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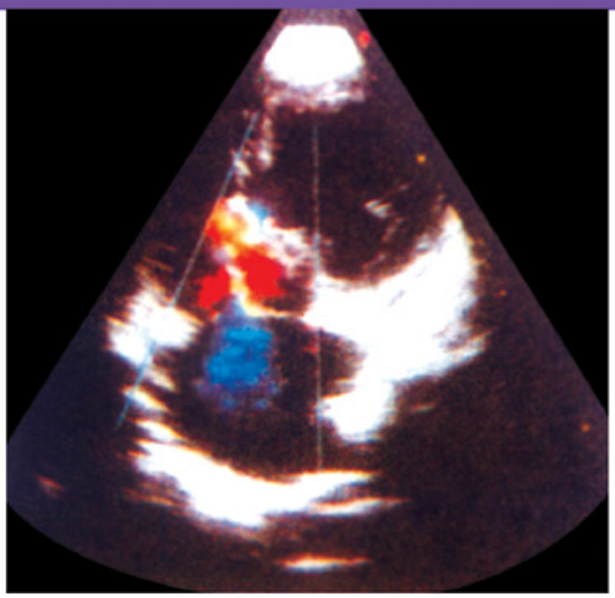
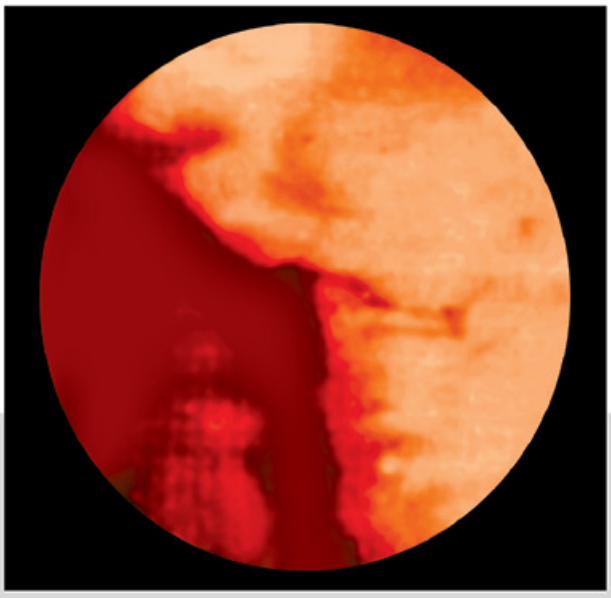
Differential Diagnosis and Medical Therapeutics





A Treatise on Clinical Medicine with 75 years Experience

5th Edition

P Siva Rama Krishna Rao

JAYPEE

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4 Syncope

Syncope (Greek—syn = with; koptein = to cut off) is a sudden, transient and complete loss of consciousness, due to decreased global cerebral blood flow (to less than half of the normal) for about 5 seconds. If the reduced cerebral perfusion is prolonged for 15 seconds, a convulsion occurs (isolated fit). This is a form of hyperacute circulatory failure with consequent anoxia and disturbed brain metabolism. It may be preceded by nausea with clouding of visual fields and generally followed by full recovery within minutes. It is not usually associated with any organic disease of the brain as such. Presyncope is reduced cerebral perfusion with lightheadedness, extreme weakness nausea, dimming vision and ringing in ears, sweating, feeling of dizziness and faintness, visual blurring and swaying wherein person feels a blackout (clouding of vision). In presyncope reduced perfusion is not that much as to produce loss of consciousness presyncope never occurs while lying in the bed. Causes of presyncope are: (i) Hypoglycaemia, (ii) Vasovagal attacks, (iii) Hyperventilation, (iv) Low cardiac output states.

The term faintness indicates feeling of extreme weakness, light-heartedness, followed by visual blurring and swaying; may occur prior to syncope. Hyperventilation, hypoglycaemia due to insufficient blood and oxygen or blackout is sometimes used synonymously with syncope. Faintness (presyncope) wherein cerebral blood flow is not decreased to that extent as to impair consciousness, although there is a sensation of impending loss of consciousness experience; whereas in (syncope) cerebral blood flow is so much decreased as to cause loss of consciousness.

The cerebral blood flow or the cerebral perfusion pressure depends on (i) Cardiac output, (ii) Arterial blood pressure, and (iii) Resistance of the cerebral arteries. (An acute increase in cerebrovascular resistance leads to cerebral vasoconstriction and increase in intracranial pressure).

AETIOPATHOGENESIS

The decreased blood flow to the brain may result from:

- Inadequate venous return and cardiac filling
  - Loss of vasomotor tone with dilatation of systemic arterioles and sudden fall of peripheral resistance as in vasovagal syncope and postural syncope
  - Hypovolaemia as in acute haemorrhage.
  - Severe cough or breath holding spells.
- Inadequate cardiac output
  - Cardiac arrhythmias.
  - Obstruction to left ventricular outflow tract (aortic stenosis and hypertrophic obstructive cardiomyopathy)
  - Acute myocardial infarction
  - Cardiac tamponade
  - Pulmonary embolism
  - Arrhythmogenic right ventricular dysplasia.
- Vascular Insufficiency of outgoing vessels to the brain
  - Hypersensitive carotid sinus
  - Carotid artery obstruction
  - Subclavian steal syndrome (refer to chapter titled 'Vertigo')
  - Cervical Spondylosis- (refer to chapter titled 'Vertigo').
- ***Vascular insufficiency of cerebral vessels:*** Cerebral syncope
- ***Metabolic disturbances:*** Hypocapnia as in hyperventilation, hypoglycaemia
- ***Anoxaemia:*** Obstruction to blood flow due to sudden gravitational effects of anoxia from sudden decompression, e.g. aviation.

TYPES OF SYNCOPE

- Neurocardiogenic syncope
  - Vasovagal syncope (vasodepressor)
  - Carotid sinus syncope (cardioinhibitory)
  - Reflex or situational: Micturition, defecation, coughing.
- Postural syncope or orthostatic hypotension
- Cardiac syncope
- Cerebral syncope
- Metabolic syncope
- Anoxic syncope
- Psychogenic.

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Neurocardiogenic Syncope

This consists of two components, cardioinhibitory (Bradycardia) and vasodepression (hypotension due to peripheral vasodilatation).

