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Review

Complex Pathology of Cardiac Syncope in Old Patients

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Abstract

Syncope is characterized by a transient and rapid loss of consciousness for a short duration, with full spontaneous recovery within minutes. Syncope causes up to 2% of all emergency medical consultations. The incidence of syncope is similar in men and women, is higher in old patients (slightly higher in old women). The prevalence of syncope is up to 23% for the institutionalized elderly. In elderly patients, syncope often presents atypically, such as with falls; these patients might also have difficulty recalling events. The true incidence and prevalence of syncope in elderly patients are expected to be higher than those estimated in most studies. The causes of syncope are highly age-dependent. Reflex or neurally mediated syncope is the most common cause, particularly in younger patients. As individuals age, orthostatic hypotension and cardiac syncope become more frequent. In elderly patients, neurally mediated syncope is the most prevalent form of syncope. Orthostatic syncope is more frequent in the elderly than in young patients. In the elderly, cardiac causes account for about 15% of all cases, and in about 10% of the cases, the origin of syncope is unknown. A combination of different etiologies is common in geriatric patients because many pathophysiologies coexist, including age-related physiological changes, co-morbidities (e.g., neurological pathologies), multiple medications (with interactions, or with lowering of BP

and HR), malnutrition, sarcolepsy, and prolonged bed rest. The available clinical guidelines for the diagnosis, investigation, and treatment of syncope are insufficient to address syncope in elderly patients. A comprehensive geriatric approach that considers the functional and cognitive capacities of individuals along with the medical and psychosocial aspects would be more appropriate. Modern medicine can be used to treat syncope in some geriatric patients and should be offered whenever possible.

Keywords
Cardiac syncope; geriatric cardiology

1. Introduction

Syncope is characterized by a transient and rapid loss of consciousness for a short duration, with full spontaneous recovery within minutes. Syncope accounts for up to 2% of all emergency medical consultations. The incidence of syncope is higher in old patients [1-3]. The incidence of syncope is 5.4 events per 1,000 person-years in 60 to 69-years old people, 11.1 events in those aged 70 to 79, and 19.5 events in those aged more than 80 [3]. The incidence is higher in older women [1-3]. The prevalence of syncope is up to 23% for the institutionalized elderly [3]. Syncope in elderly patients often occurs atypically, such as with falls, and elderly patients might also have difficulty recalling events. The true incidence and prevalence of syncope in elderly patients are expected to be higher than those estimated in most studies [1-3].

2. Causes of Syncope in the Elderly

Syncope is caused by a fall of systolic blood pressure (BP), sometimes below 70 mm Hg, due to global cerebral hypoperfusion for 4 sec or longer. BP results from “stroke volume × heart rate” (HR) multiplied by peripheral resistance [4]. Many cardiac pathologies can reduce stroke volume, and several arrhythmias might alter the HR. Tonic loss or vasodilation might decrease peripheral resistance. The causes of syncope are highly age-dependent [4]; reflex or neurally mediated syncope is the most common cause of syncope, particularly in younger patients. As individuals get older, orthostatic hypotension and cardiac syncope might occur more frequently. In elderly patients, neurally mediated syncope is the most prevalent form of syncope (almost 67%). The neuro reflex form of syncope (vasovagal, situational, and carotid sinus syndrome) is more common in younger patients than in older patients (62% versus 36%), while orthostatic syncope is more frequent in the older group than in the younger group (31% versus 4%). In the elderly, cardiac causes account for about 15% of all cases, and in about 10% of the cases, the origin of syncope is unknown. A combination of different etiologies is common in geriatric patients because many pathophysiologies coexist, including age-related physiological changes, co-morbidities (e.g., neurological pathologies), multiple medications (with interactions, or with lowering of BP and HR), malnutrition, sarcolepsy, and prolonged bed rest [1-5].

