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ORIGINAL ARTICLE

Diagnostic utility and outcomes of inpatient investigations for syncope in a regional setting

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Abstract

Background: Syncope is a common presentation to the emergency department with a wide spectrum of aetiology. The identification of the underlying cause can be diagnostically challenging, as are the choice of investigations and the decision for inpatient versus outpatient disposition.

Aims: This study aimed to evaluate the aetiology of syncope as documented, the diagnostic yield of inpatient investigations and outcomes for adult patients admitted for syncope.

Methods: A single-centred, retrospective cohort study was conducted in adult patients admitted for syncope within a 2-year period. A total of 386 patients were identified after exclusion. Information regarding syncope aetiology, investigations and outcomes were established via chart review of electronic records.

Results: The most common cause of syncope was neural-mediated (43%), followed by orthostatic (36.5%) and cardiogenic (20.5%). The investigations performed in order of frequency included: telemetry electrocardiogram (ECG) (75.4%), computed tomography head non-contrast (58.8%), transthoracic echocardiogram (TTE) (20.2%), computed tomography pulmonary angiogram (CTPA) (6.5%), MR brain (3.9%), electroencephalogram (1.3%) and carotid ultrasound (0.3%). Telemetry ECG, TTE and CTPA led to the diagnosis of syncope in a minority of patients only. As a result, 17.5% of patients had a new intervention on discharge, 5.4% were readmitted for syncope and 9.6% of patients died.

Conclusions: In the context of the inpatient evaluation of syncope, this study supports the use of telemetry ECG and TTE. Neuroimaging demonstrates a low diagnostic yield for the cause of syncope, but it may have a role to play in excluding other pathologies. Our study does not support the routine use of CTPA, EEG or carotid ultrasound in the evaluation of syncope.

Introduction

Syncope is defined as a transient loss of consciousness triggered by global cerebral hypoperfusion.[1] It is one of the most common presentations in the emergency department (ED) in Australia, representing approximately 1% of all ED presentations.[2] Additionally, it is estimated that around 30% of all syncope ED presentations are then admitted to hospital.[2] The causes for syncope are highly variable and are typically subdivided into three main categories: neural-mediated (also known as reflex), orthostatic and cardiogenic syncope.[1] These syncopal aetiologies vary between those that are benign in nature, such as vasovagal syncope, to potentially life-threatening causes, such as cardiac arrhythmias.[1] The identification of the underlying cause of syncope can be diagnostically challenging, as are the decisions regarding necessity of an admission and investigations performed.

Several clinical practice guidelines exist regarding the inpatient work-up for syncope; however, a clear

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consensus on the essential investigations for syncope remains unclear.[3–5] Commonly performed investigations include telemetry electrocardiogram (ECG), Holter monitoring, computed tomography (CT) imaging of the brain, echocardiography and electroencephalography (EEG).[3–5] Despite this, the most recent Australian Government of Health guidelines, via the Choosing Wisely initiative, recommend against the use of most of these mentioned investigations in cases of uncomplicated syncope.[6] It is therefore important to evaluate via research which inpatient investigations for syncope offer the highest yield in establishing the underlying aetiology in order to guide physician decision making. Furthermore, research in this area may reveal investigations that are relatively low yield for detecting the underlying cause of syncope but important to exclude other sinister pathology, such as a CT brain.[1] Finally, by identifying investigations that offer low diagnostic utility, this may allow avoidance of unnecessary investigations to overall minimise associated patient risk and burden on the healthcare system.

Research regarding inpatient investigations for syncope also serves to determine patient outcomes following an admission for syncope. More specifically, establishing rates of readmission for syncope and patient mortality rates is important in the overall evaluation of the utility of inpatient admission for syncope. Previous studies demonstrated a large spectrum in admission rates for syncope internationally; however, it remains mostly unclear how an inpatient admission affects overall morbidity or mortality.[2] Further research in this area is important in order to help clinicians determine inpatient versus outpatient disposition for patients presenting with syncope. This study was performed to establish the aetiology of syncope as recorded by treating clinicians, the diagnostic yield of inpatient investigations and outcomes for adult patients admitted for syncope within a 2-year period.