Vasovagal Syncope (Vasodepressor)

Vasovagal syncope (vasodepressor): It is the commonest form of neurocardiogenic syncope, which includes the common faint. It is mediated by Bezold-Jarisch reflex (increased vagal tone). The predisposing factors are extreme exertion, prolonged standing, sudden painful or emotional stimuli like fright, environmental at heat or crowding in hot stuffy room. This is most often due to sudden depression of vasomotor centre with loss of vasomotor tone, resulting in pooling of peripheral blood, and consequent fall of blood pressure as well as due to reflex bradycardia. The subject is invariably standing still before the sudden attack.

The onset is not instantaneous (over seconds). Weakness, nausea, sweating, dimness of vision, and ringing in the ears, precede before the subject gradually sinks to the ground without any injury. There is pallor of the body with perspiration. The skeletal muscles are relaxed and flaccidity is elicited. The pulse rate is usually slow and feeble. The systolic blood pressure is around 60 mmHg and the respirations are slow and shallow. Pupils are dilated with sluggish light reflex. Tonic convulsions are not associated but limbs may jerk and the sphincter tone is intact without urinary incontinence. The duration is usually measured in seconds to minutes (<2 min) and recovery is complete without any residual symptoms like headache, drowsiness or confusion. The history of such episodes under similar circumstances is contributory for the diagnosis.

It will not occur while lying down. (Autonomic dysfunction results in bradycardia and hypotension and cerebral hypoperfusion accounts for unconsciousness).

Carotid Sinus Syncope

This result from hypersensitivity of the carotid sinus. which contains sensory end organs (baroreceptors) whose axons are connected with the nerves from the carotid body. The slightest pressure whether due to tight collar or turning the head to one side results in syncope. The attack always occurs in the standing upright position. In a normal person pressure over the carotid sinus causes a slight reduction in the pulse rate of 6 beats per minute and a slight fall in the blood pressure of 10 mm of mercury.

In the carotid sinus hypersensitivity the pressure over the sinus may result in two types of responses.

- Cardiac slowing (vagal type)
- Fall of blood pressure with no effect in the pulse rate (vasodepressor type).

The latter is less common than the former and the symptoms may last longer, say, for a few hours.

The carotid sinus syndrome may be associated with an identifiable lesion like carotid sinus tumour or cervical lymphadenopathy or scars. The diagnosis is confirmed by gently applying light pressure on one side in the region of carotid sinus at the level of superior border of thyroid cartilage for 20 seconds at a time, in supine position; and recording the abnormal responses like (a) Sinus bradycardia or even ventricular asystole of 3 seconds duration, and (b) A decrease in systolic arterial blood pressure to the extent of 50 mmHg and above.

Carotid sinus stimulation is not usually associated with any complications. Nevertheless it may result in cardiac standstill and hemiplegia, particularly in the elderly hypertensive patients with cerebral vascular disease.

Situational Syncope

- ***Visceral reflex syncope:*** Micturition syncope is a type of postural syncope wherein the elderly person empties his distended bladder after arising from a recumbent position.
  It is of abrupt onset and occurs during or after micturition.
- ***Post-tussive syncope:*** After a paroxysm of cough specially in obstructive pulmonary diseases the loss of consciousness occurs due to high intrathoracic pressure, may be even 300 mm of mercury, which is well above the arterial pressure. This results in the total impediment of venous return to the heart and consequent fall of the cardiac output. Simultaneously, the intracranial pressure is increased. Both these factors produce reduction in the pressure of the cerebral vessels with precipitous impairment of cerebral perfusion.
  Other examples are swallow syncope, defecation syncope.

Postural Syncope or Orthostatic Hypotension

Here the syncope occurs when the subject suddenly stands up from a recumbent position, due to impairment of autonomic control of peripheral vasculature. The consequent inadequate reflex vasoconstriction leads to an abrupt fall of blood pressure (postural or orthostatic hypotension). The pulse remains unchanged although orthostatic tachycardia may be associated. The pallor and nausea associated with vasovagal syncope are absent.

This may occur after exercise or a sumptuous meal. In young women, upright posture assumption may result in tachycardia with insignificant change in blood pressure (postural orthostatic tachycardia syndrome and autonomic dysfunction), and autonomic dysfunction. It is a long-standing reproducible symptom.

The mechanism underlined is pooling of blood in the lower extremities, which is normally prevented by:

- Pumping action of the muscles to facilitate the venous return

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- Reflexes which constrict the peripheral arterioles and venules, and
- Acceleration of the heart through carotid and aortic reflexes.

The postural syncope is likely to occur after acute blood loss, in debilitating illnesses, use of hypotensive drugs or diuretics; or near term pregnancy may obstruct inferior vena cava (supine hypotensive syndrome) or autonomic dysfunction. This may be chronic in certain neurological disorders like diabetic neuropathy or syringomyelia or primary autonomic insufficiency (chronic orthostatic hypotension). The postural hypotension in chronic idiopathic orthostatic hypotension is associated with hypohidrosis, impotence, sphincter disturbances, asymmetrical pupils and without compensatory tachycardia. The lack of perspiration may be partial or complete, unilateral or bilateral. There is response (sweating) to pilocarpine although heat does not. Degeneration of post-ganglionic and preganglionic neurons is striking. This may occur as part of multisystem atrophy or associated with Parkinson's disease.

Secondary orthostatic hypotension occurs in neurological disorders as cited above, which involve the autonomic nervous system resulting in autonomic dysfunction. The autonomic insufficiency is suggested by unchanging pulse rate, in spite of hypotension.

This impairment of autonomic control of peripheral vasculature causing postural hypotension is diagnosed by:

- Recording the blood pressure in the supine position as well as immediately after arising from supine to standing position or preferably 5 minutes after standing (after recumbency for 10 min, standing BP is recorded at 3 min intervals) when the systolic blood pressure falls by more than 20 mmHg and diastolic pressure falls by more than 10 mm
- ***Valsalva manoeuvre:*** The subject in supine position exhales for at least 10 against a closed glottis forcibly when the intrathoracic pressure is elevated. It can also be done by blowing up the column of mercury so as to maintain the pressure at 40 mmHg for 10 seconds. Normally the blood pressure falls and pulse rate rises during the strain, and there is an over shoot, i.e. rise of arterial pressure) as well as a reflex bradycardia when the strain is released. In postural hypotension, there is no overshoot of arterial pressure and reflex bradycardia after releasing the strain due to impairment of autonomic reflexes, although the blood pressure which continues to fall during the manoeuvre, gradually returns to original level subsequently.

