46	38.40	39.50	38.90
MAYA SAHA	2	2	2	1	2	83	88	85	81	86	82	88	92	91	85	85	46.40	46.40	47.00
RAMITA ROY	2	2	1	2	2	97	97	107	99	109	96	114	133	126	122	122	43.00	42.40	43.00
PABITRA KUMAR GHOSH	1	2	2	2	1	70	77	78	82	86	84	105				82	45.80	43.60	43.60
PADMANABHAN	2	2	1	2	2	80	73	86	76	75	91	122				80	43.00	43.50	42.40
EDYFAIRLAND K	2	2	2	2	2	87	86	90	85	90	83					46	39.50	40.70	39.50
ASHIS MANDAL	2	1	1	2	1	102										42	45.80	45.10	
SWAPAN BASU	2	2	1	1	2	94	94	95	95	95	95	100	116			60	45.70	45.70	43.20
PAVITHRA N	2	2	2	2	2	85	84	97	95	99	99	128	136			70	38.10	39.80	38.10
BIMAN KUMAR BANERJEE	2	2	2	2	2	64	66	62	64	66	65	73	76	78	60	60	48.30	47.40	46.60
SUBRAMANIAM NAIDU L	2	2	1	2	2	99	98	103	102	106	105					46	41.50	40.60	40.60
MAMTA SHARMA	2	2	2	2	2	101	104	114	117	112	112	112	112	118	112	112	46.60	46.60	44.90
SARBANI BANDYAPADHYDAY	2	1	2	2	2	90	90	95	89	90	95	103				47	41.50	41.50	40.60
PUNITHA KASI	2	1	2	2	2	96	96	100	98	98	109	105	134	140		60	39.80	38.10	38.10
MUTHURAMU P	2	2	2	2	2	52	41	53	52	60	56	62	62			36	41.50	42.30	43.20

name	Sweating	Prolongedsitting	Postureonset	Recovery	Palpitations	HRTILT	HR5MIN	HR10MIN	HR15MIN	HR20MIN	HRNITRATE	HR25MIN	HR30MIN	HR35MIN	HR40MIN	HREND	LVIDDbase	LVIDDTILT	LVIDD5MIN
LAXMIDEVI	1	2	2	2	2	92	93	93	96	96	94	106	100	102	109	109	33.90	32.20	33.90
VENKATARAMANG	2	2	1	2	2	61	64	62	67	65	61	76	65	81	100	100	39.00	39.80	40.60
KALYANI DAS	2	2	1	2	2	93	101	102	109	104	110	122	135	137	130	130	45.70	45.70	44.90
MOHAMMED VR	2	2	2	1	2	74	74	72	76	80	79	88	86	90	88	88	45.70	45.70	44.90
BHASWARTI SADHUKHAN	2	2	1	2	2	76	75	75	81	85	82	124				60	42.30	42.80	42.60
MANJU BOWRI	2	2	1	2	2	80	83	84	80	86	90	100	120	118		58	42.30	42.30	41.50
KAMAL CHAKRABORTHY	2	2	1	2	2	66	71	66	68	71	82	88	94	93	89	89	39.10	39.10	38.60
SAURAV VERMA	1	2	2	2	1	94	110	105	107	107	102	129				63	45.60	44.00	44.20
UJJAL KUMAR GHOSAL	2	1	2	2	2	98	109	105	105	106	106	125	120	138		64	42.20	42.30	42.00
GOPAN NANDY	2	2	1	2	2	98	97	95	96	97	102	102	115	112	112	112	42.00	42.20	42.20
DEBASIS PAL	2	2	2	2	2	80	80	85	78	78	90	120	145			40	44.30	43.20	43.40
SUNIDRA SARKA	2	2	2	2	2	93	84	81	80	86	85	86				60	43.60	42.20	42.20
AJAYKUMAR KHAMRUI	2	1	2	2	2	85	84	84	86	85	109	117	105	98	96	96	45.80	45.80	46.40
SANDIP NATH	2	1	2	2	2	82	81	92	90	92	96	110	121			70	45.70	43.70	43.40
SNEHASISH MUKHERJEE	2	2	1	2	2	85	106	105	108	106	118	115	122	104		104	40.60	41.50	39.80
GNANASEKAR N	2	1	1	2	2	95	94	95	100	98	98	102	104	103	102	102	40.60	41.50	41.00
