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Abnormalities in Cardiac Mechanics and Autonomic Function in People With Syncope Using Pulse Wave Doppler Echocardiography – A Pilot Study

Walter Uronu[1*], Dr Mukund A Prabhu[2], Dr Krishnananda Nayak[3]

[1]*Department CVT, MCHP, Manipal, Karnataka, India.*

[2]*Department of Cardiology, KMC Manipal, Karnataka, India.*

[3]*Department of CVT, MCHP, Manipal, Karnataka, India.*

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***Correspondence:** Mr Walter Uronu Department CVT, MCHP, Manipal, Karnataka, India. Email: uronuwalter10@gmail.com*

ABSTRACT

Background: Vasovagal syncope is a type of reflex syncope characterised by a transient loss of consciousness and postural tone due to temporary, self-limiting global cerebral hypoperfusion. Syncope is broadly classified into four categories: reflex (neurally mediated), orthostatic hypotension, cardiac arrhythmias, and structural cardiac causes. This study aimed to evaluate the extent of reduction in cardiac output and stroke volume associated with syncope.

Methods: A prospective observational case-control study of 21 participants, comprising 11 males (52.3%) and 10 females (47.7%), who underwent head-up tilt table (HUTT) testing and echocardiographic evaluation for syncope at Kasturba Medical College Hospital, Manipal, India, between July 2023 and April 2024. Participants were categorised into syncope and non-syncope groups based on HUTT outcomes.

Results: Of the 21 participants, 5 developed syncope during the HUTT test, while 16 did not. Participants with syncope demonstrated a significant reduction in cardiac output and stroke volume, associated with a decrease in blood pressure (<90/50 mmHg). Systemic vascular resistance increased significantly during the syncope stage, likely due to baroreflex-mediated vasoconstriction in the splanchnic and skeletal muscle vascular beds.

Conclusion: The findings suggest that a reduction in cardiac output secondary to hypotension is the primary determinant of syncope. An increase in systemic vascular resistance appears to act as a compensatory mechanism to prevent a further decline in cardiac output. The combined use of HUTT and echocardiography is valuable in assessing cardiac mechanics during syncope.

Trial registration: The trial was registered under CTRI (Registration No: CTRI/2023/07/055804).

Keywords: Syncope, Head-up tilt test, Pulse wave Doppler echocardiography, Cardiac output, Blood pressure, Systemic vascular resistance.

INTRODUCTION

Vasovagal syncope is a type of reflex syncope characterised by a transient loss of consciousness and postural tone due to temporary, self-limiting global cerebral hypoperfusion. The term “vasovagal” was first introduced in 1907 by Sir William Gowers to describe a

constellation of vagal symptoms associated with vasomotor disturbances. In 1932, Sir Thomas Lewis defined vasovagal syncope as a decrease in blood pressure accompanied by a slowing of the ventricular rate[1,2].

Syncope can be categorised into four types: (1) reflex (neurally mediated) syncope (60%), (2) orthostatic hypotension (15%), (3) cardiac arrhythmias (10%), and (4) structural cardiac causes (5%).[1,3]

The mechanism of vasovagal syncope typically occurs upon standing, when there is pooling of venous blood in the lower extremities (below the abdominal organs), estimated at approximately 500–1000 mL. This results in a sudden reduction in venous return, leading to decreased ventricular filling, reduced stroke volume, and consequently reduced cardiac output, ultimately causing cerebral hypoperfusion and loss of consciousness.[6,7] The associated fall in blood pressure is mediated by activation of the parasympathetic nervous system.[6]

The head-up tilt test (HUTT) allows reproduction of neurally mediated reflexes under controlled laboratory conditions and is indicated in patients with suspected vasovagal syncope not confirmed by initial evaluation.[8] During the HUTT procedure, four phases of syncope can be observed based on haemodynamic changes: (1) early reduction in venous return during standing, (2) circulatory adaptation, (3) hypotension leading to syncope, and (4) recovery.[5]