Cardiac Syncope (Arrhythmias and Structural Mechanical)

In this group, the cardiac output falls as a result of underlying heart diseases like

- Cardiac arrhythmias, i.e. long QT syndrome, sick sinus syndrome, heart block or tachycardia (SVT and VT) **(Figs. 4.1 to 4.3)**.

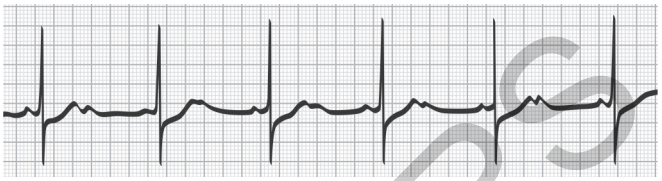

Fig. 4.1A: Second degree heart block (Mobitz II)—Ventricular response to every second sinus impulse only, with constant PR interval (2:1 block).



Fig. 4.1B: Second degree heart block with Wenckebach phenomenon (Mobitz I)—Progressive increase in PR intervals, until ventricular complex is dropped. The same sequence is repeated.

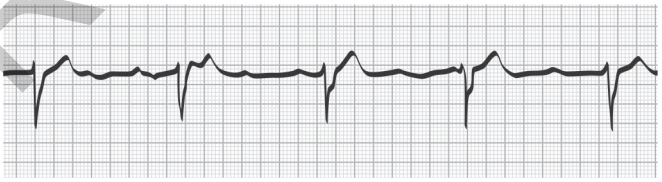

Fig. 4.2: Third degree (Complete) heart block—Ventricular rate is less than the atrial rate. NO constant relationship between the QRS complexes and P waves, as atria and ventricles contract independently.

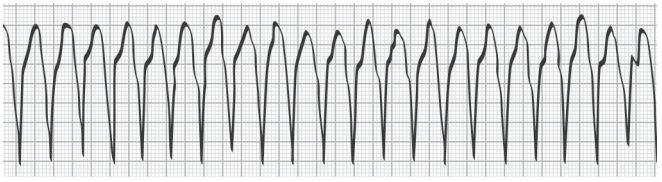

Fig. 4.3A: Ventricular tachycardia—Run of abnormal (wide) and uniform (monomorphic) QRS complexes with approximately regular rhythm. P waves are obscured by ventricular complexes.

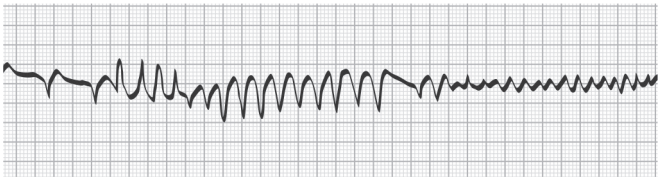

Fig. 4.3B: Ventricular tachycardia degenerates into ventricular fibrillation: Rapid oscillations, varying in amplitude and width, representing QRS complexes with an undulating baseline and absence of apparent isoelectric interval.

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Cardiac standstill and cardiovascular collapse occurs in:
- Pulseless ventricular tachycardia
- Ventricular fibrillation
- Asystole is caused by (i) Failure of conduction, (ii) Damage to ventricle as in myocardial infarction. PEA is caused by reversible conditions, PE, cardiac rupture. In PEA, there is no effective cardiac output despite electrical activity, where as in asystole there is no electrical activity (permanent pacemaker implantation is to be done if survives)
- Pulseless electrical activity (electromechanical dissociation).

Note: Without instituting cardiopulmonary resuscitation (CPR) within 4–6 months is fatal usually.

- Obstruction to outflow tracts, i.e. aortic or pulmonary stenosis and hypertrophic obstructive cardiomyopathy
- Cyanotic heart disease, e.g. Fallot's tetralogy
- Ischaemic heart disease, e.g. acute myocardial infarction
- Pericardial tamponade
- Intracardiac masses, e.g. myxoma or clots
- Hypertrophic obstructive cardiomyopathy (HOCM)
- Pulmonary embolism
- Arrhythmogenic right ventricular dysplasia (ARVD).

Cardiac Arrhythmias (Refer to Chapter Titled 'Palpitations')

- ***Long Q-T syndrome:*** It may be congenital or acquired, which may be secondary to certain drugs or electrolyte disturbances or myocardial infarction. It indicates a risk for Torsades de pointes type of polymorphic ventricular tachycardia. QTc is more than 440 ms.
- ***Stokes–Adams syndrome:*** It is characterized by sudden loss of consciousness without any warning and may fall to the ground. Recovers rapidly. It is due to ventricular asystole or heart rate below 20/min. Constant ventricular rate of 35/min or above does not cause syncope usually. This in turn occurs due to AV block (2nd or 3rd degree) or ventricular tachycardia/fibrillation and occasionally in sick sinus syndrome cerebral blood flow ceases resulting in syncopal attack within 5 seconds and if more than 5 seconds, results in loss of consciousness. And if asystole is prolonged for 2 minutes it may prove fatal. Pallor without pulses breathing continues even though heart has stopped if heart does not restore within 10 seconds, anoxic seizures occur and within 4–6 minutes irreversible damage occurs to brain. It may be paroxysmal occurring several times a day due to reduced cardiac output or at longer intervals, or complete heart block or ventricular tachycardia/fibrillation. Nevertheless spontaneous recovery is usual. Carotid sinus if sensitive when compressed, in a normal heart, can result in asystole.

Obstruction to Outflow Tracts

In severe pulmonary stenosis effort syncope occurs, due to inadequate filling of left ventricle disproportionate to increased demands. In aortic stenosis, there is maximal cardiac output and adequate blood flow at rest but on exertion further increase in cardiac output does not take place and the effort syncope results. In hypertrophic obstructive cardiomyopathy, syncope results from inadequate cardiac output during or after exertion. It occurs in erect position, after prolonged standing, or after paroxysms of cough.

Cyanotic Heart Disease

In tetralogy of Fallot, the syncope is not related to decreased cardiac output but due to increased right to left shunt caused by a fall in systemic resistance or infundibular spasm resulting in arterial hypoxia.

Ischaemic Heart Disease

Syncope may be the presenting manifestation in some cases of myocardial infarction due to intense visceral pain and a fall in cardiac output.

Pericardial Tamponade

Refer to chapter titled 'Dyspnoea'.

Intracardiac Masses

Mechanical obstruction at the mitral orifice due to left atrial thrombus may account for syncope, specially when there is concomitant mitral stenosis. So is the case with myxoma.