Methods

A single-centre, retrospective cohort study was conducted in adult patients admitted for syncope in the Townsville Hospital and Health Service between the period of 1 January 2019–1 January 2021. Institutional ethics approval was obtained in conjunction with the local clinical research unit (approval number THHSACQUIRE 1404). Patients were identified using the ICD-10 code (or similar) for syncope and synonyms (ICD10 R55, T67.1 and G90.01). A total of 259 patients were then excluded prior to data collection for the following reasons: admission at a peripheral hospital, admission reason was not syncope, duplicate record or admission coding error (Fig. 1). Patients at a peripheral hospital within the health service were excluded as these hospitals use paper instead of electronic records, rendering the data inaccessible remotely.

The remaining patients each had an attached encounter location for the admission, which revealed data regarding the location of admission. Rates of readmission were determined by searching for additional admissions and specifically admissions associated with ICD-10 codes for syncope within the 2-year period. Rates of death were determined via electronic coding that indicates if a patient was deceased. The data regarding the diagnosis of syncope and inpatient investigations were performed via manual chart review using the electronic record system. The cause of syncope was coded into three main categories: neural-mediated, cardiogenic and orthostatic. Subcategories into each of the stated categories were utilised. The diagnosis of syncope was determined based on what was documented by the treating clinicians, typically located in the issues list, impression or final diagnosis. Division into subcategories was also achieved by the clinical documentation. For example, a patient with micturition syncope was classified as 'neural-mediated: situational'. For this study, we defined 'cardiogenic: unknown' as patients who were admitted under the cardiology department with presumed cardiogenic syncope, but a specific cause was not identified.

Once primary data were extrapolated, data analysis was performed using IBM SPSS Statistical Software (Version 27.0; IBM Corp., Armonk, NY). Demographic data and other characteristics are presented using descriptive analysis such as numbers and percentages. Data will be stored on a secure Excel spreadsheet available only to investigators and stored for 5 years following the completion of the study.

Results

A total of 645 patients were identified as having an admission for syncope between the dates of 1 January 2019–1 January 2021. Two hundred fifty-nine patients were excluded prior to data collection for the following reasons: 169 were excluded due to admission at a peripheral hospital (65.3%), 41 were excluded as the patient did not have a syncopal event (15.8%), 19 were excluded due to a duplicate record (7.3%), and 30 were excluded due to an admission coding error (11.6%) (Fig. 1). The final number of patients included for data collection was 386.

In terms of patient characteristics, 197 patients were male (51.0%) and 189 were female (49.0%). The overall mean age was 63.35, with an age range of 18–104 years (Table 1). Patients arrived to the ED via several methods,