Based on haemodynamic responses, three types of syncope are typically identified during HUTT: (1) cardioinhibitory response, (2) vasodepressor response, and (3) mixed response.[5] Despite extensive research, the primary determinant of hypotension in vasovagal syncope remains controversial, likely due to limited studies evaluating haemodynamic changes across different classifications of vasovagal syncope.[5,6]

Previous studies have utilised impedance cardiography (ICG), Fick's method, and finger blood pressure monitoring techniques to measure cardiac output during syncope.[5-7] In contrast, the present study employs pulse wave (PW) Doppler echocardiography to evaluate cardiac output.

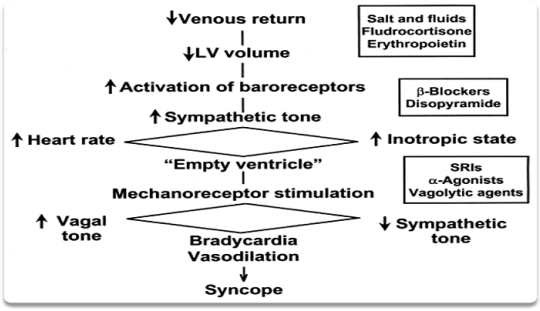

Fig 1.1: Syncope mechanism

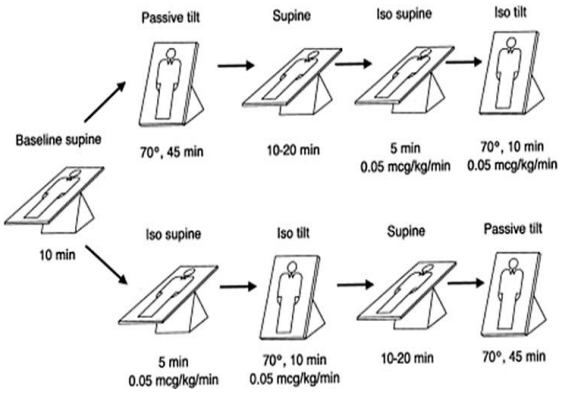

Fig 1.2: HUTT test protocol

METHODS

Study Design

This was a prospective observational case–control study conducted at a single centre, Kasturba Medical College Hospital, Manipal, India, between July 2023 and April 2024. The study protocol was approved by the Institutional Ethics Committee prior to commencement. Written informed consent was obtained from all participants before enrolment.

Study Population

Participants aged ≥18 years with a history of vasovagal syncope were evaluated in the Department of Cardiology at Kasturba Medical College Hospital, Manipal. All participants underwent baseline investigations, including electrocardiography (ECG), echocardiography (ECHO), and routine blood tests prior to the head-up tilt table (HUTT) test to exclude cardiac causes of syncope. Brain computed tomography (CT) or magnetic resonance imaging (MRI) was advised in cases with suspected seizure disorders.

Inclusion Criteria

- Age ≥18 years
- History suggestive of vasovagal syncope
- Referred for head-up tilt table (HUTT) testing
- Willingness to provide informed consent

Exclusion Criteria

- Known structural heart disease
- Documented cardiac arrhythmias
- Neurological disorders (e.g., epilepsy)
- Patients on medications affecting blood pressure or autonomic function
- Incomplete clinical or investigational data

Instruments and echocardiographic views used:

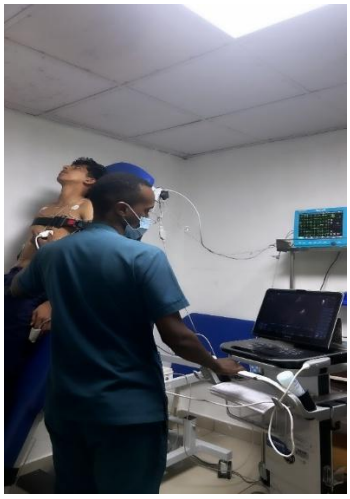

Fig 1.3: Echocardiogram, BP, ECG and HUTT test combined

Fig 1.4: Vivid Iq (GE) version: 204

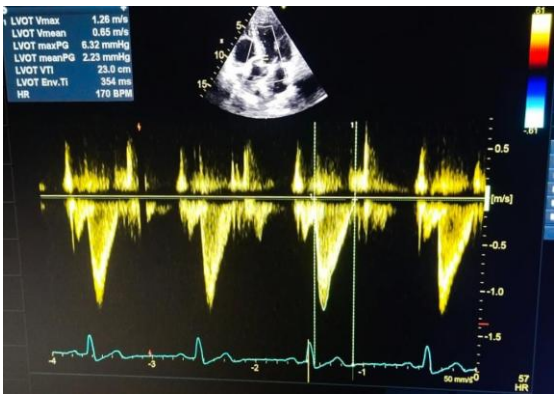

Fig 1.5: A5C view for measuring LVOT VTI and SET

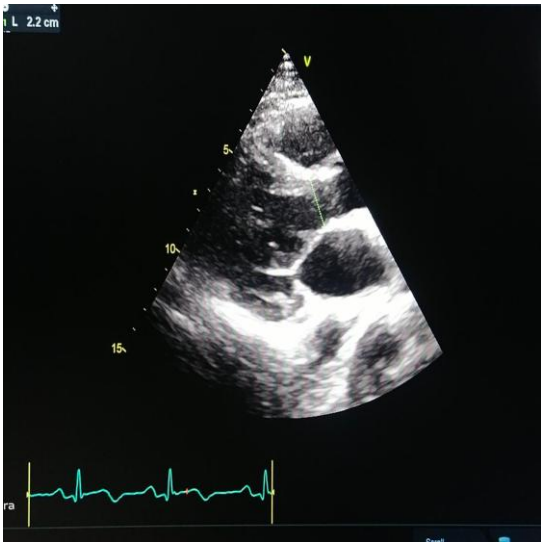

Fig 1.6: PLAX view for measuring LVOT diameter

Clinical data

Clinical data comprised the participant's age, gender, height, weight, BSA, BP, and HR.

Data analysis

All data were entered and analysed using EZR software. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages.

The Student's *t*-test was used to compare continuous variables between two groups (with and without adverse outcomes) and to determine the mean differences between independent groups. One-way analysis of variance (ANOVA) was used for comparisons across multiple

groups. The Chi-square test was applied to assess associations between categorical variables.

A p-value <0.05 was considered statistically significant.

RESULTS

The study included 21 participants, of whom 11 (52.3%) were male, and 10 (47.7%) were female. The mean age, height, weight, and body surface area (BSA) were 38.33 ± 17.94 years, 161.57 ± 10.44 cm, 59.52 ± 10.04 kg, and 1.64 ± 0.18 m², respectively.

Among the participants, 16 (76%) did not develop syncope, while 5 (23.8%) developed syncope during the HUTT test. Of those with syncope, 4 (19%) exhibited a type 1 (vasodepressor) response and 1 (4.8%) exhibited a type 2 (cardioinhibitory) response.

During the baseline stage of the HUTT test, statistically significant differences were observed in diastolic blood pressure (DBP) and mean arterial pressure (MAP), with lower values in the syncope group compared to the non-syncope group. The mean DBP values were 62.60 ± 5.81 mmHg and 75.25 ± 11.75 mmHg for syncope and non-syncope groups, respectively. Similarly, the mean MAP values were 78.37 ± 7.66 mmHg and 90.22 ± 11.68 mmHg, respectively.

During the pre-syncope stage, DBP and MAP demonstrated statistically significant changes. In the non-syncope group, DBP and MAP were maintained or showed minimal increase compared to baseline values, with mean DBP and MAP of 81.06 ± 7.97 mmHg and 96.61 ± 8.92 mmHg, respectively.