Pulmonary Embolism

Syncope may result in pulmonary embolism due to acute circulatory obstruction and involvement of the vagal reflexes.

Arrhythmogenic Right Ventricular Dysplasia (ARVD)

This heart muscle disease is responsible for arrhythmias ranging from ventricular premature beats to monomorphic ventricular tachycardia. Recurrent unexplained syncope or palpitations are associated with ventricular tachycardia. Right ventricular failure may be encountered.

The ECG shows epsilon waves (small notches just after QRS complex). Ventricular tachycardia reflects right ventricular origin with left bundle block configuration, unlike right ventricular outflow tract (RVOT). ECHO shows right ventricular enlargement unlike in right ventricular outflow tract.

Cerebral Syncope (Neurologic)

Syncope due to cerebrovascular disease is usually caused by transient occlusion of either carotid arteries or vertebrobasilar system or vascular spasm of the cerebral arteries (TIA). Compression of the vertebral arteries by osteophytes in

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cervical spondylitis can cause syncopal attacks especially when there is abrupt movement of the neck. Diminished cranial arterial blood flow may account for syncope in migraine. Following head injury recurrent episodes of syncope occur even for months.

Metabolic Origin

Hyperventilation may be associated with syncope which usually develops gradually. Other symptoms of hyperventilation syndrome like numbness and tingling or visual disturbances or tetany may be elicited. Forced breathing results in decreased cerebral blood flow due to vasoconstriction caused by carbon dioxide wash out and consequent low carbon dioxide tension (hypocapnia). This is usually a hysterical phenomenon or may follow a valsalva's manoeuvre. These stereotyped attacks can be reproduced by forced breathing, which can be used as a test for syncope. Sometimes hypoglycaemia may be incriminated (neuroglycopenia).

Anoxic Syncope

This occurs (a) At high altitudes, (b) With extreme exertion or exercise in chronic pulmonary disease or congenital heart diseases, or (c) When flying high or during ascent of a plane from a sharp dive (aviation). The reduction of arterial oxygen saturation with consequent cerebral anoxia is the underlying mechanism for syncopal attacks. Inability to increase the cardiac output on demand and consequent decreased cerebral perfusion which occurs during exertion or exercise, due to the associated cardiac disorders, may be contributory. Anoxaemia is due to lowered O_2 in arterial blood or venous blood with increased AV difference or deficient O_2 carrying power of blood.

Psychogenic

Usually syncopal attacks are frequent. Somatic complaints will be forthcoming. The heart is normal.

Hysteria; anxiety (refer to chapters titled 'Coma' and 'Palpitations').

CLINICAL APPROACH

Usually in cases of syncope, physical examination during the attack may not be feasible. Nevertheless, careful history taking and examination may be contributory to the diagnosis of syncope. Whether neurocardiogenic or cardiogenic. It has to be differentiated from epilepsy, hypoglycaemia, vertebrobasilar insufficiency, cataplexy—drop attacks, or somatization disorder.

Methods of Enquiry

- Predisposing factors (like emotional stress, blood loss especially GI bleeding, exertion, change of posture, rotation of neck, diabetes mellitus or intake of drugs)
- Type of onset (symptoms developing, gradually over a period of few seconds or minutes (vasovagal) or sudden or abrupt without any premonition (cardiac)
- Decubitus at the onset (usually prolonged standing still in vasovagal) or standing suddenly from supine position as in postural or any position as in cardiac arrhythmias
- Duration of attack (few seconds to few minutes in vasomotor, more than few minutes in hyperventilation)
- Associated features like striking pallor (vasovagal); dyspnoea, palpitations, cyanosis and signs of underlying cardiac disorder (cardiac syncope); tingling and numbness or tetany (in hyperventilation type); other neurological features like hemiparesis, diplopia, vertigo, thick speech, and slow stertorous breathing (cerebral)
- Situational, i.e. related to micturition or paroxysms of cough.

History from an eye-witness regarding syncopal episode may be complementary for the diagnosis.

Physical Examination

General

- Look for any clubbing or cyanosis, or anaemia
- Feel all the pulses and asses rate and rhythm
- Take blood pressure in both arms and legs, recumbent and standing positions
- Simultaneous pulse and heart rates and blood pressure response to carotid sinus pressure. Carotid sinus massage for 5–10 seconds reproduces the clinical picture and the consequent ventricular pause of more than 2 seconds in diagnostic of carotid sinus hypersensitivity
- Whether turning movements of the head produce any syncope.

Systemic Examination

Cardiovascular System

- Auscultate for neck bruits (vascular obstruction)
- Examine the heart for any valvular disease or any congenital heart disease.

Respiratory System

It examines for evidence of:

- Acute cor pulmonale (pulmonary embolism), and
- Chronic cor pulmonale.

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Central Nervous System

Examine for any evidence of:

- Cerebrovascular disease especially for vertebrobasilar system;
- Features related to structures of posterior fossa (visual, auditory and vestibular); and
- Neurological diseases associated with paralysis of circulatory reflexes.
  - In clinical practice, syncope has to be differentiated from epilepsy:
    - Though, there are no tonic clonic convulsions in syncope, occasionally few clonic twitches are seen after becoming unconscious, especially in heart block or cardiac standstill. The characteristic features of epilepsy are:
      (a) The seizures can occur in any position either during the day or by night during sleep; (b) Aura present; (c) The attack is abrupt onset; (d) Personal injury invariably present after the fall due to loss of protective reflexes; (e) Tonic and clonic convulsions with rolling of the eyes upwards usually precede loss of consciousness; (f) Biting of the tongue; (g) Incontinence of urine; (h) Absence of pallor and presence of rigidity; and (i) Postictal state of confusion and drowsiness
      In akinetic seizures the subject falls to the ground without any warning and becomes unresponsive with generalised muscular hypotonia. Unawareness of the fall is the evidence for brief loss of consciousness.
  - Hysterical fainting or malingering occurs in hysterical personalities with acute anxiety state. Absence of any obvious physical signs especially changes in blood pressure, pulse rate or skin colour will be diagnostic
  - Drop attacks—The subject often falls to the ground without any warning and often injuring himself; brief loss of consciousness is indicated by unawareness of the fall patient gets up at once
    - The underlying cause is postulated to be basilar artery ischaemia.
  - Alcoholic blackouts
  - Post-concussion amnesia following brief loss of consciousness
  - Hypoglycaemia can produce brief confusional states, sympathetic overactivity, twitching of muscles, incoordination followed by loss of consciousness which may be prolonged. Diagnosis depends upon detection of blood sugars of less than 40 mg% during the attack or after prolonged fasting for 72 hours
  - Acute intracranial hypertension may produce brief loss of consciousness in colloid cysts of 3rd ventricle which obstruct the cavity intermittently or produce plateau waves
  - Cerebrovascular ischaemia (TIA).