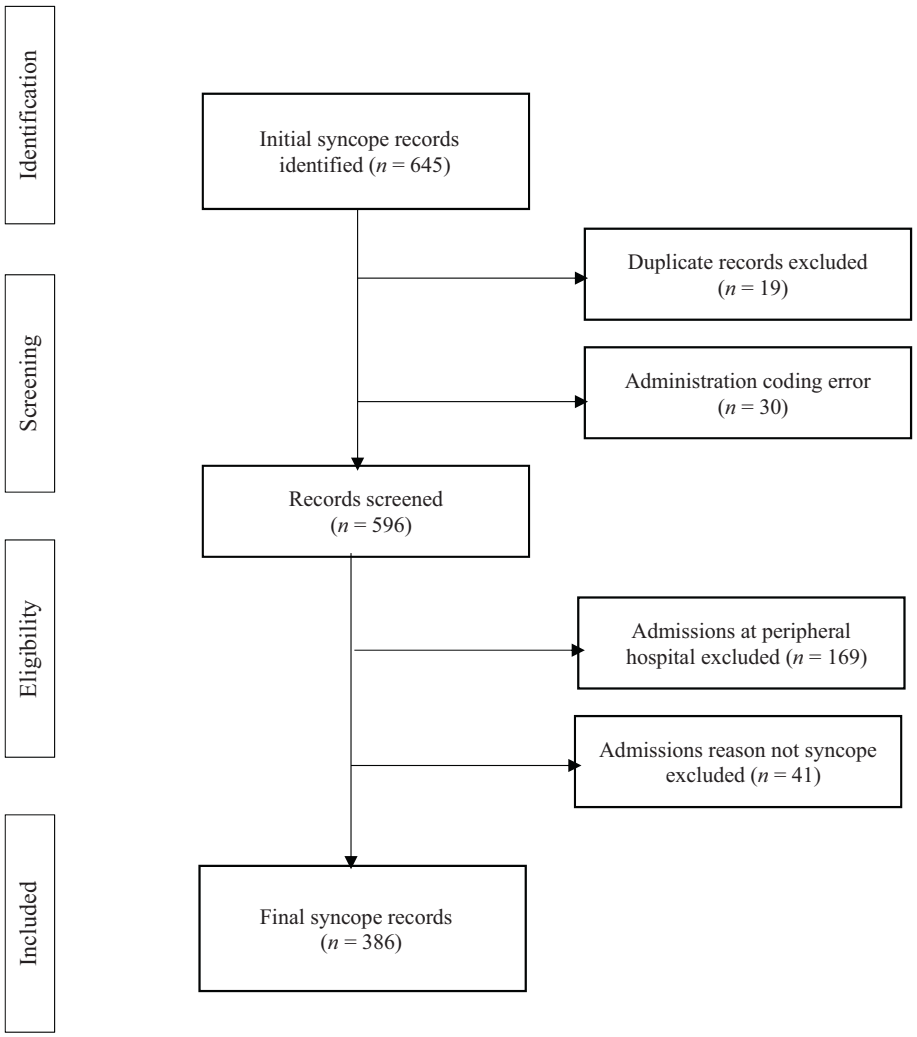

Figure 1 Audit flow diagram.

with the majority arriving via road ambulance paramedic service, representing 297 patients (76.9%). General medicine was the most common admission unit, representing 43% of patients, followed by ED short stay unit (25.1%), cardiology (24.9%), geriatrics (3.4%), other medical specialties (3.1%) and other surgical specialties (0.5%).

In order of frequency, the most common cause of syncope was neural-mediated in 166 patients, with the subcategory causes being vasovagal (n = 136) and situational (n = 30) (Table 2). A total of 141 patients' syncopal event was orthostatic in nature, with the subcategory causes being volume depletion (n = 103), medication related (n = 29) and autonomic dysfunction (n = 9). Finally, 79 patients experienced a cardiogenic syncopal event secondary to bradyarrhythmia (n = 19), structural heart disease (n = 12), tachyarrhythmia (n = 8) or a cardiopulmonary cause, such as pulmonary embolism (n = 2). Thirty-eight patients with cardiogenic syncope did not have a specific cause identified.

A total of 291 patients had telemetry as part of their investigations, leading to the diagnosis of the cause of the syncopal event in 28 patients (9.6%) (Table 3). Seventy-eight patients underwent a transthoracic echocardiogram (TTE) and 25 underwent a computed tomography pulmonary angiogram (CTPA) leading to the diagnosis in eight patients (10.3%) and one patient (0.3%) respectively. A total of 227 patients underwent at CT head, 15 patients underwent magnetic resonance imaging (MRI) head, five patients underwent an EEG and one patient underwent a carotid ultrasound scan. Not all of these investigations resulted in the diagnosis of syncope in patients.