During the syncope stage, participants who developed syncope showed a significant reduction in multiple HUTT parameters, including heart rate (HR), stroke volume (SV), cardiac output (CO), systolic blood pressure (SBP), DBP, MAP, cardiac power output (CPO), and cardiac index (C.I), compared to the pre-syncope stage. The mean values were 59.00 ± 9.67 bpm, 27.02 ± 4.19 mL/beat, 1.60 ± 0.38 L/min, 85.00 ± 3.53 mmHg, 42.80 ± 7.01 mmHg, 56.72 ± 4.84 mmHg, 0.20 ± 0.04 Watts, and 0.94 ± 0.24 L/min/m², respectively.

In contrast, non-syncope participants demonstrated statistically significant but less pronounced changes during the same stage, with slight reductions in systolic ejection time (SET), HR, SV, CO, SBP, DBP, MAP, CPO, and C.I. The mean values were 216.69 ± 15.80 ms, 80.13 ± 18.40 bpm, 33.95 ± 3.98 mL/beat, 2.69 ± 0.57 L/min, 112.13 ± 12.27 mmHg, 73.38 ± 7.99 mmHg, 86.16 ± 8.63 mmHg, 0.50 ± 0.10 Watts, and 1.68 ± 0.40 L/min/m², respectively.

During the recovery stage, no statistically significant differences were observed in HUTT parameters for either group. The values returned to levels comparable to baseline in both syncope and non-syncope participants.

Table 1: Demographic parameters of people with syncope.

Variables	Mean ± SD
Age	38.33 ± 17.94
Height (cm)	161.57 ± 10.44
Weight (kg)	59.52 ± 10.04
BSA (m²)	1.64 ± 0.18

Table 2: Clinical presentation parameters showing the distribution of two groups between nonsyncope and syncope people with their type of syncope.

Variables		Frequency (Percentage)
No syncope		16 (76%)
Syncope		5 (23.8%)
Syncope Type	1	4 (19%)
	2	1 (4.8%)

Table 3: Correlations of Baseline HUTT test parameters between syncope and non-syncope people.

Parameters	Mean ± SD		P value	
	Positive test N= (5)	Negative test N= (16)	Positive test N = (5)	Negative test N = (16)
Systolic ejection time (msec)	275.00 ± 26.92	282.81 ± 14.88	0.564	0.410
LVOT Area (cm^2)	2.96 ± 0.40	3.12 ± 0.35	0.449	0.390
LVOT VTI (cm)	18.66 ± 0.56	18.60 ± 0.92	0.878	0.905
HR (bpm)	70.20 ± 10.30	72.50 ± 15.30	0.709	0.759
SV (mL/beat)	55.31 ± 8.00	58.01 ± 5.15	0.509	0.381
CO (L/min)	3.88 ± 0.83	4.18 ± 0.86	0.504	0.496
SBP (mmHg)	110.40 ± 12.17	120.63 ± 15.04	0.160	0.184
DBP (mmHg)	62.60 ± 5.81	75.25 ± 11.75	0.006	0.034
MAP (mmHg)	78.37 ± 7.66	90.22 ± 11.68	0.024	0.048
SVR (Woods unit)	20.59 ± 2.62	22.86 ± 7.92	0.337	0.543
CPO (Watts)	0.68 ± 0.21	0.83 ± 0.17	0.216	0.132
C.I ($L/min/m^2$)	2.26 ± 0.38	2.63 ± 0.64	0.140	0.241

Table 4: Correlations of Pre-syncope HUTT test parameters between syncope and non-syncope people.