Investigations

Haematology

Red blood cell counts; haemoglobin percentage and packed cell volume.

Biochemical Evaluation

- Normally norepinephrine increases on assuming upright position, much above the supine levels whereas in orthostatic hypotension, there is little or no rise
- Glucose tolerance test for evidence of diabetes mellitus or hypoglycaemia
- Raised LDH levels for coronary thrombosis or pulmonary embolism
- Serum sodium levels.

Other Investigations

- ***ECG:*** Syncope of cardiac origin may invariably reveal changes of either hypertrophic cardiomyopathy, ischaemic pattern or arrhythmias. Twenty four hours ECG especially during the routine activities is useful for effectively detecting cardiac arrhythmias (Holter monitoring), implantable loop recorder exercise testing
- *ECHO*
- *Cardiac MRI or CT*
- *Carotid doppler.*

Evaluation of Autonomic Function

- Standing position normally may result in a fall of systolic blood pressure by less than 20 mmHg with a modest tachycardia whereas in autonomic dysfunction the fall will be more than 20 mmHg with an unaltered pulse
- Deep breathing results in more than 10 heart beats per minute normally. The loss of this respiratory variation of heart rate (<10) indicates autonomic neuropathy due to vagal degeneration
- Valsalva manoeuvre (autonomic procedure), vide supra
- ***Pupillary reaction:*** Instilling either 0.1% adrenaline or 2.5% methacholine does not result in dilatation or constriction respectively if normal.

Pharmacological Tests

- When nitroglycerine is given in autonomic failure blood pressure, falls, without reflex tachycardia
- Noradrenaline infusion shows hyposensitivity in autonomic failure
- Pilocarpine induces sweating which may reveal the abnormal sweat pattern of hyperhidrosis on one side and anhidrosis on other side in autonomic dysfunction
- Isoproteronol and adenosine also used to induce vasovagal syncope through Bezold-Jarisch reflex.

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Reproducing Attacks of Syncope

- By applying pressure or massage over carotid sinus so as to activate the vasomotor reflexes (contraindicated if carotid bruit is present)
- ***Hyperventilation:*** Rapid and deep breathing for 2–3 minutes
- ***Head-upright tilt table test (HUT):*** It is done only if ECG is normal without any heart disease—In those who can not stand at all tilt table can be used to put the subject in upright position by tilting the table to +60° for 45 minutes or more (monitor ECG and BP) when positive response results in syncope (reproduced) due to defective vasomotor reflexes and consequent increased fall of blood pressure (normally BP falls but stabilises at a lower level), backing up neurocardiogenic syncope. If symptoms/signs appear, they are reversed by adopting flat position. If they do not develop, provocative tests done with Isoprenaline (slow infusion) or glyceryl trinitrate (inhaled) and tilt is repeated.

This test is contraindicated in coronary artery disease, mitral stenosis, left ventricular outflow obstruction.

Further Investigations

If Indicated.

- ***EEG:*** In the majority of cases, EEG shows some changes in the interval between seizures whereas in syncope it is normal. Twenty four hours EEG may prove useful. EEG reveals slow waves (2–5 s) of high voltage during loss of consciousness followed by flattening of the record transiently
- Cerebral angiography
- CT scanning or MRI brain
- Useful to differentiate other causes of brief loss of consciousness
- Intracardiac electrophysiologic studies to assess sinus node function and AV conduction
- (It may be utilised judiciously for delineating some of the syncopal episodes, which elude exact diagnosis).

Disorders like epilepsy, (Syncope is more gradual both onset and cessation; without convulsive movements, and limp instead of rigidity) or drop attacks (Though falls on ground, there is no warning, no unconsciousness no vertigo and no confusion after recovery) must be carefully distinguished. So is the case with Ménière's disease.

TREATMENT OF SYNCOPE

Episodic impaired consciousness with a feeling of momentary weakness and slipping on to the ground motionless may be really alarming. Such fainting attacks must be carefully delineated from attacks of fits. Any ordinary episode of syncope needs only little treatment since it is self limited.

However, critical approach to ascertain the aetiology of syncope inclusive of predisposing and precipitating factors must be pursued and measures must be undertaken to prevent future attacks.

Symptomatic Relief

- Place the patient in a horizontal position with the head low or legs elevated
- Loosen tight clothing and facilitate fresh air
- Keep smelling salts (aromatic spirits of ammonia) near the nostrils
- Sprinkle cold water over the face.

Such elementary measures restore consciousness usually in patients with simple (common) faint or rarely vasopressors or atropine may be required.

Specific Treatment for Specific Causes

Inadequate Venous Return and Cardiac Filling

1. ***Vasovagal syncope:*** (Simple fainting, vasodepressor syncope; neurocardiogenic syncope)
   - Avoid prolonged standing, hot stuffy rooms or extreme exertion horizontal position is better adopted
   - Leg bandages and/or abdominal binders help venous return
   - Adequate hydration and salt intake to be increased
   - Beta-blockers indicated, if Bezold-Jarisch reflex is incriminated
   - Anticholinergic are beneficial if resting bradycardia is present
   - Midodrine (Alpha-I adrenergic agonist – 5–15 mg tds) causes vasoconstriction
   - Fludrocortisone is of some benefit.
2. ***Situational:*** Post-tussive syncope
   - Severe bouts of cough can be suppressed with cough suppressants (like linctus codeine or noscapine or dextromethorphan) and antibiotics, if necessary.
3. ***Postural syncope*** (Orthostatic hypotension)
   - Tilt the head up by 6–10 inches while sleeping
   - Advise the patient to rise slowly from supine to sitting position and wait for a couple of minutes before standing
   - Increase daily salt intake—Sodium chloride 4–10 g/day (1 g of NaCl contains 17 mEq of sodium)
   - Withdraw or reduce the dosage of hypotensive drugs or other offending drugs
   - Elastic stockings and/or abdominal binders may be helpful. Volume expanders if necessary advocated
   - Nine-alpha fludrocortisone (0.1 mg twice daily) and/or indomethacin (up to 75 mg daily) or adrenergic drugs like ephedrine (up to 75 mg daily) may control attacks in the early stage midodrine (5 mg tds). Pyridostigmine

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improves orthostatic hypotension and droxydopa is beneficial for neurogenic orthostatic hypotension
- Encourage exercises like plantar dorsiflexion, tensing abdominal muscles, isometric exercise of upper limbs before and after standing
- Paroxetine (20 mg od) or escitalopram (10–20 mg/day)
- Octreotide to produce splanchnic mesenteric vasoconstriction and increase venal return
- Sympathomimetic like midodrine are worth trying in dysautonomia
- Correct the underlying cause of autonomic dysfunction if the orthostatic hypotension/syncope is chronic
- Orthostatic training
- Hypovolaemia is corrected by volume replacement with appropriate fluids
- If it is due to pulmonary hypertension bosentan is advocated
- Anaemia is to be corrected appropriately.