Of the previously listed investigations, a total of 172 patients (44.6%) had two investigations, followed by one investigation in 93 patients (24.1%) and three

Table 1 Baseline characteristics of all patients

Characteristics	Number/Frequency
Age	
Mean ± SD (years)	63.3 ± 19.4
Median IQR (years)	68.0 IQR (51.00–78.0)
Minimum age (years)	18
Maximum age (years)	104
Range	86
Gender	
Male	197 (51.0%)
Female	189 (49.0%)
ED arrival transport mode	
Ambulance (helicopter)	4 (1.0%)
Ambulance (road paramedic)	297 (76.9%)
Ambulance (road patient transport)	3 (0.8%)
Community services	1 (0.3%)
Walked in	46 (11.9%)
Other	4 (1.0%)
Not specified	31 (8.0%)
Admission unit	
General medicine	166 (43.0%)
Cardiology	96 (24.9%)
ED (short stay)	97 (25.1%)
Geriatrics	13 (3.4%)
Other medical specialty	12 (3.1%)
Other surgical specialty	2 (0.5%)

ED, emergency department.

investigations in 57 patients (14.8%) (Fig. 2). Fifty-six patients (14.5%) underwent zero of the stated investigations. A total of six patients (1.5%) underwent four investigations and two patients (0.5%) underwent five of the listed investigations. No patients underwent a total of six or seven of the listed investigations.

A total of 21 patients were readmitted for syncope within the data collection period, and a total of 37 patients died (Table 4). None of these deaths were secondary to the syncopal event or the cause of the syncopal event. Sixty-eight patients had a notable new intervention on discharge. In order of frequency, interventions included loop recorded insertion ($n = 17$), alteration to regular medications ($n = 15$), permanent pacemaker insertion ($n = 10$), course of antibiotics prescribed ($n = 9$), rate control commenced ($n = 5$), other intervention ($n = 5$), implantable cardioverter defibrillator (ICD) inserted ($n = 2$) or referral for coronary artery bypass grafting (CABG) ($n = 2$).

Table 2 Aetiology of syncope findings

Cause of syncope	Number/Frequency
Neural-mediated (total)	166 (43.0%)
Vasovagal	136 (35.2%)
Situational	30 (7.8%)
Cardiogenic (total)	79 (20.5%)
Tachyarrhythmia	8 (2.1%)
Bradyarrhythmia	19 (4.9%)
Structural heart disease	12 (3.1%)
Cardiopulmonary	2 (0.5%)
Unknown	38 (9.8%)
Orthostatic (total)	141 (36.5%)
Volume depletion	103 (26.7%)
Medication related	29 (7.5%)
Autonomic dysfunction	9 (2.3%)

Table 3 Inpatient investigations and link to determining aetiology of syncope

Investigation	Number/Frequency
Telemetry (total)	291 (75.4%)
Lead to syncope diagnosis	28 (9.6%)
Did not lead to syncope diagnosis	263 (90.4%)
Transthoracic echocardiogram (total)	78 (20.2%)
Lead to syncope diagnosis	8 (10.3%)
Did not lead to syncope diagnosis	70 (89.7%)
Electroencephalogram (total)	5 (1.3%)
Lead to syncope diagnosis	0 (0.0%)
Did not lead to syncope diagnosis	5 (100.0%)
CTPA (total)	25 (6.5%)
Lead to syncope diagnosis	1 (4%)
Did not lead to syncope diagnosis	24 (96%)
CT head non-contrast (total)	227 (58.8%)
Lead to syncope diagnosis	0 (0.0%)
Did not lead to syncope diagnosis	227 (100.0%)
MRI brain ± contrast (total)	15 (3.9%)
Lead to syncope diagnosis	0 (0.0%)
Did not lead to syncope diagnosis	15 (100.0%)
Carotid ultrasound (total)	1 (0.3%)
Lead to syncope diagnosis	0 (0.0%)
Did not lead to syncope diagnosis	1 (100.0%)

CT, computed tomography; CTPA, computed tomography pulmonary angiogram; MRI, magnetic resonance imaging.