MOHANA SUNDARAM	2	2	2	2	2	95	92	102	101	95	95	114				64	44.90	43.90	42.70
N MANAVALAN	2	2	2	2	2	84	85	86	91	93	98	100	110			72	45.70	46.60	45.70
SK MOBARAK	2	2	2	2	2	90	79	84	86	82	83	88	96	104	110	110	39.00	39.00	39.80
ASHOK PRASAD TARUN	2	2	2	2	2	91	87	94	97	95	96	111	122	121		32	41.50	38.10	39.00
LYDIA	2	2	2	2	2	101	98	104	100	96	100	120				70	38.70	38.70	38.70
JITENDRA TALUKDAR	2	2	2	2	2	90	76	80	77	79	96	106	110	108	107	107	44.00	43.80	44.10

name	LVIDD10MIN	LVIDD15MIN	LVIDD20MIN	LVIDDNITRATE	LVIDD25MIN	LVIDD30MIN	LVIDD35MIN	LVIDD40MIN	LVIDEND	LVIDSbase	LVIDStilt	LVIDS5MIN	LVIDS10MIN	LVIDS15MIN	LVIDS20MIN	LVIDSNITRATE	LVIDS25MIN	LVIDS30MIN	LVIDS35MIN
MOHAMMED TOHID	42.80	41.10	41.10	41.10	41.10	40.03	41.10	40.03	40.03	28.60	28.60	30.70	28.30	28.40	27.60	28.40	26.80	26.10	26.80
SANTIRAM BANERJEE	33.30	32.80	33.40	33.40	32.20	31.50	32.80		32.80	29.90	23.80	23.10	22.50	21.60	21.80	21.80	22.10	21.50	20.80
SHYAM PRASAD SINGH	31.20	31.30	31.20	29.80	27.20	27.90			27.90	24.30	21.60	20.60	20.80	20.80	19.60	17.60	17.30	16.90	
SRIHARI DAS	40.10	39.40	38.10	38.10	37.50	37.50	36.80		35.60	28.40	27.10	26.50	25.20	23.90	23.30	23.30	23.30	24.60	23.90
RAJAVELU P	34.70	35.40	35.00	35.10	34.90	33.10			33.10	26.50	26.40	26.30	25.10	25.20	25.00	25.30	24.90	23.80	
KSHUDIRAN JANA	43.30	42.00	43.30	43.60	42.70	42.70	40.70	41.40	41.40	25.50	25.50	25.20	25.90	23.90	25.50	25.80	26.50	25.90	24.60
BALAIKUMAR SAHA	37.90	37.90	37.80	36.40					36.40	30.40	26.10	26.10	26.40	26.00	26.30	23.80			
PRADIP SARKAR	39.80	39.80	38.90	40.30	37.80	37.80	37.90		37.90	24.90	24.90	24.70	24.50	24.60	23.80	24.10	24.30	23.00	23.00
KIRAN DEVI	39.30	36.70	36.80	33.70	34.00	33.70	34.80		34.90	30.60	29.10	27.60	26.70	26.60	25.50	24.00	24.40	23.90	24.70
NITAI CHANDRA MAJI	43.00	43.00	43.50	43.50	42.40	43.00	40.10	42.40	42.40	30.40	32.60	31.50	30.90	30.40	29.80	29.80	29.80	29.20	28.10
MOSTAQUE AHMED	42.90	42.10	42.40	41.60	41.60	40.60	40.20	39.70	39.70	28.10	27.40	28.10	28.10	28.80	28.90	28.10	27.20	26.90	26.80
JAGADISH CHANDRA SAHA	46.70	46.00	44.20	50.40	50.40				50.40	35.10	36.30	32.00	32.50	32.00	32.50	36.00	36.00		
SHAIK MAHABOOB SAHEB	51.80	51.90	50.20	49.30	49.80	51.70	50.60	51.00	51.00	38.10	38.10	40.80	40.30	40.60	39.30	37.60	37.60	38.20	39.50
TULSI DE	39.40	39.50	39.50	38.30	37.60	36.90	36.50		36.30	27.90	27.80	26.90	24.80	26.60	26.60	25.00	24.90	24.80	24.00
NANU TABAURI	40.70	41.20	40.10	41.20	40.10	39.50	40.70	40.70	40.70	27.50	28.10	26.90	26.90	27.50	27.50	27.50	26.30	25.80	27.50