Parameters	Mean ± SD		P value	
	Positive test N= (5)	Negative test N= (16)	Positive test N= (5)	Negative test N= (16)
Systolic ejection time (msec)	242.00 ± 25.88	251.13 ± 9.52	0.480	0.237
LVOT Area (cm^2)	2.96 ± 0.40	3.12 ± 0.35	0.449	0.390
LVOT VTI (cm)	13.18 ± 0.34	13.36 ± 0.88	0.495	0.651
HR (bpm)	77.00 ± 12.43	81.00 ± 16.76	0.580	0.630
SV (mL/beat)	39.04 ± 5.30	41.79 ± 5.10	0.344	0.311
CO (L/min)	3.02 ± 0.75	3.37 ± 0.75	0.396	0.374
SBP (mmHg)	118.60 ± 12.60	128.19 ± 14.41	0.192	0.199
DBP (mmHg)	71.20 ± 9.03	81.06 ± 7.97	0.071	0.030
MAP (mmHg)	86.84 ± 9.68	96.61 ± 8.92	0.090	0.050
SVR (Woods unit)	29.62 ± 4.94	30.66 ± 10.95	0.770	0.840
CPO (Watts)	0.59 ± 0.21	0.71 ± 0.15	0.274	0.169
C.I ($L/min/m^2$)	1.74 ± 0.29	2.12 ± 0.54	0.071	0.165

Table 5: Correlations of During syncope HUTT test parameters between syncope and nonsyncope in people.

Parameters	Mean ± SD		P value	
	Positive test N= (5)	Negative test N= (16)	Positive test N= (5)	Negative test N= (16)
Systolic ejection time (msec)	197.00 ± 25.88	216.69 ± 15.80	0.169	0.050
LVOT Area (cm^2)	2.96 ± 0.40	3.12 ± 0.35	0.449	0.390
LVOT VTI (cm)	9.08 ± 0.56	10.20 ± 2.46	0.108	0.332
HR (bpm)	59.00 ± 9.67	80.13 ± 18.40	0.005	0.025
SV (mL/beat)	27.02 ± 4.19	33.95 ± 3.98	0.016	0.003
CO (L/min)	1.60 ± 0.38	2.69 ± 0.57	0.001	0.001
SBP (mmHg)	85.00 ± 3.53	112.13 ± 12.27	<0.001	<0.001
DBP (mmHg)	42.80 ± 7.01	73.38 ± 7.99	<0.001	<0.001
MAP (mmHg)	56.72 ± 4.84	86.16 ± 8.63	<0.001	<0.001
SVR (Woods unit)	37.83 ± 12.96	34.11 ± 11.64	0.587	0.550
CPO (Watts)	0.20 ± 0.04	0.50 ± 0.10	<0.001	<0.001
C.I ($L/min/m^2$)	0.94 ± 0.24	1.68 ± 0.40	<0.001	0.01

Table 6:Correlations of Recovery HUTT test parameters between syncope and non-syncope people.

Parameters	Mean ± SD		P value	
	Positive test N= (5)	Negative test N= (16)	Positive test N= (5)	Negative test N= (16)
Systolic ejection time (msec)	254.80 ± 33.59	267.19 ± 15.82	0.465	0.261
LVOT Area (cm^2)	2.96 ± 0.40	3.12 ± 0.35	0.449	0.390
LVOT VTI (cm)	17.56 ± 0.68	17.43 ± 0.90	0.743	0.773
HR (bpm)	64.40 ± 10.45	71.88 ± 14.13	0.234	0.291
SV (mL/beat)	52.06 ± 7.62	54.35 ± 4.90	0.554	0.432
CO (L/min)	3.37 ± 0.81	3.84 ± 0.80	0.298	0.269
SBP (mmHg)	107.40 ± 13.40	117.50 ± 14.93	0.194	0.194
DBP (mmHg)	62.80 ± 8.67	72.31 ± 9.10	0.072	0.053
MAP (mmHg)	77.51 ± 10.01	87.22 ± 10.13	0.103	0.076
SVR (Woods unit)	23.81 ± 5.11	23.58 ± 7.40	0.939	0.949
CPO (Watts)	0.59 ± 0.21	0.75 ± 0.17	0.188	0.106
C.I ($L/min/m^2$)	1.97 ± 0.45	2.44 ± 0.58	0.093	0.118