3. Diagnosis of Cardiac Syncopes

Cardiac syncopes are associated with an increase in the risk of mortality. Table 1 shows the known risk factors, and Table 2 shows the most frequent causes of cardiac syncope. The 2018 ESC Guidelines [4] recommended that the syncope should be assessed by performing specific anamnesis (see Table 3), clinical examination, ECG, and laboratory analysis (hematology and chemistry). Cardiac biomarkers are useful. An abnormal ECG and/or pathologic biomarkers indicate the need for hospitalization. For syncope due to myocardial ischemia, the 2019 ESC Guidelines [6] recommended two different approaches. In patients with a low pre-test probability for a coronary artery disease and in those who report atypical chest pain, a coronary CT or a cardio-stress-MRI/SPECT or /PET examination is recommended. If the ECG shows ST-elevation changes and troponin values are pathologic, a coronarography is suggested. Echocardiography is required for assessing valval pathologies (e.g., severe aortic valve stenosis, dysfunctional prosthetic heart valve, and myxoma), myocardial anatomy, and kinetics (dyskinesia, ejection function, and stroke volume), and the presence/absence of pericardial effusion. In suspected arrhythmic syncopes, a dynamic-ECG recording might be performed. The exercising test is not a routine diagnostic tool, but it might be considered if the syncope occurred during or after physical exertion [4]. Several scores have been developed for cardiac syncopes and are described in the 2018 ESC Guidelines [4] but are less useful than clinical examination. Head-up tilt testing has been used for over 25 years to investigate suspected neurally orthostatic-mediated syncopes, but it cannot be recommended as a routine tool because of poor reproducibility, lack of prognostic role, and insufficient randomized studies [7]. High sensitivity troponin-I and BNP tests are useful for diagnosing patients with cardiac syncopes, as they allow risk-stratification [8-10]. However, cardiac biomarkers were not recommended in the 2018 ESC Guidelines [4] because there is no evidence showing that their routine use can contribute to a better and more cost-effective diagnosis.

Table 1 Risk markers in syncope.

Cardiac etiology
Anamnesis
Major criteria
Recent onset of dyspnea or chest pain
Syncope during effort
Tachyarrhythmia before syncope
Relevant structural cardiac disease (coronary heart disease, heart valve pathology, heart failure, pacemaker, and ICD)
Minor criteria
Valuable as the major criteria when the ECG is pathologic or in the presence of a relevant cardiac disease.
Absence of warning symptoms or a short time (<10 s) to syncope
Syncope in the sitting position
The sudden death of a young relative
ECG

Major criteria
Ischemic changes or Q waves
Atypical or severe cardiac hypertrophy
Cardiac pacemaker or ICD-dysfunction
Bradycardia:
• Persisting sinus bradycardia (<40 beats/min), several sinus pauses >4 sec or sinoatrial block in non-sporty individuals
• AV-block II Mobitz 2 or AV-block III
• Bradycardic atrial fibrillation
Intraventricular blocks
Supraventricular tachycardic arrhythmias, self-limiting or permanent ventricular tachycardia
Brugada ECG type 1
Long QTc-interval (>460 msec) in 12 lead ECG recording
Minor criteria
Valuable as the major criteria if the anamnesis suggests a rhythmogenic etiology.
Asymptomatic, inadequate sinus bradycardia or bradycardic atrial fibrillation
Pre-excitation (short PR-intervals, delta waves)
AV-block II Mobitz 1 and long AV-block 1
Paroxysmal supraventricular tachycardic arrhythmias and atrial fibrillation
Short QT-c interval (<340 msec)
Brugada ECG type 2 and 3
Negative right precordial T waves, epsilon waves
Clinical and laboratory findings
Unexplained low systolic blood pressure (<90 mm Hg)
Pulsus paradoxus or pulsus alternans
Persisting sinus bradycardia (<40 beats/min), several sinus pauses >3 sec or sinoatrial block in a non-sporty person
Heart murmur of unknown etiology
Pathologic cardiac biomarkers
Signs of heart decompensation
Less frequent cardiac diseases (e.g., cardiomyopathies and channelopathies)
Myocarditis
Stress-cardiomyopathy (Takotsubo)
Cardiac tumors, sarcoidosis
Amyloidosis, infiltrative cardiopathies
Hypertrophic obstructive cardiomyopathy
Several cardiac channelopathies
Generally non-cardiac etiology
Anamnesis
Headache or abdominal pain
The occurrence of syncope in the lying position is a red flag for non-cardiac causes (often neurological pathology)
Clinical findings

Bleeding (e.g., from the intestinal tract) usually argues against a cardiac etiology unless the patients were being treated with antiplatelets or anticoagulants, with dual or triple therapy for coronary heart disease and atrial fibrillation.
Signs of pneumothorax, pulmonary embolization, and leg thrombosis

Table 2 Causes of cardiac syncope.

