Chapter 56
Dizziness and Syncope

Girish Narayan

Key Points
- Dizziness and syncope are common symptoms that have multiple differential diagnoses.
- History and clinical examination are crucial to determine the aetiology of dizziness or syncope.
- Directed and focussed investigations are required to ascertain the diagnosis.

Introduction

Dizziness is a frequent but complex symptom that patients present with to the emergency department. The term "dizziness" is used by patients to describe a wide-ranging group of symptoms from the benign to the life threatening. Hence, the determination of aetiology and management requires precision.

The prevalence of dizziness and syncope among patients varies with age. Approximately 3 % of patients present with dizziness and the incidence of syncope vary from 1 to 5 %, with an increasing prevalence among patients aged greater than 50 years, along with more likelihood of dangerous causes [1, 3, 5].

G. Narayan, MBBS, MD (Med), MCEM
The Department of Emergency Medicine, St. John's Medical College Hospital, Sarjapur Road, Koramangala, Bangalore 560 034, India
e-mail: girishn.md@gmail.com

S.S. David (ed.), *Clinical Pathways in Emergency Medicine: Volume I*,
DOI 10.1007/978-81-322-2710-6_56

Definitions

(A) **Syncope**
Syncope is defined as a transient loss of consciousness accompanied by loss of postural tone followed by recovery to a normal neurological state [4–6].

(B) **Dizziness**
Dizziness as a symptom is often vague and inexact. From a patient's point of view, it can be difficult to explain and for a physician its analysis can be vexing.

Generally, the term dizziness is used to describe one or a combination of the following [7, 8, 10]:

1. Vertigo: An illusory rotatory or a spinning sensation
2. Light-headedness/presyncope: An impending sensation of faintness or feeling woozy
3. Imbalance/disequilibrium: A sensation of unsteadiness while walking or loss of posture

Pathophysiology

Syncope occurs due to cerebral or brainstem hypoperfusion resulting in the sequelae of loss of consciousness and loss of postural tone.

The perception of balance is maintained by sensory input from visual, proprioceptive and vestibular systems which are integrated via the cerebrum, cerebellum and brainstem to the subsequent motor outflow. Any disturbance in any of these systems or their connections can result in the symptom of dizziness or vertigo [3, 5, 7, 11].

Aetiology

The causes of dizziness and syncope are many (Table 56.1). The mechanisms through which they occur may overlap. Vestibular and otologic causes usually cause vertigo. Vertebrobasilar insufficiency and stroke like lateral medullary and anterior inferior cerebellar artery syndromes and cerebellar haemorrhage/infarction may present with vertigo, imbalance or even syncope at times. Cardiovascular causes usually cause symptoms as a result of decreased perfusion. Systemic causes and medications result in dizziness or syncope by a number of mechanisms, and there may be an overlap as with hypoglycaemia secondary to Addison's disease or drugs. Medications and toxins will usually cause symptoms through various pathophysiological mechanisms but may precipitate other aetiologies, especially in the elderly [2–5, 10, 12].

Table 56.1 Causes of dizziness and/or syncope [2–5, 10, 12]

Vestibular or otologic[a]	Neurologic	Systemic	Drugs and toxins
(a) Benign paroxysmal positional vertigo (b) Vestibular neuronitis (c) Labyrinthitis (d) Meniere's disease (e) Foreign body in ear (f) Otitis media (g) Otosclerosis (h) Perilymphatic fistula (i) Ramsay Hunt syndrome (j) Concussion (post-head trauma) (k) Motion sickness	(a) Acoustic neuroma (b) Cerebellopontine angle tumours (c) Migraine (d) Stroke (e) Subarachnoid haemorrhage (f) Multiple sclerosis (g) Seizure/postictal (h) CNS infections (i) Peripheral neuropathy (j) Myelopathy **Cardiovascular** (a) Acute coronary syndromes (b) Hypertensive emergency/urgency (c) Aortic dissection (d) Angina (e) Arrhythmias (f) Valvular lesions (g) Cardiomyopathy (h) Pulmonary embolism/ hypertension (i) Subclavian steal syndrome	(a) Anaemia (b) Orthostatic hypotension (c) Blood loss (d) Polycythaemia (e) Hyperviscosity syndrome (f) Dehydration (g) Chronic renal failure (h) Vasovagal (i) Trauma (j) Concussion (k) Cervical spine disorders (l) Addison's disease (m) Hypothyroidism (n) Diabetes insipidus (o) Hypoglycaemia (p) Hyperventilation (q) Psychogenic (anxiety, stress)	(a) Aminoglycosides[a] (b) Erythromycin (c) Minocycline (d) Fluoroquinolones (e) Antimalarials (f) Diuretics (g) Beta blockers (h) Vasodilators (i) Sympatholytics (j) Digoxin (k) Salicylates (l) NSAIDS (m) Cisplatin (n) Vincristine (o) Phenytoin (p) Antidepressants (q) Anticonvulsants (r) Antipsychotics (s) Anxiolytic/sedative (t) Mood stabilizers (u) Insulin (v) Oral hypoglycaemic agents (w) Substances of abuse (alcohol, cannabis, cocaine) (x) Carbon monoxide