RUMIKA BIBI	39.20	38.30	37.60	36.30	37.30	35.00	35.60	33.90	33.90	29.90	29.90	29.80	28.80	28.20	26.80	25.30	25.30	25.00	24.90
LATA KAHAR	37.80	38.40	37.20	36.10	35.60	35.30	34.60	35.50	35.50	25.60	26.30	26.90	27.50	28.10	26.30	25.80	25.00	24.60	23.30
KARUNA SAHA	44.00	43.00	43.00	42.40	37.20				37.20	28.60	29.20	29.20	30.40	30.40	30.60	29.80	23.80		
SIVAPRAKASAMR	38.20	38.90	36.70	35.70	36.20	35.20	36.10	34.70	34.70	23.50	25.50	24.80	25.10	25.20	23.50	24.10	25.20	24.80	25.20

name	LVIDD10MIN	LVIDD15MIN	LVIDD20MIN	LVIDDNITRATE	LVIDD25MIN	LVIDD30MIN	LVIDD35MIN	LVIDD40MIN	LVIDDEND	LVIDSbase	LVIDStilt	LVIDS5MIN	LVIDS10MIN	LVIDS15MIN	LVIDS20MIN	LVIDSNITRATE	LVIDS25MIN	LVIDS30MIN	LVIDS35MIN
NIBARAN CHANDRA DAS	32.50	32.80	32.40	31.00	30.60	29.20	28.60	29.20	29.20	21.10	20.80	21.60	21.60	22.80	22.80	22.30	21.20	20.90	20.10
AKHIL CHANDRA DEBNATH	32.20	32.20	32.40	33.10	33.20	32.80			32.80	27.00	27.00	25.30	20.60	21.30	21.60	21.60	21.60	20.60	
ANANDA PAUL	37.20	37.80	38.90	37.20	38.90	37.80	37.20	37.60	37.60	29.80	26.30	26.30	25.80	26.30	26.90	25.20	26.30	25.20	25.80
ASHUTOSH PAUL	37.50	38.20	37.10	36.90	35.60	35.70	36.00	35.60	35.60	26.10	26.10	26.10	26.70	27.30	26.00	26.20	23.80	23.80	24.70
UMASRI ARCHANA	38.40	39.50	37.20	37.40	37.40				37.40	25.80	25.20	26.30	25.20	26.90	25.80	25.80	24.60		
MAYA SAHA	45.80	48.10	47.00	46.40	47.00	47.00	45.80	46.40	46.40	32.60	32.60	32.10	30.90	33.20	32.60	33.20	33.80	33.80	33.20
RAMITA ROY	41.80	42.40	41.20	41.80	41.20	41.20	39.50	40.70	40.70	25.10	26.90	26.30	25.80	26.30	26.90	25.80	25.20	26.30	25.20
PABITRA KUMAR GHOSH	43.20	42.60	41.80	42.40	40.90				40.90	31.50	30.20	29.70	29.20	28.50	28.10	28.10	22.30		
PADMANABHAN N	42.40	41.20	41.80	42.40	37.70				37.70	28.60	28.10	27.50	28.60	26.90	26.90	27.50	22.90		
EDYFAIRLAND K	40.10	39.50	38.40	36.70					36.70	26.30	27.50	26.30	26.90	25.20	25.20	22.90			
ASHIS MANDAL									45.10	32.60	30.20								
SWAPAN BASU	43.20	44.00	44.00	44.00	43.20	44.90			44.90	32.20	32.20	30.50	28.80	29.60	29.60	30.50	29.60	30.50	
PAVITHRA N	38.10	37.30	37.30	36.40	36.40	36.40			36.40	26.70	26.30	26.30	25.40	24.60	24.60	23.70	23.70	22.00	
BIMAN KUMAR BANERJEE	46.60	47.40	46.60	46.60	46.00	45.90	46.40	46.00	46.00	30.50	30.50	29.60	31.30	30.50	29.60	29.60	28.60	28.70	29.20
SUBRAMANIAM NAIDU L	39.80	40.60	39.00	40.60					40.60	27.10	27.10	26.30	26.30	26.30	25.40	25.40			
MAMTA SHARMA	44.00	43.20	44.00	44.00	43.20	43.20	44.00	43.20	43.20	28.80	28.80	26.30	26.30	25.40	26.30	26.30	26.30	27.10	26.30