Inflow/outflow tract obstruction
Severe valvular stenosis, dysfunctional prosthetic heart valve
Hypertrophic obstructive cardiomyopathy
Subvalvular aortic stenosis
Rarely, myxoma or other cardiac tumors
Pulmonary emblism or severe pulmonary arterial hypertension
Heart failure with a decrease in ejection fraction (LVEF <40%)
Many cardiac diseases (e.g., infarction, cardiomyopathy, and congenital structural heart diseases)
Oncologic post-radiation and chemotherapeutic treatment
Left ventricular aneurysm, left ventricular compaction cardiomyopathy
Rare, infiltrative cardiomyopathies (e.g., amyloidosis, hemochromatosis)
Rare diseases that significantly reduce right ventricular function
Rare, primary, or metastatic cardiac tumors
Coronary artery pathology
Ruptured plaques, thrombosis, embolization, dissection, muscular bridging, and abnormal origin of the coronary artery from the pulmonary circulation
Cardiac compression
Pneumothorax, pleural effusion (especially malignant), and pulmonary metastases
Pericardial pathology
Tamponade; calcified pericardium
Decrease in intravascular volume
Hemorrhage, e.g., following aortic dissection
Ventricular rupture and other rare conditions

Table 3 Anamnesis for syncope.

Physical activity:	At rest; walking; during or after physical effort or playing a sport?
Body Position:	Lying; sitting; standing, after standing up?
Time:	In the morning; postprandial, at night?
Triggers:	Emotion, cough, abdominal pressing, external temperature, fever, pain, head elevation/rotation, compression of the neck?
Prodromata:	Without. With: ● Chest pain, dyspnea, palpitations, dizziness (*cardiac etiology*)

Consequences:	● Giddiness, sweating, heat, nausea, vomit, visual disturbances, tremor (vasovagal etiology) Tongue biting, urine/stool loss, traumatic lesions due to the fall?
Postdrome:	Information about the occurrence, required time (<5 min) to regain consciousness, cardiac symptoms, focal neurologic deficits, following confused mental status?
Third person's observation:	Muscle tone, convulsions, abnormal eye movements, pallor, duration of the episode, time to recover consciousness?
History of a previous syncope:	Number, frequency, triggers, prodromata, course, trauma following the syncope?
Medical regimen:	New medications? Changed dosage? Drugs favoring orthostasis (diuretics, antihypertensive drugs, vasodilators, some antidepressants, benzodiazepines, antipsychotic drugs, etc.)? Drugs affecting bathmotropic, dromotropic, or chronotropic cardiac activity? Drugs prolonging the ECG QT interval?
Noxious substances/circumstances:	Alcohol consumption or withdrawals, narcotics, psychic stress?
Family anamnesis:	Sudden death (especially in young relatives), cardiac pathologies?
Personal medical history	Coronary heart disease, heart valve pathology, arrhythmias, heart failure, cardiac surgery, implanted pacemaker or ICD, hypercoagulability or prothrombotic condition, pulmonary embolism, epilepsy, fever with convulsions?

4. Therapy of Cardiac Syncopes

The treatment depends on the etiology of the cardiac syncope [1-6, 11, 12]. Urgent surgery might be necessary for a dysfunctional prosthetic heart valve or a ruptured aortic aneurysm; a rapid intervention (e.g., with TAVI) might be required for aortic stenosis; cardioversion and/or the implantation of a pacemaker or ICD for cardiac arrhythmia, pericardial drainage for cardiac tamponade, and urgent revascularization in the presence of an acute coronary syndrome might be required. Finally, pulmonary embolism and acute heart failure might require special treatment.