DISCUSSION

In the present study, 21 participants aged over 18 years were included, comprising 11 males and 10 females. Of these, 5 participants developed syncope during the HUTT test (positive test), while 16 did not (negative test). Among the syncope group, 4 participants exhibited a vasodepressor response (type 1), and 1 participant exhibited a cardioinhibitory response (type 2) without asystole. None of the participants had a prior history of cardiac disease that could have influenced the study outcomes. Exclusion of structural heart disease is important because the mechanisms, prognosis and management of syncope differ substantially from those observed in reflex-mediated syncope.[20] Syncope remains a common clinical problem encountered across a wide range of healthcare settings and continues to represent a significant diagnostic challenge. [11,14,15]

Recent literature continues to support the role of HUTT as an important diagnostic tool for identifying reflex syncope and understanding the haemodynamic mechanisms responsible for transient loss of consciousness. [9,13] The Italian protocol for the head-up tilt table (HUTT) test was followed. This protocol includes both passive tilt testing without pharmacological agents and tilt testing with pharmacological provocation. In the present study, only the passive tilt test without the use of drugs was performed. The procedure consisted of three stages: (i) a baseline supine phase of 10 minutes for rest, (ii) a tilt phase in which participants were positioned at an angle of 70–80° for approximately 40–45 minutes, and (iii) a recovery phase in the supine position lasting 10–15 minutes, depending on whether syncope occurred.

During the HUTT procedure, blood pressure (systolic and diastolic) was measured at 1-minute intervals using an automated blood pressure monitor, and heart rate was continuously recorded using electrocardiography (ECG). In addition, echocardiography with pulse wave (PW) Doppler was used to measure systolic ejection time (SET), left ventricular outflow tract velocity time integral (LVOT VTI), and LVOT area for the calculation of stroke volume (SV) and cardiac output (CO) throughout the test.

Other haemodynamic parameters, including mean arterial pressure (MAP), systemic vascular resistance (SVR), cardiac power output (CPO), and cardiac index (C.I), were derived from these primary measurements. The following equations were used to calculate these parameters:

SV = LVOT VTI × LVOT area

CO = SV × HR

MAP = DBP + 1/3 (SBP – DBP)

MAP – RAP = CO × SVR, Since MAP>>RAP we assumed to be RAP = 0mmHg,

so MAP = CO × SVR

CPO = (MAP x CO) ÷ 451

C.I = CO ÷ BSA

From Ohm's Law **V = I × R**, V= voltage, I = current, R = resistance, this equation

correlated with the equation of Darcy's Law **MAP = CO × SVR;**

I = CO

V = MAP

R = SVR

The following were the four (4) stages and information obtained by the HUTT test and ECHO parameters;

i) Baseline supine HUTT test

- Participants were instructed to rest in the supine position for approximately 10 minutes to allow stabilisation of blood pressure (BP) and heart rate (HR), thereby minimising potential variability in baseline measurements. The head-up tilt table (HUTT) procedure was explained in detail, and informed consent was obtained prior to initiation of the test. A blood pressure cuff and a five-lead electrocardiogram (ECG) system were then applied for continuous monitoring.
- Echocardiography was performed to exclude underlying cardiac pathology and to obtain baseline measurements, including left ventricular outflow tract velocity time integral (LVOT VTI), LVOT area, and systolic ejection time (SET).
- During the baseline stage, no significant haemodynamic differences were observed between syncope and non-syncope groups, except for a slight reduction in diastolic

blood pressure (DBP) in participants who subsequently developed syncope.