Inadequate Cardiac Output (Cardiac Syncope)

- ***Cardiac arrhythmias:*** Refer to chapter titled 'Palpitations'.
- ***Long QT interval syndrome:*** Offending agent is to be withdrawn. Beta-blockers are beneficial. Pacing and implantable cardioverter defibrillator (ICD) considered. Torsades de pointes (TdP) treated with magnesium IV (vide infra). If TdP is sustained, treatment is DC cardioversion. In some cases, isoproterenol infusion advocated, as it increases heart rate with consequent shortened QT interval.
- Disturbance in impulse conduction:
  - *Sick sinus syndrome*: Though life span is not affected, the treatment is indicated in the presence of disturbing symptoms. Drug therapy with atropine or isoproterenol is beneficial only in a few patients and even in these cases the side effects prohibit long-term treatment. (Isoproterenol is not desirable in myocardial ischaemia). Permanent pacemaker implantation is effective if symptomatic, in controlling troublesome symptoms Tachycardia may be treated with anti-arrhythmic drugs after implantation **(Fig. 4.4)**. (Refer to chapter titled 'Palpitations')
  - *AV block*: (second and high degree) block and third degree block
    - Drug treatment with sympathomimetic drugs like isuprel; ephedrine or cholinergic blocking drugs like atropine, has relatively little benefit though prescribed especially to prevent Stokes-Adams attacks. The survival rate can be greatly improved by long-term treatment with permanent pacemaker implantation even if asymptomatic



Fig. 4.4A: Sick sinus syndrome: Sinus arrhythmia (non-phasic) with intermittent sinus pause. (The longest R-R interval is prolonged by greater than 40% of the shortest R-R interval).



Fig. 4.4B: After pacing (permanent): Each vertical line (pacing impulse) is immediately followed by wide ventricular QRS complex.

    - For the immediate treatment of complete heart block with asystole, i.e. Stokes–Adams syndrome (if pulseless for 5 seconds (dizziness); more than 5 seconds (loss of consciousness); 10 seconds (seizures); 2 minutes (uneventful recovery unlikely); 4–6 minutes (irreversible brain damage)
    - Deliver a vigorous blow over the precordium and elevate both legs to 90 for 15 seconds. If the heart does not commence beating immediately, CPR must be started within 2 minutes, lest irreversible cerebral damage should occur
    - Drug therapy simultaneously—Asystole due to ventricular techycardia or fibrillation must be excluded before instituting the following drug regime. (Refer to chapter titled 'Palpitations')
      - Adrenaline (0.2 mL 1 in 1000 every 1–2 h SC or 3 mL 1 in 1000, i.e. 3 mg in dextrose infusion) may be effective. If necessary, intracardiac injection of adrenaline (5 mL 1 in 10,000) is given. The entry of the needle itself may stimulate the heart to beat again
      - Atropine (0.6 mg IV initially to a maximum of 3 mg) may be an useful adjunct to improve AV conduction
      - Calcium gluconate 10% 10 cc IV may be beneficial. (Avoid sodium bicarbonate line)
      - Correct acidosis (50 mL of 8.4% of $NaHCO_3$ IV), hypoxaemia and/or hypokalaemia, appropriately
      - Recovery from seizures is spontaneous, seldom requiring specific treatment
      - Temporary pacing electrode can be inserted in the mean while. External pacing (cutaneous) offers effective therapy

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- ◊ In high degree AV block (3:1 block or greater) cardiac pacing advocated. Chronic AV block is treated with permanent pacemaker. Also it is indicated in Mobitz type II block.
- ○ Tachycardias (refer to chapter titled 'Palpitations').

If the attack is prolonged CPR must be commenced.

Cardiopulmonary Resuscitation (CPR)

This is indicated in cardiopulmonary stand still (pulseless, apnoea, unconsciousness) which may occur in myocardial infarction, cardiac arrhythmia (VT/VF or CHB or PEA), metabolic disturbances (hypothermia, hypoxia, hypercapnia), drugs sensitivity, operations/anaesthesia, electrocution.

The CPR includes (a) Basic life support; (b) Advanced life support; (c) Post-resuscitation case.

Basic Life Support—A-B-C

Precordial thump over middle and lower one-third of the sternum followed by CPR for 20–30 minutes.

- ***Air way:*** Open the airway by hyperextending the neck and lift the chin and clear the mouth, i.e. the chin is moved forward in the "head tilt-chin lift method" by lifting the tongue from the back of the throat and extracting the solid or liquid debris with the index and middle fingers covered with handkerchief
- ***Breathing:*** Mouth to mouth or mouth to mask breathing (ventilation through Brooks airway or AMBU system, if available) must be done at the rate of 12–15/min (Keeping the nostrils closed), if there is no spontaneous breathing. Initially, give two full breaths, each lasting for 2 seconds and continue
- ***Circulation:*** Look for signs of circulation for not more than 10 seconds. If circulation is present, rescue breathing may be continued. If circulation is absent, chest compressions (external cardiac massage) and simultaneous rescue breathing to be done as follows. (CPR sequence).

The 100 chest compressions (by placing one hand over the other, above the xiphisternum at a rate of 100/min, with 4 cm depth of compression) at a sequence of 30 compressions followed by two breaths alternatively if alone (30:2 for 2 min), or 10 compressions to one breath, if two persons are available (pausing after every 5th compression while the other inflates the lung), i.e. the two persons should change their roles after 5 cycles of CPR or every 2 minutes are to be accomplished. Chest compressions and mouth to mouth breathing should not coincide, so as to avoid high intrapulmonary pressure and rupture.