5. Example of a Geriatric Patient with Cardiac Syncope

An 80-year-old female patient (height 167 cm and weight 59 kg) was referred for a second syncope that had occurred while she was sitting and not in a postprandial phase. Six months before the presentation, she had the first syncope while going to the toilet at around 2 a.m. Her physician had diagnosed a vasovagal reaction. She was a retired sports teacher and was physically fit. When she was 5, she had undergone surgery for a patent ductus Botalli, an atrial septal defect Type 1, and severe pulmonary valve stenosis. She was treated for arterial hypertension and had

moderate chronic kidney disease (stage 3b). Medications included torsemide, metoprolol retard, azilsartan, and calcium vitamin D for osteoporosis.

Upon examination, her body temperature was 36.1°C, respiratory rate was 18 breaths/min, blood pressure was 148/84 mm Hg, and pulse was 60 beats/min, slightly irregular. The results of a complete blood count and tests of coagulation were normal, as were serum levels of sodium, potassium, carbon dioxide, glucose, total bilirubin, aminotransferases, and amylase. The eGFR was abnormal (eGFR cystatin C 34 mL/min/1.73 m^2). Urinalysis revealed that protein (50 mg per deciliter) was present in the urine. Cardiac troponin was normal (<0.04 ng/mL) and the NT-proBNP was elevated (2,340 ng/mL, corrected for eGFR 825 ng/mL). A chest radiograph revealed cardiac biventricular enlargement and signs of mild pulmonary venous congestion. An ECG (Figure 1) showed sinus rhythm at 60 beats/min with isolated ectopic beats, a 1st-degree AV-block Mobitz 1, a left anterior fascicular block, broad QRS complexes, Q-waves from V_1 to V_4, ST-upsloping in $V_{1–4}$, light down-sloping in I and V_6, biphasic T waves in I and V_6, and a long QTc-interval. The echocardiogram (in color Doppler ultrasound examination after injecting saline) detected multiple post-surgical reflexes in the atrial septum but no interatrial shunt. The left atrium was slightly enlarged (area 20 cm^2), but the right atrium was normal in size. The parasternal long-axis and short-axis views showed a dilated (EDd 54 mm and EDV 148 mm) left ventricle, with remodeling (eccentric moderate hypertrophy with posteroinferior predominance, mass index 78 g/m^2), hypokinesia of the interventricular septum, with reduced mid-range systolic function (LVEF 45%) and diastolic dysfunction grade II. The right ventricle was hypertrophied, with a delayed contraction of the free wall and with normal function (RVEF 45%, TAM 25). The aortic valve was calcified with fibrin deposits, without stenosis (maximal pressure 8 mm Hg) and with minor regurgitation (due to reduced coaptation of the mitral leaflet). The calcified mitral valve showed severe regurgitation (due to dilation of the annuls and reduced coaptation of the anterior leaflet). The tricuspid valve was myxoid, with a dilated annulus and light regurgitation (right ventricle/right atrial gradient 40 mm Hg). The pulmonary valve was myxoid, with minor regurgitation. The aorta was sclerotic with normal dimensions. The pulmonary artery was dilated (24 mm). There was no pericardial effusion. During a 48-h dynamic ECG the patient did not have cardiac symptoms. The sinus rhythm was normal with a normal HR variability: 46 minimal and 102 maximal HR (median: 64 beats/min). There were no pathologic RR-intervals (maximal duration 3 sec). The QRS-complexes were broad, and the QT-interval was permanently increased (up to 472 msec) due to the known ventricular pathology. There were <1% supraventricular ectopic beats and <2% isolated ventricular ectopic beats, with eight supraventricular tachycardic runs. The longest self-limiting supraventricular tachycardia run (eight ectopic beats with an HR of 161/min) is shown in Figure 2. The cause of the two syncopes was considered to be cardiac due to multiple conditions: post-surgical right atrial scars, fibrotic and electrophysiologic abnormalities due to congenital heart disease, abnormal anatomic and functional ventricles, and degenerative valval pathology. Because of the patient's age, conservative therapy was chosen. The dosage of torsemide and metoprolol was reduced, and azilsartan was stopped. Valsartan/sacubitril, eplerenone, and dapaglifozine were added to the pharmacologic regimen. A mild degree of ambulatory rehabilitation for heart failure was prescribed. At the one-year follow-up, the patient was healthy, without cardiac symptoms or syncope. The NT-proBNP decreased (840 ng/mL, corrected for the eGFR 128 ng/mL). A dynamic ECG detected <2% supraventricular and <1% ventricular ectopic beats without runs. The echocardiogram showed a significant improvement with a decrease in the left ventricular