SARBANI BANDYAPADHYDAY	39.80	39.80	40.60	40.60	39.90				39.90	27.10	26.30	24.60	23.70	24.60	24.60	24.60	23.70		
PUNITHA KASI	40.60	39.80	39.00	39.00	38.10	37.30	37.30		37.30	26.30	25.40	24.60	26.30	25.40	25.40	25.40	24.60	23.60	22.90
MUTHURAMU P	42.30	41.50	41.50	40.60	40.60	41.40			41.40	28.80	28.80	28.80	28.80	26.30	27.10	26.30	26.30	26.10	

name	LVIDD10MIN	LVIDD15MIN	LVIDD20MIN	LVIDDNITRATE	LVIDD25MIN	LVIDD30MIN	LVIDD35MIN	LVIDD40MIN	LVIDDEND	LVIDSbase	LVIDSilt	LVIDS5MIN	LVIDS10MIN	LVIDS15MIN	LVIDS20MIN	LVIDSNITRATE	LVIDS25MIN	LVIDS30MIN	LVIDS35MIN
LAXMIDEVI	33.90	33.00	32.20	31.30	32.20	33.00	32.20	31.30	31.30	22.90	22.90	22.90	23.70	21.20	21.20	20.30	21.20	21.20	21.20
VENKATARAMANG	40.60	41.10	40.60	41.50	40.60	40.60	40.60	39.80	39.80	27.90	28.40	27.10	28.80	29.60	28.80	30.50	29.60	28.80	28.80
KALYANI DAS	44.90	44.90	44.00	44.90	43.20	43.20	44.90	44.90	44.90	27.10	26.30	26.30	26.30	25.40	25.40	25.40	23.70	23.70	25.40
MOHAMMED VR	45.70	44.90	44.00	44.90	45.70	44.90	44.00	44.00	44.00	29.60	29.60	29.60	30.50	30.50	28.80	30.50	30.50	30.50	29.00
BHASWARTI SADHUKHAN	41.50	41.50	40.90	41.50	40.60				40.60	27.60	27.60	27.40	26.30	26.30	26.30	27.10	25.40		
MANJU BOWRI	40.60	40.60	41.50	39.80	39.80	39.80	39.80		39.80	26.70	26.00	25.40	24.60	24.60	25.40	24.60	23.70	22.90	22.90
KAMAL CHAKRABORTHY	38.60	38.10	39.10	36.10	37.00	37.00	37.70	38.00	38.00	27.70	27.90	28.50	28.50	27.60	27.90	25.90	26.20	26.70	26.20
SAURAV VERMA	44.00	44.50	43.80	44.50	43.80				43.80	31.70	30.40	29.70	29.90	29.10	28.60	29.00	27.80		
UJJAL KUMAR GHOSAL	42.10	40.80	41.90	41.50	41.50	41.00	41.30		41.30	29.80	29.80	29.60	29.00	28.20	28.80	27.90	29.80	27.10	26.40
GOPAN NANDY	41.00	41.00	40.90	41.60	40.80	41.30	41.10	41.80	41.80	29.00	29.00	29.10	28.60	28.50	28.80	29.00	28.20	28.40	27.90
DEBASIS PAL	43.20	42.80	43.20	43.20	43.20	43.10			43.10	29.80	28.80	29.00	28.20	28.00	28.00	28.00	27.60	27.60	
SUNIDRA SARKA	42.00	42.40	41.80	41.80	41.80				41.80	30.50	29.40	29.00	28.60	28.70	28.00	28.00	26.90		
AJAYKUMAR KHAMRUI	45.80	44.10	45.80	44.10	41.80	41.80	42.40	42.40	42.40	32.10	32.10	32.10	31.50	30.90	32.60	29.80	28.10	28.60	28.60
SANDIP NATH	42.80	43.30	44.00	42.50	43.20	41.50			41.50	27.90	28.00	27.80	26.00	26.30	26.30	25.90	25.90	23.90	
SNEHASISH MUKHERJEE	40.60	39.80	41.50	41.50	40.60	39.00			39.00	24.60	25.40	24.60	23.70	24.60	25.40	24.60	23.70	22.30	
GNANASEKAR N	40.80	40.80	40.00	39.50	40.70	39.80	41.00	39.50	39.50	28.80	29.60	28.90	27.70	27.70	27.10	26.80	28.60	27.70	28.90