Check for the return of the pulse. If pulse continues to be absent, sequences must be repeated for at least 30 minutes till the patient resumes breathing and heart starts beating again, with previously dilated pupils remaining constricted.

The effectiveness of the procedure is to be checked intermittently (feel for the carotid pulse every minute) without interruption of the cardiopulmonary resuscitation as far as possible (however, better not interrupt CPR for >7 s, at a time either to give shocks in ventricular tachycardia/ventricular fibrillation or to intubate or gaining IV access).

Advanced (Cardiac) Life Support

- Pulseless ventricular tachycardia or ventricular fibrillation associated with shock (shockable rhythm)
  - ○ Defibrillate 360 J if monophasic or 200 J if biphasic, and resume CPR immediately (30:2) for 2 minutes, i.e. 5 cycles. One cycle consists of 30 continuous compressions and 2 breaths–reversible causes corrected
  - ○ Check rhythm every 2 minutes. If shock still persists, repeat defibrillation and CPR to be done immediately after shock
  - ○ If unsuccessful, establish IV access and set up suction and oxygenation adrenaline (epinephrine 1 mg IV), i.e. adrenaline 1 in 10,000 (100 μg) recommended as 1 mg, i.e. 10 mL given. Repeat in 3–5 minutes or give vasopressin (40 units IV) once only
  - ○ Defibrillate and consider antiarrhythmic drugs, i.e. lidocaine 1–1.5 mg/kg IV first dose, then 0.5–0.75 mg—repeat in 3–5 minutes; maximum 3 doses or 3 mg/kg) or amiodarone (150 mg IV over 10 min diluted in 20 mL of 5% glucose)
  - ○ About 8.4% sodium bicarbonate (1 mEq/kg IV) given if needed
  - ○ Magnesium (1–2 g IV for TP)—torsades de pointes)—TdP

  ***Note*:** To sum up; defibrillate 360 J, again defibrillate, if necessary; drugs; defibrillate; drugs.
- Asystole and pulseless electrical activity (PEA) (electromechanical dissociation) with no shock (non-shockable rhythm) PEA patient is unconscious, pulse or blood pressure not detected. Contractions are not effective to pump blood, though electrical activity is present, as revealed by presence of QRS complexes or P waves even.

  It is treated as follows: Treat underlying cause CPR and adrenaline (epinephrine) administered (1 mg IV), repeat if necessary, asystole is treated with atropine (1 mg IV and repeat in 3–5 months and up to 3 doses to generate electrical rhythm) CPR can be stopped after 20 minutes, if there is no response.
- Additional therapy:
  - ○ Advanced airway, using oral and nasal airways; or endotracheal intubation
  - ○ Pacing if electrical activity dose not appear
  - ○ Dexamethasone (4 mg every 6 h) to reduce cerebral oedema if the patient is not fully conscious

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- Therapeutic hypothermia (32°–34°C)—recommended for improving cerebral function and survival). Actually prevents brain damage after cardiac standstill for 4–6 minutes.

- Correct reversible causes, like hypoxia, hypovolaemia and electrolyte disturbances.

Post-resuscitation Case

Ensure (i) Adequate airway; (ii) Continuing cardiac monitoring; (iii) Using drugs like dobutamine, norepinephrine or epinephrine and fluids to support circulation as needed; (iv) Treating precipitating cause.

Long-term management: Implantable defibrillator improves survival rate in post-resuscitation patients.

Note: (i) Consider advanced airway and capnography, i.e. continuous graph of CO_2 content of expired air obtained by an instrument if necessary. (ii) If IV access is unsuccessful, intraosseous route can be used as endotracheal route is discarded now.

Obstruction to Left Ventricular Outflow Tract

- ***Aortic stenosis:*** Aortic valvotomy or valve replacement can be considered. Balloon valvuloplasty is a palliative measure. Similarily valvotomy, for pulmonary stenosis, can be accomplished
- ***Hypertrophic obstructive cardiomyopathy:*** Beta adrenoreceptor blocker is the mainstay of medical management. Calcium antagonists like diltiazem improve diastolic filling and function. Disopyramide or amiodarone may be beneficial for arrhythmias. Antibiotic prophylaxis for endocarditis may be considered. Digitalis, diuretics, nitrates and beta receptor stimulants are better not entertained. Severe exercise must be avoided. Surgical septal myotomy—myectomy is utilised if refractory to medical treatment. Septal ablation is an alternative.
- *Acute myocardial infarction* and other causes (refer to chapter titled 'Chest Pain')
- ***Cardiac tamponade:*** Rapid collection of fluid with intrapericardial pressure >15 mmHg leading to tamponade, can be dealt with urgent pericardiocentesis or pericardiectomy, (if possible pericardiocentesis can be done in conjunction with haemodynamic measurements). Malignant pericardial effusion causing cardiac tamponade can be managed not only with pericardiocentesis but radiation therapy and local or systemic chemotherapy. Purulent effusion with tamponade needs surgical extensive drainage and effective antibiotics
- Pulmonary embolism (refer to chapter titled 'Chest Pain')
- ***Arrhythmogenic right ventricle dysplasia:*** (ARVD) Beta-blocker, amiodarone considered. Implantable cardioverter defibrillator is recommended.

Vascular Insufficiency of Outgoing Vessels to the Brain

- *Hypersensitive carotid sinus* (Reflex syncope/Carotid sinus syncope vide infra).
- ***Carotid sinus syncope:*** If the carotid sinus is hypersensitive, minimum doses of belladonna and/or ephedrine may be necessary. Strict caution must be offered not to wear tight collars and avoid sudden turning of the head. A majority of patients require implantation of pacemaker. Mixed cardioinhibitory and vasodepressor carotid sinus hypersensitivity may require either radiation therapy or surgical denervation of the sinus.
- ***Carotid artery obstruction:*** Carotid endarterectomy may be considered, if >70% stenosis present at the origin of internal carotid artery. The operative risk of a stroke or a fatality has to be weighed against syncopal episodes. Extracranial-intracranial anastomosis (superficial temporal artery to the middle cerebral trunk) may be beneficial.
- ***Subclavian steal syndrome:*** (Refer to chapter titled 'Vertigo').
- ***Cervical spondylosis:*** (Refer to chapter titled 'Vertigo').