[a]Aetiologies particularly presenting with the symptom of vertigo

Clinical Features (Tables 56.2 and 56.3)

Physical examination remains the backbone of evaluation of dizziness and syncope. Patients may have returned to their presymptom state at the time of examination; therefore, a thorough examination is required to establish differentials.

Neuro-cardiogenic or vasovagal syncope is a type of situational syncope which can be precipitated by number of triggers. Emotion, pain and uneasy high-stress situations can be the inciting factor. There may be a prodrome of light-headedness, blurring of vision or diaphoresis. The event occurs when the patient is in upright posture, gradual in onset followed by a rapid recovery once the patient is supine. The pathogenesis is due to increased sympathetic outflow and sudden vagal modulation [4, 11].

Benign paroxysmal positional vertigo (BPPV) is the most common aetiology of vertigo. It occurs when otoconia (crystals of calcium carbonate) are dislodged from the utricle and drift into the semicircular canals, usually the posterior. The patient will experience vertigo when turning the head towards the affected side and it is

Table 56.2 Symptomology [3, 7, 9, 11]

	Syncope	Dizziness
Preceding event	Exertion, heat exposure, micturition, defecation, emotional stress	Prior history of an ear disorder may be present if a peripheral cause
Event	Patients will usually describe it as fainting or passing out with loss of muscle strength Position: standing/supine/sitting Onset: abrupt/gradual	Patients will describe it as a feeling of unsteadiness on standing or walking or whirling or spinning when stationary which may be aggravated on head movement Duration: several hours to days
Post-event	Spontaneous recovery, duration and rate to be inquired to rule out postictal state	Maybe episodic with spontaneous recovery with symptom-free intervals. Can be persistent also
Associated symptoms	Chest pain, dyspnoea, palpitation, sweating	Nausea, vomiting, ear disturbances, head or neck injury, neurologic symptoms
Past history	Coronary artery disease, CVA/TIA, diabetes, hypertension, arrhythmias	Coronary artery disease, CVA/TIA, diabetes, hypertension, arrhythmias, trauma, ENT disorders, drugs

Table 56.3 Examination of patients with dizziness or syncope [3–5, 7, 10, 11]

System	Signs	Possible aetiology
General physical examination		
Pulse: rate and rhythm (all peripheral pulses to be examined)	Tachycardia, bradycardia, arrhythmias, pulse volume, asymmetrical pulses, carotid bruits	Cardiac, vascular, fluid overload, hypovolemia, infective
Respiratory rate and volume	Tachypnea	Hyperventilation, hypoxia, pulmonary embolus, AMI, heart failure
Blood pressure	Hypotension, hypertension, orthostatic hypotension, variation in limbs	Shock, hypertensive crisis, hypovolemia, vascular
Temperature	Increase or decrease	Fever, infection, shock
Appearance	Pallor	Anemia, shock, blood loss, heart failure
	Cyanosis	
Neurological system	Higher mental functions	Stroke, raised ICP, localization of neurological lesion (cerebrum/cerebellar/brainstem/spinal cord)
	Papilloedema	
	Focal neurological deficits	
ENT	Peripheral nystagmus: horizontal, sudden, episodic, short duration, fatigable, triggered by head movement	Peripheral or central vertigo Otitis media, trauma, BPPV
	Central nystagmus: Any direction, insidious, constant , sustained duration, nonfatigable, nonprovocative	
	Tympanic membrane abnormalities, deafness, Dix Hallpike Test	