MOHANA SUNDARAM	43.90	42.30	43.10	42.10	42.10				42.10	30.10	29.20	29.10	29.20	27.10	28.50	26.30	26.80		
N MANAVALAN	46.60	44.90	45.70	45.70	43.30	43.00			43.00	30.30	30.50	29.60	29.60	29.60	29.60	29.60	26.60	24.30	
SK MOBARAK	39.80	40.60	39.80	39.00	38.10	38.10	39.00	38.10	38.10	25.40	26.30	26.30	26.30	27.90	27.10	26.30	24.60	25.40	25.40
ASHOK PRASAD TARUN	37.30	37.30	36.40	37.30	36.40	36.40	35.60		35.60	24.60	22.90	23.70	22.00	21.20	21.20	22.00	21.20	21.20	19.50
LYDIA	37.70	37.80	37.80	36.90	36.90				36.90	25.30	25.30	24.60	24.60	23.40	23.40	23.00	22.30		
JITENDRA TALUKDAR	44.00	43.70	43.50	43.20	44.00	43.00	43.20	43.20	43.20	30.40	29.90	30.20	30.20	30.00	29.90	29.90	29.90	28.90	29.90

name	LVIDS 40MIN	LVIDSEND	EFENDTIME	MAPbase	MAPtilt	MAP5MIN	MAP10MIN	MAP15MIN	MAP20MIN	MAPNITRATE	MAP25MIN	MAP30MIN	MAP35MIN	MAP40MIN	MAPEND	FSbaseline	FStilt	FS5min	FS10min
MOHAMMED TOHID	28.50	28.50	40.00	95	96	97	100	95	99	96	99	98	95	97	97	33.80	33.80	30.54	33.88
SANTIRAM BANERJEE		20.80	35.00	86	90	85	85	86	86	90	90	60			53	32.20	32.96	32.26	32.43
SHYAM PRASAD SINGH		16.90	31.00	97	105	97	100	95	100	99	103	67			43	28.74	32.71	33.76	33.33
SRIHARI DAS		22.00	39.00	70	70	75	74	72	75	75	72	68	57		40	36.32	34.54	35.99	37.16
RAJAVELU P		22.60	32.00	76	75	81	86	86	81	81	76	79			43	26.18	26.46	26.54	27.67
KSHUDIRAN JANA	24.60	24.60	40.00	62	86	74	74	73	72	72	76	74	71	67	67	41.11	41.11	41.80	40.18
BALAIKUMAR SAHA		23.80	20.00	89	92	92	92	92	93	96					45	31.07	31.68	31.68	30.34
PRADIP SARKAR		23.00	35.00	82	104	97	82	80	82	80	78	75	59		49	39.27	37.59	37.15	38.44
KIRAN DEVI		23.70	38.00	91	91	86	83	84	85	86	83	85	82		40	23.69	27.97	29.77	32.06
NITAI CHANDRA MAJI	29.80	29.80	40.00	85	96	89	81	91	83	89	89	80	75	73	73	31.99	29.74	29.53	28.14
MOSTAQUE AHMED	27.70	27.70	40.00	105	101	97	97	101	102	102	102	88	89	97	97	33.57	34.45	33.41	34.50
JAGADISH CHANDRA SAHA		36.00	25.00	99	128	142	134	125	130	138	85				59	25.16	21.09	28.89	30.41
SHAIK MAHABOOB SAHEB	36.70	36.70	40.00	97	101	102	102	98	98	102	95	91	83	80	80	26.45	26.45	24.72	22.20
TULSI DE		23.80	38.00	92	90	91	87	83	75	83	84	86	83		45	30.25	30.85	31.03	37.06
NANU TABAURI	26.90	26.90	40.00	101	112	105	105	105	101	101	108	104	91	94	94	33.25	31.80	32.92	33.91
RUMIKA BIBI	24.20	24.20	40.00	83	89	91	76	78	78	79	84	81	73	88	88	25.06	25.06	25.31	26.53
LATA KAHAR	24.10	24.10	40.00	71	71	71	79	58	64	64	63	64	67	74	74	31.00	29.30	28.84	27.25
KARUNA SAHA		23.80	25.00	94	89	84	84	86	88	85	79				57	32.55	32.09	33.79	30.91
SIVAPRAKASAMR	23.70	23.70	40.00	73	72	71	75	70	74	74	73	67	67	70	70	35.97	33.77	38.15	34.29