Vascular Insufficiency of Cerebral Vessels

Cerebral syncope: Inadequacy of the brain circulation due to cerebrovascular diseases (including extracranial vascular insufficiency like vertebrobasilar or carotid artery disease) or any other predisposing intracranial pathology like recent head injury or intracranial tumours or vascular malformations leading to displacement or insufficiency of cerebral circulation must be probed and managed appropriately. In TIA, there is no brain damage and priority is to prevent further vascular events with antiplatelets. If atrial fibrillation present anticoagulants are to be considered.

Metabolic Disturbances

If the hyperventilation with hypocapnia results in cerebral vascular insufficiency, oxygen and 5% CO_2 or rebreathing his own CO_2 by breathing into a paper-bag is beneficial. Calcium intravenously is given if tetany is present.

Sometimes metabolic abnormalities like hypoglycaemia may be the underlying cause when parenteral glucose is to be administered or glucopon given (1–2 mg) 1M, Dexamethasone (4 mg every 6 h). Considered if hypoglycaemia is prolonged to control cerebral oedema.

Anoxic Syncope

- ***High altitude:*** Initial manifestations (dizziness, drowsiness, dyspnoea, syncopal episodes) may be relieved by oxygen administration. If symptoms are severe and persistent, returning back to the lower altitudes is required

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- ***Aviation:*** There is no specific treatment except the same principles being applied as on ground. However, flying at greater heights or positive acceleration in the long axis of the body are better avoided as they can precipitate cerebral anoxia
- Exertion in congenital heart disease or decompensated chronic pulmonary disease. Increased arterial oxygen unsaturation or inability to increase the cardiac output on exertion (in congenital heart diseases or decompensated chronic pulmonary disease) with consequent cerebral circulatory insufficiency and cerebral anoxia, is better prevented by avoiding unwarranted exertion.

  Although oxygen administration may be beneficial, the fact that inhalation of oxygen in cor pulmonale may lead to syncope (due to respiratory depression and consequent hypercapnia) must be borne in mind
- ***Extreme exertion:*** Excessive muscular activity during exercise makes the venous blood leaving the muscles become more unsaturated with oxygen. When this blood joins the systemic circulation, arterial oxygen saturation gets lowered. The consequent syncope due to cerebral anoxia is prevented by avoiding undue exertion.

Psychogenic

- ***For anxiety:*** Benzodiazepines (refer to chapter titled 'Fatigue') recommended
- Hysteria or somatization disorder/conversion disorder Psychological, behavioural and social interactions may be much more beneficial than medical treatment. (Refer to chapter titled 'Coma').

  Hyperventilation associated syncope may be usually a hysterical phenomenon or may follow valsalvas manoeuvre, and other clinical features of hyperventilation syndrome may be discernible.

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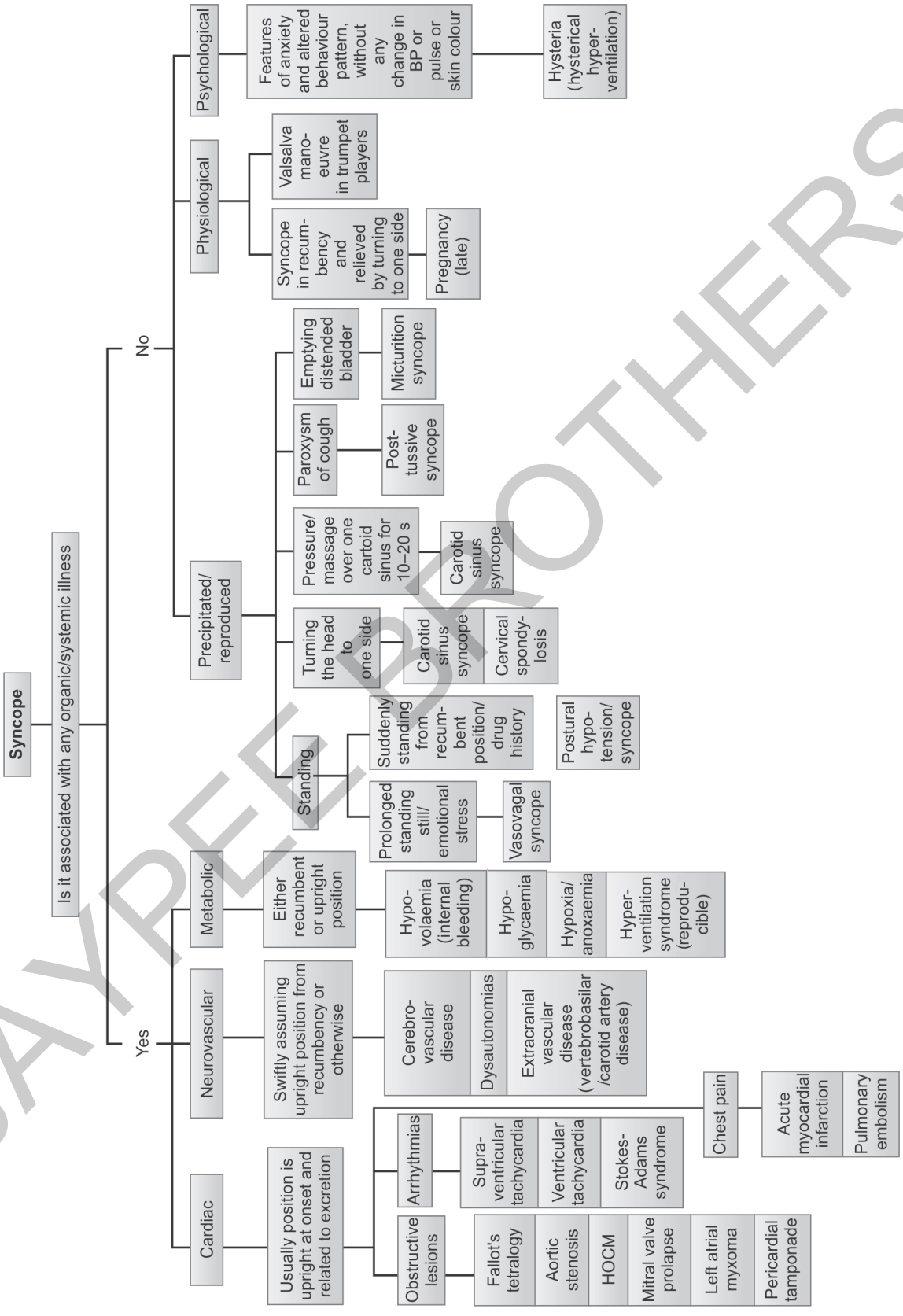

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