Table 56.3 (continued)

System	Signs	Possible aetiology
Cardiovascular system	Raised JVP	Heart failure, pulmonary embolus, tamponade, cardiomyopathy, valvular pathology, pericarditis
	Apical impulse displaced	
	Heart sounds muffled, rubs, murmurs, S3, S4	
Respiratory system	Crepitations	Pneumonia, pulmonary oedema, haemothorax
	Decreased breath sounds	
Abdomen	Pulsatile mass	Aortic aneurysm, abdominal or pelvic trauma, GI bleed
	Tenderness, abrasions	
	Melaena	
Genitourinary	Bleeding per vagina	Ectopic pregnancy, genitourinary trauma
Extremities	Pedal oedema	Fluid overload, heart failure, trauma
	Swelling, tenderness	

usually episodic. It is diagnosed with Dix-Hallpike test which is done by placing the patient in a supine position with his head hanging off the edge of the examination table. The patient's head is then turned to either side rapidly and held in that position and is observed for nystagmus which confirms BPPV. The nystagmus usually starts after a few seconds of latency and will reduce with repeat testing and time. In central causes, there will be no latency or reduction with time. Meniere's disease is poorly understood pathophysiologically. Increased endolymph production resulting in elevated pressure is suspected. Episodes of rotational vertigo lasting for hours accompanied with nausea, vomiting, hearing loss and tinnitus can occur. Nystagmus will be present but it will not be related to position.

Vestibulitis typically presents with severe vertigo peaking in hours, plateauing over the next few days and may persist for weeks. Symptoms can worsen with change in position. Associated symptoms such as nausea and vomiting may be present but auditory symptoms are absent. When hearing loss is present, labyrinthitis is to be considered. Nystagmus will be spontaneous towards the affected side [3, 7, 10, 13].

Investigations

Studies estimate that physical examination and history are able to potentially diagnose 40 % of causes of syncope and dizziness. Therefore, investigating patients becomes imperative in most emergency rooms. Judicious and targeted investigations

Table 56.4 Directed investigations in patients with dizziness or syncope [4, 5, 7, 10, 11, 13]

Investigation	Possible diagnosis
ECG	Ischaemia, arrhythmias
Blood sugar	Hypo-/hyperglycaemia
Haemoglobin, haematocrit	Blood loss, anaemia
WBC count	Infection, sepsis
Urine pregnancy test	Pregnancy, ectopic
Beta HCG	
Blood gas	Hypoxia
Electrolytes	Metabolic abnormalities, hypo-/hyperkalaemia, kidney injury
Cardiac enzymes	Acute coronary syndromes
B-natriuretic peptide	Heart failure
D-dimer	Pulmonary embolism
Drug screen (urine/blood)	Medication/toxin identification
X-rays	Trauma, pneumonia, aortic aneurysm
USG (FAST, RUSH)	Trauma, shock, ectopic pregnancy
ECHO	Acute myocardial infarction, valvular pathologies, tamponade
Doppler	Aneurysm, DVT in a suspected case of PE
CT	Trauma
	Pulmonary embolism
	Stroke, subarachnoid haemorrhage
MRI	Stroke (particularly posterior cranial fossa and brainstem pathologies), space-occupying lesions

complimenting a thorough examination are essential for a quick diagnosis and to limit costs for a patient (Table 56.4).

When investigating patients in the emergency department, life-threatening causes need to be identified quickly. Without a supportive history or positive clinical examination, diagnostic tests can have a low yield. An ECG is still recommended though yield is low. It is used extensively in most risk stratification guidelines. ECG in addition to history and examination helps increase the yield of laboratory investigations in particular cardiac enzymes [4, 5].

Ultrasound has proven to be a very useful tool in identification of free fluid in trauma. Echocardiography is useful in patients with structural heart disease presenting with syncope or dizziness. Identification of impaired ejection fraction helps manage patients at serious risk. Other investigations help an emergency physician risk stratify the patient after a cause has been determined by a clinical examination or by radiological evaluation [5, 7].