name	LVIDS40MIN	LVIDSEND	EFENDTIME	MAPbase	MAPtilt	MAP5MIN	MAP10MIN	MAP15MIN	MAP20MIN	MAPNITRATE	MAP25MIN	MAP30MIN	MAP35MIN	MAP40MIN	MAPEND	FSbaseline	FStilt	FS5min	FS10min
NIBARAN CHANDRA DAS	20.10	20.10	40.00	81	81	81	80	78	80	78	85	78	73	70	70	34.06	35.20	36.28	33.54
AKHIL CHANDRA DEBNATH		20.60	30.00	82	82	84	85	85	84	82	81	83			56	27.22	27.22	27.71	36.02
ANANDA PAUL	25.80	25.80	40.00	74	74	73	75	70	70	70	70	69	65	65	65	26.78	32.39	29.30	30.65
ASHUTOSH PAUL	23.50	23.50	40.00	98	99	99	100	99	101	98	89	89	93	93	93	32.03	32.38	30.95	28.80
UMASRI ARCHANA		24.60	25.00	70	69	69	70	68	69	68	66				53	32.81	36.20	32.39	34.38
MAYA SAHA	33.20	33.20	40.00	99	92	93	92	94	91	93	89	87	89	79	79	29.74	29.74	31.70	32.53
RAMITA ROY	25.80	25.80	40.00	72	73	74	75	82	86	83	79	75	69	70	70	41.63	36.56	38.84	38.28
PABITRA KUMAR GHOSH		22.30	25.00	79	87	83	77	78	80	79	79				48	31.22	30.73	31.88	32.41
PADMANABHAN N		22.90	25.00	100	100	94	103	106	103	107	89				59	33.49	35.40	35.14	32.55
EDYFAIRLANDK		22.90	20.00	92	92	91	89	88	87	83					54	33.42	32.43	33.42	32.92
ASHIS MANDAL		30.20	2.00	92	75										51	28.82	33.04		
SWAPAN BASU		30.50	30.00	112	115	115	117	116	113	108	103	105			64	29.54	29.54	29.40	33.33
PAVITHRA N		22.00	30.00	75	87	87	77	77	80	86	92	90			54	29.92	33.92	30.97	33.33
BIMAN KUMAR BANERJEE	28.40	28.40	40.00	71	68	67	70	68	70	70	71	62	52		54	36.85	35.65	36.48	32.83
SUBRAMANIAM NAIDU L		25.40	21.00	100	103	111	114	108	105	97					64	34.70	33.25	35.22	33.92
MAMTA SHARMA	26.30	26.30	40.00	95	93	91	90	85	97	92	96	96	92	95	95	38.20	38.20	41.43	40.23
SARBANI BANDYAPADHYDAY		23.70	25.00	82	85	74	74	78	78	72	79				52	34.70	36.63	39.41	40.45
PUNITHA KASI		22.90	35.00	64	77	80	78	77	77	78	76	77	72		61	33.92	33.33	35.43	35.22
MUTHURAMU P		26.10	30.00	74	77	82	81	79	82	75	68	65			56	30.60	31.91	33.33	31.91

name	LVIDS40MIN	LVIDSEND	EFENDTIME	MAPbase	MAPtilt	MAP5MIN	MAP10MIN	MAP15MIN	MAP20MIN	MAPNITRATE	MAP25MIN	MAP30MIN	MAP35MIN	MAP40MIN	MAPEND	FSbaseline	FStilt	FS5min	FS10min
LAXMIDEVI	20.30	20.30	40.00	96	91	88	87	84	86	84	83	84	83	83	83	32.45	28.88	32.45	30.09
VENKATARAMANG	27.90	27.90	40.00	94	93	92	88	89	91	97	97	97	90	86	86	28.46	28.64	33.25	29.06
KALYANI DAS	25.40	25.40	40.00	84	84	84	86	85	87	89	89	81	83	77	77	40.70	42.45	41.43	41.43
MOHAMMED VR	29.00	29.00	40.00	80	83	84	83	87	88	88	75	76	71	85	85	35.23	35.23	34.08	33.26
BHASWARTI SADHUKHAN		25.40	25.00	81	83	82	81	79	75	76	82				35	34.75	35.51	35.68	36.63