Discussion

We conducted a single-centre, retrospective cohort study over a 2-year period to evaluate the yield of inpatient investigations and outcomes for adult patients admitted for syncope. The mean age of the patients was 63 years, and the distribution of males and females was close to equal. The main admission units were general medicine, ED short stay and cardiology. Although not reflected in the data, patients admitted to the ED short stay unit underwent fewer investigations overall, likely reflecting lower risk syncopal episodes. Of note, the ED short stay at the study tertiary facility does not routinely offer telemetry ECG, which may indicate some degree of patient selection for this unit.

Several syncope risk stratification scores are available to help guide the decision on admission versus discharge

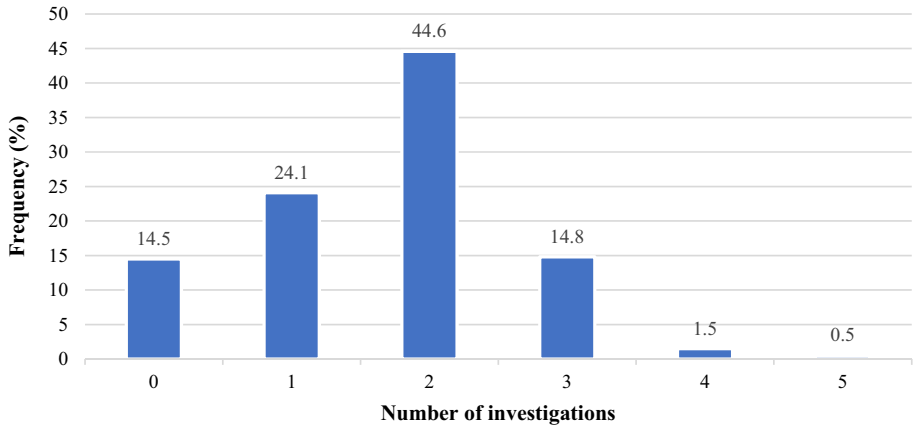

Figure 2 Number of inpatient investigations performed for syncope.

for patients presenting with syncope. Examples of these scoring systems include the San Francisco, Canadian and OESIL scores.[3,4] The San Francisco and Canadian syncope scores describe the risk of a serious event in 7 and 30 days respectively, whereas the OESIL score correlates with 1 year total mortality.[3,4] The most recent European Society of Cardiology and American College of Cardiology/American Heart Association guidelines both recommend against the use of syncope scores as they have not proved to be superior to clinical judgement.[3,4] Of note, syncope scores are not part of a clinical pathway at our study centre, and they are not used consistently.

The aetiology of syncope was determined based on what was documented by the treating clinicians. Neural-mediated syncope was the most common cause of syncope observed in 43% of patients. Vasovagal syncope was the most frequent diagnosis of syncope, which is in keeping with other similar observational studies.[2,7] Orthostatic syncope was the cause in 36.5% of patients, which is, interestingly, a higher prevalence than a rate of 10%–15% revealed in similar studies.[1,7] This may be explained by the subtropical climate where the tertiary hospital is located, leading to an increased risk of dehydration and exacerbation of orthostatic symptoms experienced in the elderly and/or those on regular antihypertensives. Finally, cardiogenic syncope accounted for 20.5% of patients, which is consistent with previous observational studies.[1] Notably, other studies suggest an unexplained syncope rate varying from 15% to 30%,[1,7] but our study did not reflect this. In this study, 9.8% of patients were classified as 'cardiogenic: unknown', which may encompass the classically observed unexplained cohort of patients. In addition, it can be speculated that vasovagal syncope may have been utilised as a diagnosis of exclusion in some cases, potentially also encompassing some patients with an unknown aetiology.