Presyncope stage of the HUTT test

A rapid gravitational redistribution of blood volume, estimated at approximately 0.5–1.0 L, occurred from the thoracic compartment to the lower extremities within 10–15 seconds of tilting the table from 0° to 70°. This resulted in significant venous pooling in all participants, leading to a reduction in venous return (VR), as evidenced by decreases in left ventricular outflow tract velocity time integral (LVOT VTI) and systolic ejection time (SET) measured by echocardiography.

Cardiac output (CO) decreased by approximately 10–20%; however, systolic blood pressure (SBP) was largely maintained. In contrast, diastolic blood pressure (DBP), mean arterial pressure (MAP), and systemic vascular resistance (SVR) showed significant increases. These findings are consistent with previous studies.[6]

The increase in SVR can be attributed to baroreflex-mediated vasoconstriction, affecting both skeletal muscle and splanchnic arterioles, thereby reducing blood pooling within the capacitance vessels of the lower extremities. Although heart rate (HR) increased slightly, this compensatory response was insufficient to offset the reduction in stroke volume (SV) and cardiac output. Current physiological models of reflex syncope also emphasise the interaction between autonomic nervous system activation and cardiovascular reflexes in determining the haemodynamic response during orthostatic stress. Accurate classification and risk stratification of syncope remain important for identifying patients who may require additional cardiovascular investigation and long-term follow-up.[12] Syncope is increasingly recognised as a multifactorial syndrome with diverse underlying mechanisms, emphasising the importance of a systematic diagnostic approach. [10,21]

Venous return was partially supported by arterial vasoconstriction and the skeletal muscle pump mechanism.

ii) During the Syncope stage of the HUTT test

- Over time, a progressive increase in systemic vascular resistance (SVR) was observed in all participants, mediated by baroreflex-induced vasoconstriction of the splanchnic and skeletal muscle vascular beds, as cardiac output (CO) continued to decline. Participants who developed syncope demonstrated a more pronounced increase in SVR, associated with a progressive reduction in left ventricular outflow tract velocity time integral (LVOT VTI), stroke volume (SV), cardiac output, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP).
- In contrast, non-syncope participants maintained or showed only a mild increase in SVR, accompanied by less marked reductions in LVOT VTI, SV, CO, SBP, DBP, and MAP.
- Heart rate (HR) in syncope participants initially increased as a compensatory response to declining cardiac output; however, this response was insufficient, and HR subsequently decreased to approximately 50–60 beats per minute, depending on the haemodynamic subtype of vasovagal syncope. All participants who developed syncope experienced a marked reduction in SBP and DBP (typically <90/50 mmHg), leading to cerebral hypoperfusion.
- Prodromal symptoms, including nausea, vomiting, a sensation of warmth, dizziness, and sweating, were commonly observed prior to loss of consciousness.

iii) Recovery stage of the HUTT test

- Following the tilt phase, participants were returned from 70° to the supine position. In individuals who developed syncope, a rapid overshoot in systolic and diastolic blood pressure (SBP and DBP) was observed. In contrast, non-syncope participants demonstrated either stable or slightly reduced SBP and DBP, as hypotension had not occurred.
- Heart rate (HR), left ventricular outflow tract velocity time integral (LVOT VTI), stroke volume (SV), and cardiac output (CO) recovered towards baseline levels in both groups. Systemic vascular resistance (SVR) also returned to baseline or near-baseline values.
- Some participants in the syncope group exhibited delayed recovery of HR due to a cardioinhibitory response and required atropine administration.

Additionally, post-test symptoms such as nausea, vomiting, headache, and dizziness were reported in some individuals.