MANJU BOWRI		22.90	35.00	84	85	91	91	86	84	91	87	89	87		40	36.88	38.53	38.80	39.41
KAMAL CHAKRABORTHY	26.90	26.90	40.00	86	85	78	86	84	90	90	76	83	84	71	71	29.16	28.64	26.17	26.17
SAURAV VERMA		27.80	25.00	93	88	87	87	89	85	91	77				52	30.48	30.91	32.81	32.05
UJJAL KUMAR GHOSAL		26.40	35.00	101	100	102	105	106	101	101	99	99	79		43	29.38	29.55	29.52	31.12
GOPAN NANDY	29.00	29.00	40.00	93	94	92	90	88	86	90	93	95	97	95	95	30.95	31.28	31.04	30.24
DEBASIS PAL		27.60	30.00	87	89	87	93	81	81	81	75	68			56	32.73	33.33	33.18	34.72
SUNIDRA SARKA		26.90	25.00	93	80	87	92	84	91	86	86				49	30.05	30.33	31.28	31.90
AJAYKUMAR KHAMRUI	29.20	29.20	40.00	76	83	80	80	79	78	91	69	69	76	74	74	29.91	29.91	30.82	31.22
SANDIP NATH		23.90	30.00	75	71	68	67	69	70	76	69	67			48	38.95	35.93	35.94	39.25
SNEHASISH MUKHERJEE		22.30	30.00	83	87	97	88	89	86	95	92	89			56	39.41	38.80	38.19	41.63
GNANASEKAR N	27.60	27.60	40.00	108	115	104	98	96	96	98	98	98	97	96	96	29.06	28.67	29.51	32.11
MOHANA SUNDARAM		26.80	25.00	123	113	113	110	113	105	113	106				77	32.96	33.49	31.85	33.49
N MANAVALAN		24.30	30.00	83	78	73	75	79	75	76	73	69			56	33.70	34.55	35.23	36.48
SK MOBARAK	24.60	24.60	40.00	92	92	99	102	100	100	99	94	96	92	87	87	34.87	32.56	33.92	33.92
ASHOK PRASAD TARUN		19.50	35.00	87	90	84	88	90	91	82	83	78	78		45	40.72	39.90	39.23	41.02
LYDIA		22.30	25.00	73	76	84	79	81	78	81	80				38	34.63	34.63	36.43	34.75
JITENDRA TALUKDAR	29.90	29.90	40.00	103	99	97	93	95	90	87	86	82	85	78	78	30.91	31.74	31.52	31.36

name	FS15min	FS20min	FSnitrate	FS25min	FS30min	FS35min	FS40min	FSend
MOHAMMED TOHID	30.90	32.85	30.90	34.79	34.80	34.79	28.80	28.80
SANTIRAM BANERJEE	34.15	34.73	34.73	31.37	31.75	36.59		36.59
SHYAM PRASAD SINGH	33.55	37.18	40.94	36.40	39.43			39.43
SRIHARI DAS	39.34	38.85	38.85	37.87	34.40	35.05		38.20
RAJAVELU P	28.81	28.57	27.92	28.65	28.10			31.72
KSHUDIRAN JANA	43.10	41.11	40.83	37.94	39.34	39.56	40.58	40.58
BALAIKUMAR SAHA	31.40	30.42	34.62					34.62
PRADIP SARKAR	38.19	38.82	40.20	35.71	39.15	39.31		39.31
KIRAN DEVI	27.52	30.71	28.78	28.24	29.08	29.02		32.09
NITAI CHANDRA MAJI	29.30	31.49	31.49	29.72	32.09	29.93	29.72	29.72
MOSTAQUE AHMED	31.59	31.84	32.45	34.62	33.74	33.33	30.23	30.23
JAGADISH CHANDRA SAHA	30.43	26.47	28.57	28.57				28.57
SHAIK MAHABOOB SAHEB	21.77	21.71	23.73	24.50	26.11	21.94	28.04	28.04
TULSI DE	32.66	32.66	34.73	33.78	32.79	34.25		34.44
NANU TABAURI	33.25	31.42	33.25	34.41	34.68	32.43	33.91	33.91
RUMIKA BIBI	26.37	28.72	30.30	32.17	28.57	30.06	28.61	28.61