Telemetry ECG was performed in 75% of patients and led to the diagnosis of syncope in 9.6% of patients, which is a similar rate to that demonstrated in a comparable observational study.[8] Studies suggest that the diagnostic yield of ambulatory cardiac monitoring increases with duration,[1] which notably relates to the outcome observed in 17 patients of loop recorded insertion. Although telemetry ECG did not lead to the diagnosis of syncope in 90% of patients, its importance as an inpatient investigation for syncope can be argued. Telemetry ECG led to the diagnosis of either tachy or bradyarrhythmia in a total of 27 patients, which are potentially life-threatening pathologies. In addition, telemetry ECG is an inexpensive and non-invasive investigation, which further supports its role in the inpatient investigation of syncope.

Table 4 Outcomes of patients admitted for syncope

Outcomes	Number/Frequency
Readmission (total)	21 (5.4%)
1 readmission for syncope	18 (85.7%)
2 readmissions for syncope	3 (14.3%)
Death (total)	37 (9.6%)
Death due to syncope/syncope cause	0 (0.0%)
Death due to another pathology	23 (62.2%)
Death cause unknown	14 (37.8%)
New intervention on discharge (total)	68 (17.6%)
Alteration to regular medications	15 (22.1%)
Rate control added	5 (7.4%)
Loop recorded inserted	17 (25.0%)
Permanent pacemaker (PPM)	10 (14.7%)
Implantable cardioverter defibrillator (ICD)	2 (3.2%)
Planned for coronary artery bypass grafting (CABG)	2 (3.2%)
Course of antibiotics	9 (13.2%)
Other	6 (8.8%)

In this study, TTE was performed in 20.2% of patients and led to syncope diagnosis in 10.3% of cases. This rate of TTE is similar to that performed in other studies, however with a slightly better yield rate than 2%–3%.[8] The 2017 American College of Cardiology/American Heart Association guidelines recommend TTE for patients with unexplained syncope when the initial evaluation suggests a cardiovascular abnormality.[3] This was supported by a previous prospective study that found that TTE revealed positive findings in 27% of syncope patients with a cardiac history or abnormal ECG, versus 0% in syncope patients without a cardiac suspicion.[9] A similar retrospective cohort study of patients with syncope found that echocardiography was performed in 69.7% of patients but only led to syncope diagnosis in 2% of these patients.[10] In addition, this study found that echocardiography contributed to increased costs and length of patient hospital admission.[10] Given with the 10.3% positive rate, our study suggests that TTE should remain an important investigation to rule out structural cardiac disease and other sinister cardiac pathology, especially in patients where cardiogenic syncope is suspected.

CTPA was performed in 6.5% of patients and led to the diagnosis of syncope in 0.3% of these patients. A previous multi-centre, retrospective cohort study of patients presenting with syncope found the rate of CTPA performed to be 7.2%,[11] which is similar to the rate demonstrated in our study. However, this particular study found the yield rate of CTPA to be 7.9%,[11] which is significantly higher than the rate demonstrated in our study. Another observational study suggested that syncope was overall an atypical symptom for pulmonary embolism, occurring in only 10% of patients.[12] Given the low diagnostic yield demonstrated, our study does not support the routine use of CTPA for the diagnosis of syncope.

EEG was performed in 1.3% of patients and did not lead to the diagnosis of syncope in any of these individuals. Our study suggests that EEG has a low diagnostic yield in the investigation of syncope and does not recommend its routine use. These findings are supported by multiple previous retrospective cohort studies which demonstrated the rate of normal EEGs in patients with syncope to be around 90%.[13,14] The most common abnormal finding in both of these studies focal epileptiform discharges, occurring in approximately 5% of patients tested.[13,14]

In this study, carotid ultrasound was only performed in one patient (0.3%) and did not result in a syncope diagnosis. Due to the very minimal data, it is difficult to comment on the utility of carotid ultrasound based on our results. Previous retrospective cohort studies suggested that carotid ultrasounds were normal in approximately 90% of patients with syncope and, therefore, offer a low diagnostic yield in the work-up for syncope.[8,10,15]