dimensions (EDd 49.5 mm and EDV 112 mm), an increase in systolic ejection ((52%), and a decrease in mitral regurgitation from a severe to a moderate degree. An improvement in cardiac function might reduce the occurrence of syncope.

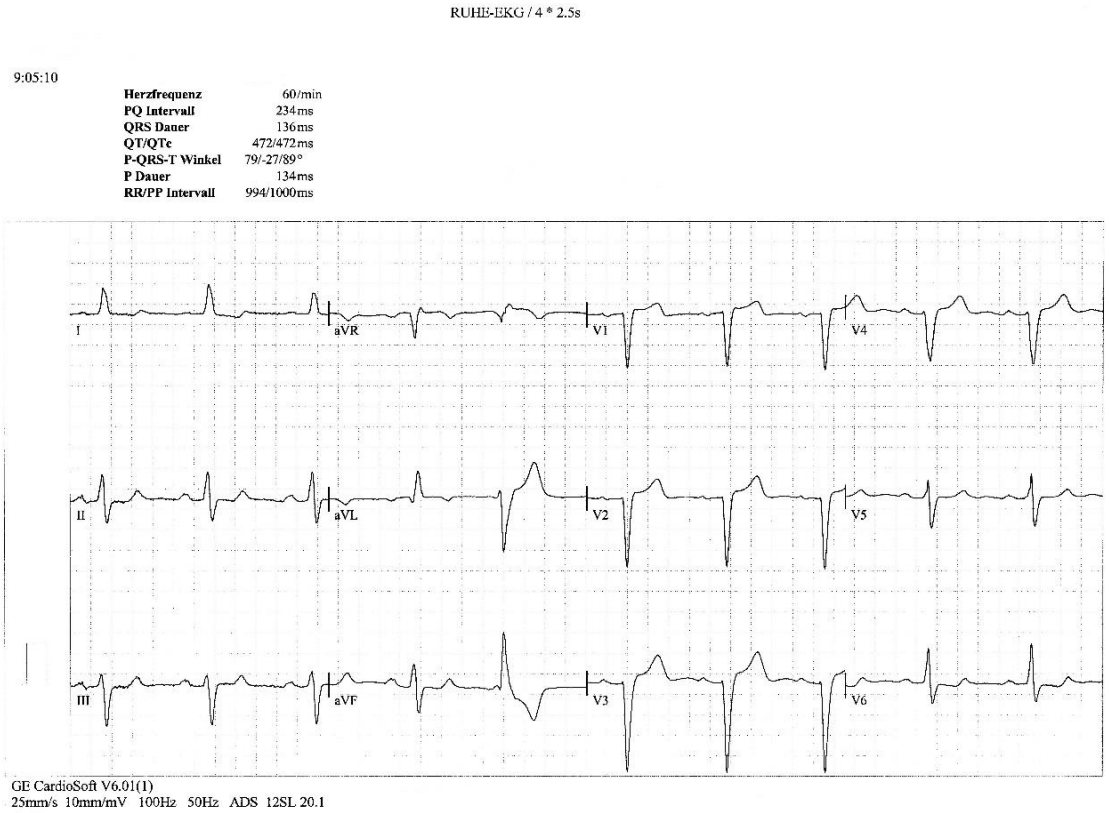

Figure 1 ECG of an 80-year-old female patient with cardiac syncope. The ECG shows a sinus rhythm of 60 beats/min with isolated ventricular ectopic beats, a 1st-degree AV-block Mobitz 1 (PR interval 234 msec), a left anterior fascicular block (R axis –27°), broad QRS complexes *(*QRS duration 136 msec), Q-waves from V_1 to V_4, ST-upsloping in $V_{1–4}$, light down-sloping in I and V_6, biphasic T waves in I and V_6, and a long QTc-interval (472 msec).