LATA KAHAR	26.82	29.30	28.53	29.78	30.31	32.66	32.11	32.11
KARUNA SAHA	29.30	28.84	29.72	36.02				36.02
SIVAPRAKASAM R	35.22	35.97	32.49	30.39	29.55	30.19	31.70	31.70
NIBARAN CHANDRA DAS	30.49	29.63	28.06	30.72	28.42	29.72	31.16	31.16
AKHIL CHANDRA DEBNATH	33.85	33.33	34.74	34.94	37.20			37.20
ANANDA PAUL	30.42	30.85	32.26	32.39	33.33	30.65	31.38	31.38
ASHUTOSH PAUL	28.53	29.92	29.00	33.15	33.33	31.39	33.99	33.99
UMASRI ARCHANA	31.90	30.65	31.02	34.22				34.22
MAYA SAHA	30.98	30.64	28.45	28.09	28.09	27.51	28.45	28.45
RAMITA ROY	37.97	34.71	38.28	38.83	36.17	36.20	36.61	36.61
PABITRA KUMAR GHOSH	33.10	32.78	33.73	45.48				45.48
PADMANABHAN N	34.71	35.65	35.14	39.26				39.26
EDYFAIRLAND K	36.20	34.38	37.60					37.60
ASHIS MANDAL								33.04

name	FS15min	FS20min	FSnitrate	FS25min	FS30min	FS35min	FS40min	FSend
SWAPAN BASU	32.73	32.73	30.68	31.48	32.07			32.07
PAVITHRA N	34.05	34.05	34.89	34.89	39.56			39.56
BIMAN KUMAR BANERJEE	35.65	36.48	36.48	37.83	37.47	37.07	38.26	38.26
SUBRAMANIAM NAIDU L	35.22	34.87	37.44					37.44
MAMTA SHARMA	41.20	40.23	40.23	39.12	37.27	40.23	39.12	39.12
SARBANI BANDYAPADHYDAY	38.19	39.41	39.41	40.60				40.60
PUNITHA KASI	36.18	34.87	34.87	35.43	36.73	38.61		38.61
MUTHURAMU P	36.63	34.70	35.22	35.22	36.96			36.96
LAXMIDEVI	35.76	34.16	35.14	34.16	35.76	34.16	35.14	35.14
VENKATARAMAN G	27.98	29.06	26.51	27.09	29.06	29.06	29.90	29.90
KALYANI DAS	43.43	42.27	43.43	45.14	45.14	43.43	43.43	43.43
MOHAMMED VR	32.07	34.55	32.07	33.26	32.07	34.09	34.09	34.09
BHASWARTI SADHUKHAN	36.63	35.70	34.70	37.44				37.44
MANJU BOWRI	39.41	38.80	38.19	40.45	42.46	42.46		42.46
KAMAL CHAKRABORTHY	27.56	28.64	28.25	29.19	27.84	30.50	29.21	29.21
SAURAV VERMA	34.61	34.70	34.83	36.53				36.53
UJJAL KUMAR GHOSAL	30.88	31.26	32.77	28.19	33.90	36.08		36.08
GOPAN NANDY	30.49	29.58	30.29	30.88	31.23	32.12	30.62	30.62
DEBASIS PAL	34.58	35.19	35.19	36.11	35.96			35.96
SUNIDRA SARKA	32.31	33.01	33.01	35.65				35.65
AJAYKUMAR KHAMRUI	29.93	28.82	32.43	32.78	31.58	32.55	31.13	31.13
SANDIP NATH	39.26	40.23	39.06	40.05	42.41			42.41
SNEHASISH MUKHERJEE	38.19	38.80	40.72	41.63	42.82			42.82
GNANASEKAR N	32.11	32.25	32.15	29.73	30.40	29.51	30.13	30.13
MOHANA SUNDARAM	35.93	33.87	37.53	36.34				36.34
N MANAVALAN	34.08	35.23	35.23	38.57	43.49			43.49
SK MOBARAK	31.28	31.91	32.56	35.43	33.33	34.87	35.43	35.43
ASHOK PRASAD TARUN	43.16	41.76	41.02	41.76	41.76	45.22		45.22
LYDIA	38.10	38.10	37.67	39.57				39.57
JITENDRA TALUKDAR	31.35	31.26	30.79	32.05	32.79	30.79	30.79	30.79