Neuroimaging in patients with dizziness and syncope should be guided by clinical examination. During evaluation of a possible cerebellar stroke, imaging is vital. Though CT scan is the most frequently used modality due to its easy availability, it cannot be used conclusively to rule out certain pathologies. Physicians should

recognize the limitations of CT in evaluation of acute strokes particularly in the posterior cranial fossa or brainstem. When available, MRI with diffusion weighted imaging should be performed as it provides a higher sensitivity than CT [7, 10, 11, 13].

Treatment and Disposition

Management of patients with dizziness and syncope will essentially be directed by the diagnosis. Treatment will also be dictated by the patient's haemodynamic status. Primary stabilization of vital parameters with identification of risk factors will begin in the emergency department. Strokes, acute coronary syndromes, arrhythmias and suspected vascular emergencies will require intensive care management after stabilization in the emergency room. Unambiguous diagnoses will usually follow definitive management pathways [4, 7, 11].

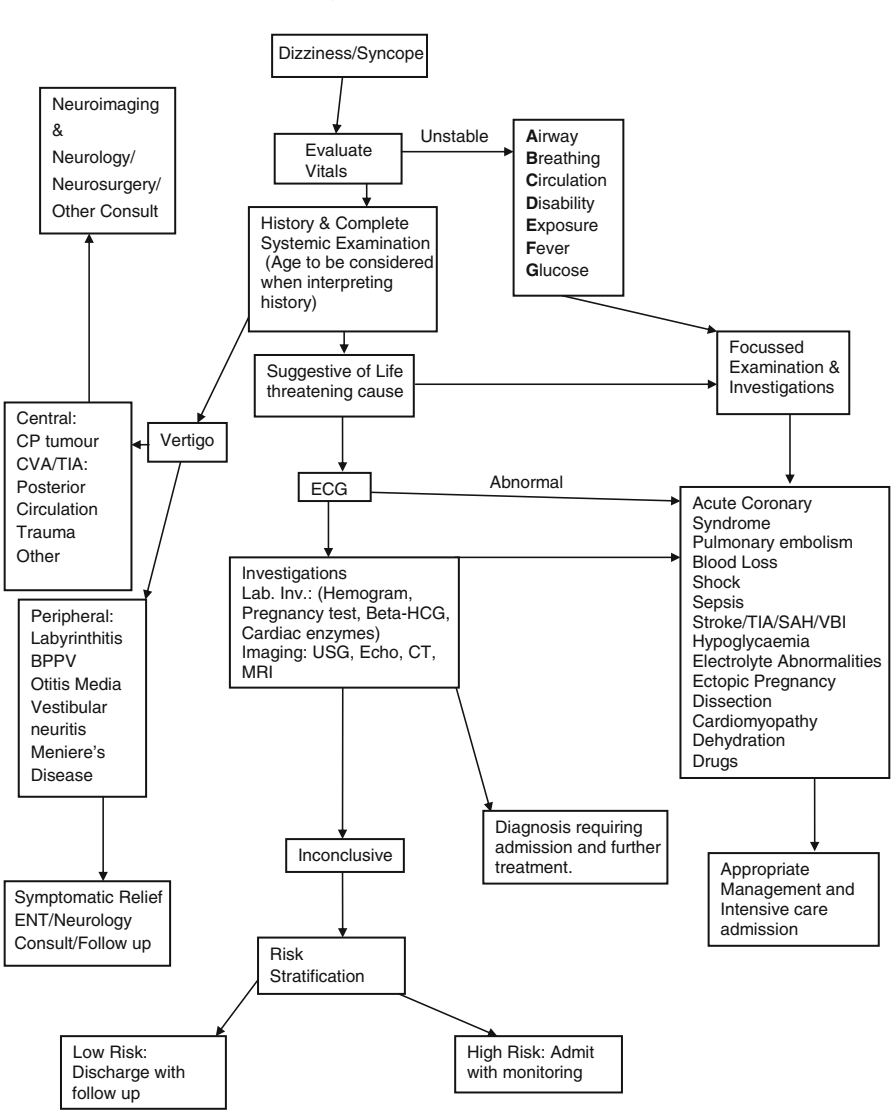
Benign paroxysmal positional vertigo can be cured by Epley's particle repositioning manoeuvre along with vestibular suppressants for acute symptoms of nausea and vomiting. Intravenous ondansetron is considered safe and effective for these symptoms. For patients presenting with severe vomiting, promethazine and benzodiazepines can be considered, but sedation is a frequent side effect. For other causes with acute symptoms, short-term therapy with these agents can be considered till definitive treatment for the cause is implemented [3, 7].