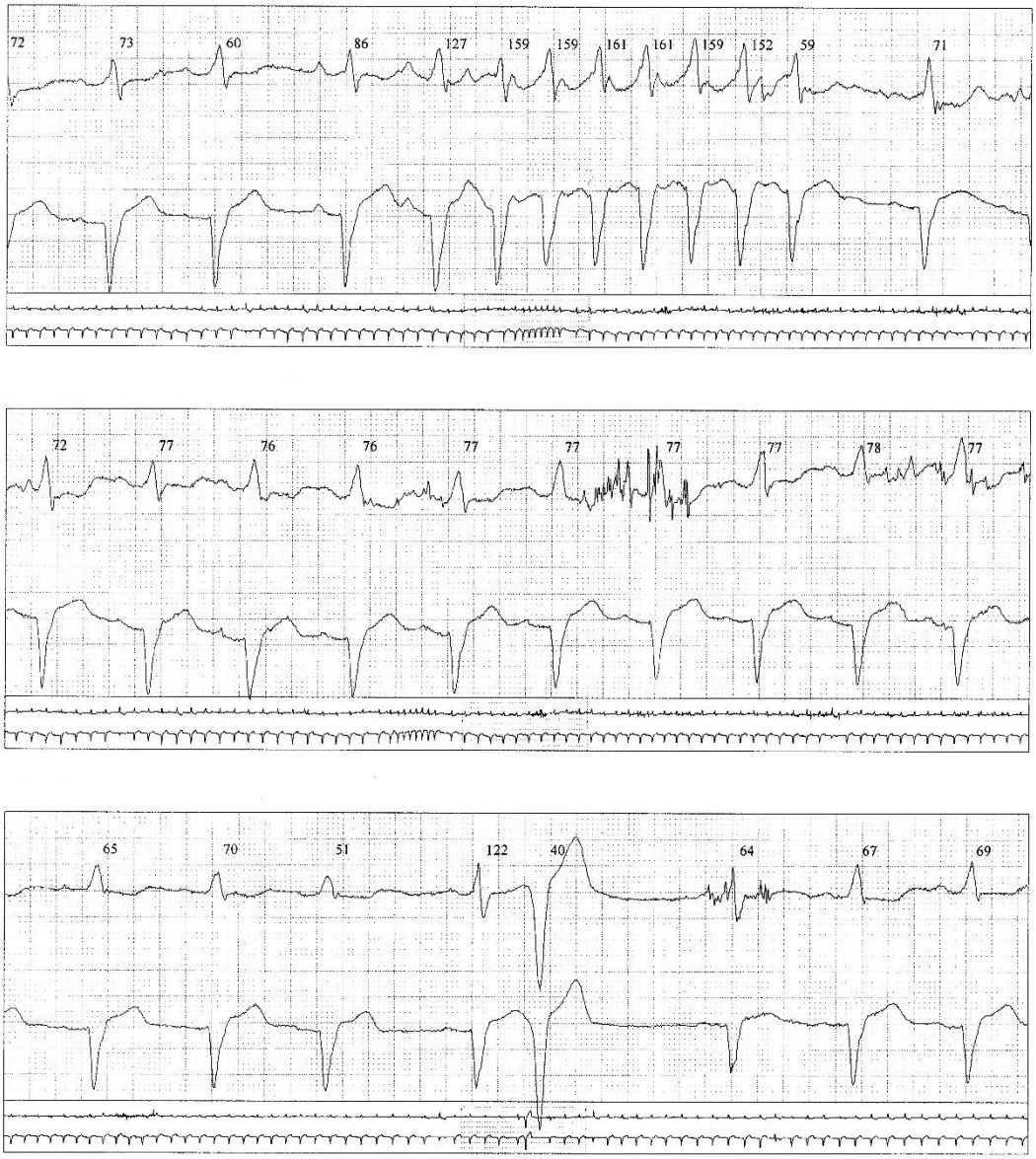

Figure 2 Dynamic ECG of an 80-year-old female patient with cardiac syncope. The tracing shows isolated ectopic beats and a self-limiting supraventricular tachycardia (broad QRS morphology because of the cardiac pathology) with eight ectopic beats and an HR of 161/min.

6. Discussion

Syncopes are frequent in geriatric patients because of the coexistence of many pathophysiologic processes, including age-related physiological changes, co-morbidities, medication, and prolonged bed rest. Syncopes might induce fall-related physical injury and affect the quality of life. The overlap between falls and syncope and the interaction between syncope and frailty complicate management. Available clinical guidelines for the management of syncope focus on diagnosis, investigation, and treatment in a general population but are insufficient to address the problems of elderly patients. A comprehensive geriatric approach that considers

individual functional and cognitive capacities, as well as medical and psychosocial aspects, would be more appropriate. Modern medicine can be used to treat some geriatric patients and should be offered whenever possible.

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Author Contributions

Both GC and SP collected the references. GC wrote the manuscript and all cardiological data. SP checked the general data and delivered the patient.

Competing Interests

The authors have declared that no competing interests exist.

References

1. Soteriades ES, Evans JC, Larson MG, Chen MH, Chen L, Benjamin EJ, et al. Incidence and prognosis of syncope. New Engl J Med. 2002; 347: 878-885.
2. Kenny RA, Bhange J, King Kallimanis BL. Epidemiology of syncope/collapse in younger and older Western patient populations. Prog Cardiovasc Dis. 2013; 55: 357-363.