- During the syncope phase, participants with syncope demonstrated higher SVR compared to non-syncope participants when hypotension was reproduced. These findings suggest that the primary mechanism underlying syncope in this study was a reduction in cardiac output secondary to hypotension. Comprehensive evaluation of syncope requires correlation of clinical history, physical examination findings and haemodynamic assessment in order to exclude alternative causes of transient loss of consciousness.[14,18] Diagnostic approaches that combine clinical assessment with haemodynamic monitoring have been shown to improve diagnostic accuracy in patients presenting with recurrent unexplained syncope.[9,13] Accurate classification and risk stratification of syncope remain important for identifying patients who may require additional cardiovascular investigation and long-term follow-up. [12] Despite decades of research, important questions remain regarding the precise haemodynamic mechanisms responsible for syncope, highlighting the need for continued investigation using modern non-invasive techniques. Recurrent syncope frequently results in emergency department attendance and may contribute to reduced quality of life and increased healthcare utilisation. [16,19] Future studies incorporating larger populations and advanced haemodynamic monitoring may further improve understanding of syncope pathophysiology and optimise patient management.[20]

Management of syncope individuals

- Intake of salt and water to increase blood volume (venous return) and increase vasoconstriction in muscle pump cells. Conservative measures, including increased fluid intake, salt supplementation, physical counter-pressure manoeuvres and lifestyle modification, remain the first-line therapeutic strategies for most patients with vasovagal syncope.[17]
- Physical exercise such as squatting and standing on the toes.
- Avoid prolonged standing, stress, anxiety, and heat areas.
- Avoid dehydration.
- Proper food intake for conservation of energy.
- Stay in well-ventilated areas.
- Consultation of a neurologist for further investigation, such as EEG, brain CT and MRI to rule out any brain abnormalities or disorders.
- Additional investigations should be guided by clinical suspicion and may include neurological and cardiovascular testing when the diagnosis remains uncertain.[18]

LIMITATIONS

The present study has several limitations. First, the relatively small sample size limited the ability to determine precise cut-off values for left ventricular outflow tract velocity time integral (LVOT VTI), stroke volume (SV), and cardiac output (CO) associated with the onset of syncope.

Second, the use of echocardiography has inherent limitations in accuracy and reproducibility, as measurements of LVOT VTI are dependent on individual acoustic windows. In some participants, suboptimal imaging quality resulted in the exclusion of certain measurements.

Third, continuous beat-to-beat monitoring of blood pressure and LVOT VTI was not feasible using the non-invasive methods employed in this study, which may have limited the precision of haemodynamic assessment.

Finally, the use of a passive tilt protocol without pharmacological provocation required prolonged tilt durations (exceeding 45 minutes), which posed practical challenges and may have affected the consistency of cardiac output measurements throughout the procedure.

CONCLUSION

The primary determinant of the reduction in cardiac output (CO) observed in this study was hypotension. Echocardiography proved valuable in quantifying stroke volume (SV) and cardiac output through measurement of the left ventricular outflow tract velocity time integral

(LVOT VTI), enabling assessment of haemodynamic changes from baseline to recovery.

Non-invasive techniques, including echocardiography and automated blood pressure monitoring, were utilised to avoid stimulation of the autonomic nervous system, which may occur with invasive procedures and potentially influence the study outcomes.

Certain parameters, such as cardiac power output (CPO), systolic ejection time (SET), and cardiac index (C.I.), were not extensively analysed due to the relatively small sample size and the limited differences observed between syncope and non-syncope groups. Although some statistically significant changes were noted during the pre-syncope and syncope stages, particularly for CPO, the findings were insufficient to draw definitive conclusions regarding its clinical relevance in this cohort.

Ethics approval and consent to participate: The study was conducted by and approved by the IEC, KMC, and Manipal. Informed consent was given by all study participants.

Consent of publication: Not applicable

Availability of data and materials: I want to make sure that all pertinent information and materials related to the manuscript are available. We are ready to supply any information required upon request to maintain accountability and transparency.

Competing interests: Nil

Funding: Nil

Authors' contributions: Walter Uronu analysed and interpreted the patient data and was a major contributor in writing the manuscript. Acknowledgements: We thank the HOD and faculty members of the Department of Cardiovascular Technology and Department of Cardiology, KMC, Manipal.

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