Patients in whom the diagnosis is elusive will require risk stratification and then appropriate disposition. A number of syncope risk stratification guidelines have been developed, but no set of rules have achieved complete validation to rely on. The focus of these rules is to admit high-risk patients and minimize admissions in low-risk patients. Currently, all rules either inadvertently miss risk stratifying some high-risk patients to reduce inpatient numbers or unnecessarily identify many patients as potential dangerous outcomes resulting in over admission. The various rules suggested are the San Francisco Syncope Rule (SFSR), the Osservatorio Epidemiologico sulla Sincope nel Lazio (OESIL) score, the European Guidelines in Syncope Study (EGSYS) score and the Risk Stratification of Syncope in the Emergency Department (ROSE) score. None of these rules have been completely validated. However, the common minimum criteria that have been present across all rules are the following:

- Older age
- History suggestive of structural heart disease or arrhythmias
- Unrelenting abnormal vitals
- Abnormal ECG [4–6, 11]

Patients who are discharged after evaluation for dizziness or syncope should be asymptomatic at discharge and advised follow up with appropriate departments.

Clinical pathway to manage dizziness and syncope



References

1. Newman-Toker DE, Hsieh Y-H, Camargo C a, Pelletier AJ, Butchy GT, Edlow JA. Spectrum of dizziness visits to US emergency departments: cross-sectional analysis from a nationally representative sample. Mayo Clin Proc. 2008;83(7):765–75.
2. Lo AX, Harada CN. Geriatric dizziness: evolving diagnostic and therapeutic approaches for the emergency department. Clin Geriatr Med. 2013;29(1):181–204.
3. Kulstad C, Hannafin B. Dizzy and confused: a step-by-step evaluation of the clinician's favorite chief complaint. Emerg Med Clin North Am. 2010;28(3):453–69.
4. Ouyang H, Quinn J. Diagnosis and evaluation of syncope in the emergency department. Emerg Med Clin North Am. 2010;28(3):471–85.
5. Benditt DG, Adkisson WO. Approach to the patient with syncope: venues, presentations, diagnoses. Cardiol Clin. 2013;31(1):9–25.
6. Kessler C, Tristano JM, De Lorenzo R. The emergency department approach to syncope: evidence-based guidelines and prediction rules. Emerg Med Clin North Am. 2010;28(3): 487–500.
7. Chang AK, Olshaker JS. Dizziness and vertigo. In: Marx JA, Hockbereger AS, Walls RM, editors. Rosen's Emergency Medicine – Concepts and Clinical Practice. 8th ed. Philadelphia: Elsevier Inc; 2014.
8. Cherian N. Dizziness. In: Carey WD, editor. Current Clinical Medicine. 2nd ed. Philadelphia: Elsevier; 2010.
9. Kerber KA, Baloh RW. Neuro-otology: diagnosis and management of neuro-otological disorders. In: Daroff RB, Fenchiel GM, Jancovic J, Mazziotta JC, editors. Neurology in clinical practice. 6th ed. Philadelphia: Elsevier; 2012.
10. Kerber KA. Vertigo and dizziness in the emergency department. Emerg Med Clin North Am. 2009;27(1):39–50.
11. Lorenzo RA De. Syncope. In: Marx JA, Hockbereger AS, Walls RM, editors. Rosen's emergency medicine – Concepts and Clinical Practice. 8th ed. Philadelphia: Elsevier; 2014.
12. Shoair O a, Nyandege AN, Slattum PW. Medication-related dizziness in the older adult. Otolaryngol Clin North Am. 2011;44(2):455–71.
13. Kutz JW. The dizzy patient. Med Clin North Am. 2010;94(5):989–1002.