3. Wong CW. Complexity of syncope in elderly people: A comprehensive geriatric approach. Hong Kong Med J. 2018; 24: 182-190.
4. Brignole M, Moya A, de Lange FJ, Deharo JC, Ellior PM, Fanciulli A, et al. Practical instructions for the 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J. 2018; 39: e43-e80.
5. Ungar A, Mussi C, Del Rosso A, Noro G, Abete P, Ghirelli L, et al. Diagnosis and characteristics of syncope in older patients referred to geriatric departments. J Am Geriatr Soc. 2006; 54: 1531-1536.
6. Knuuti J, Wijins W, Saraste A, Capodanno D, Barbato E, Funck Brentano C, et al. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes: The Task Force for the diagnosis and management of chronic coronary syndromes of the European Society of Cardiology (ESC). Eur Heart J. 2020; 41: 407-477.
7. Macedo P, Leite RL, Asirvatham SJ, Hachul DT, dos Santos Net LL, Shen WK. Head up tilt testing: An appraisal of its current role in the management of patients with syncope. J Atr Fibrillation. 2011: 4: 333.
8. Christ M, Geier F, Popp S, Singler K, Smolarsky A, Bertsch T, et al. Diagnostic and prognostic value of high-sensitivity cardiac troponin T in patients with syncope. Am J Med. 2015; 128: 161-170.e1.
9. du Fay de Lavallaz J, Badertscher P, Nestelberger T, Zimmermann T, Miró Ò, Salgado E, et al. B-type natriuretic peptides and cardiac troponins for diagnosis and risk-stratification of syncope. Circulation. 2019; 139: 2403-2418.
10. Sandhu RK, Sheldon RS. Are cardiac biomarkers the key to solving the syncope mystery? Circulation. 2019; 139: 2419-2421.

11. Albassam OT, Redelmeier RJ, Shadowitz S, Husain AM, Simel D, Etchells EE. Did this patient have cardiac syncope? The rational clinical examination systematic review. JAMA. 2019*;* 321*:* 2448-2457.
12. ESC Congress- The digital experience. ESC Congress; 2021 August 27-30.