CT head non-contrast was performed in 58.8% of patients but did not lead to a syncope diagnosis in any patients. Similarly, MRI brain was performed in 3.9% of patients and also did not lead to the diagnosis of syncope in any of these patients. Our study does not support the role of neuroimaging for the diagnosis of syncope. However, the role for detection of other pathology or pathology secondary to the syncopal event cannot be commented on as such data were not included in our study. A retrospective cohort study of 1114 patients with low-risk syncope reported a rate of CT head investigation of 62.3% and MRI head of 10.2% of cases, neither of which yielded any clinically significant findings in any patients, even in those who had experienced a minor head trauma.[16] This is similar to the findings demonstrated in our study, aside from an increased rate of MRI brain scans performed. Interestingly, a different retrospective cohort study by Grossman *et al*. revealed that CT head scans detected an abnormal finding in 5% of patients with syncope who underwent this investigation.[17] *Post hoc* analysis of these patients revealed that all of these individuals were either experiencing a headache, had a focal neurological deficit on examination or had evidence of head trauma clinically.[17] This perhaps suggests that CT head should be reserved for those with evidence of possible neurological involvement, as the diagnostic yield is greater in this patient group.

In our study, 5.4% of patients were readmitted for syncope within the 2-year period. This is less than the rate of 9.3% demonstrated in a similar retrospective, cohort study on the outcomes of patients with syncope.[18] In our study, 9.6% of patients died within the study period; however, notably all deaths occurred for reasons unrelated to the syncopal event. A comparable observational study found the all-cause mortality rate of patients presenting to the ED for syncope to be 7.6% at 12 months,[19] which is slightly below the findings of this study. Notably, this study found the death rate from causes possibly related to syncope to be 3.8%,[19] which is higher than that observed in our study. Overall, our study suggests that the rate of morbidity via readmission and mortality in patients admitted for syncope to be relatively low.

The strengths of our study include the large sample size of patients and perspective from a regional, tertiary facility in which there are few alternative hospitalisation options available in the surrounding region. In addition, we were able to collect data and comment on the utility of a broad range of investigations rather than a select few. Finally, data collection from electronic records

allowed for very reliable recordings regarding the rate of investigations performed and rate of implantable cardiac devices inserted. This is because each of these entities has an associated electronic code and/or electronic procedure note, so data could be accurately collected without relying on medical documentation.

The limitations of this observational study include that it was a single-centre study, so local practice and protocol may have influenced the results obtained. In addition, the aetiology of syncope was determined based on what was documented by the treating clinician, which may not be consistently accurate as to the cause of syncope. It was not always clear from the documentation how the treating clinicians reached this diagnosis and whether supplementary bedside tests were utilised, such as tilt table for vasovagal or postural blood pressure drop for orthostatic syncope. Similarly, vasovagal syncope was anecdotally observed to represent a diagnosis of exclusion in some cases rather than labelling such casess as unknown, which might have led to overestimation of the rate of vasovagal syncope. Finally, unlike investigations and implantation of cardiac devices with objective electronic coding, alteration of medications as a new intervention on discharge relied on medical staff documentation being detected via chart review and, hence, recorded in the final results. Therefore, if changes to medications occurred but were not documented, then these interventions would have been missed, thereby underestimating the rate of medication change on discharge.

Conclusion

The findings of this study support the use of telemetry ECG for all patients with syncope and TTE in some patients where a cardiogenic cause needs to be excluded. Neuroimaging demonstrated a low diagnostic yield for the diagnosis of the cause of syncope; however, the role of neuroimaging for the exclusion of other or secondary pathology was not explored in the results. Finally, our study does not support the routine use of CTPA, EEG or carotid ultrasound in the evaluation of syncope due to poor diagnostic yield. Future research in the area would be beneficial to further support these findings and define those clinical scenarios in which certain investigations should be utilised in the evaluation of syncope.

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