Diagnostic Evaluation of Syncope

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Abstract: Syncope is a complex clinical problem frequently confronts by primary care physician. At the age of 60-70 years, 5-20% adult may experience one or more syncopal episodes. 6% of hospital admission and 3% of emergency visit are due to syncopal accounts. Careful history taking and clinical examination may evaluate the cause of syncope in about 50% of cases. The evidence base methodical approach decreases hospital admission and unnecessary costly diagnostic and therapeutic procedures.

Keywords: Syncope, ECG, Holter monitoring, SA ECG, Loop recorder SNRT, SAC T, E P Study.

INTRODUCTION

Syncope is defined as sudden, transient loss of consciousness which resolves spontaneously without any intervention. It is a complex clinical problem frequently confronted by primary care physician. Patients with syncope not diagnosed by initial clinical evaluation and ECG are usually referred for 24 hours ambulatory Holter monitoring. At the age of 60-70 yrs. 5% to 20% adult may experience one or more syncopal episodes. 6% of hospital admission and 3% of emergency visit are due to syncopal account [1, 2].

CAUSES OF SYNCOPE

They include cardiac and non-cardiac causes.

Cardiac causes: Arrhythmic and Non-Arrhythmic.

Arrhythmic causes of syncope

- SA node disease: Sinus bradycardia, Sinus pause, Sinus arrest, Sinus exit block, Sick sinus syndrome (SSS), Chronotropic incompetence.
- AV node disease: Complete AV block, Bifascicular block, Mobitz II type 2nd degree AV block, Sick sinus syndrome, Ventricular tachycardia/ Ventricular fibrillation /SVT with Low BP.

Non-arrhythmic cardiac cause of syncope:

- Aortic Stenosis, Acute myocardial infarction, Pulmonary embolism, Pulmonary hypertension, Aortic dissection, Carotid sinus hypersensitivity, Left atrial myxoma.

Non-cardiac cause of syncope

- Transient Ischemic attack, Neurally mediated syncope, Vasovagal syncope, Subclavian steal, Seizure disorder, Situation syncope (cough, micturation & defecation) nad drug induced.

Drug causing syncope

- Nitrate
- Anti –hypertensive agents: Beta blocker, Calcium channel blockers, ACE inhibitors /ARBs
- Anti –arrhythmic & hypoglycemic agents
- Psycotherapeutic drugs.

PATHOPHYSIOLOGY OF CARDIAC SYNCOPE

There are four pathophysiological mechanisms:

- Transient reduction of cerebral blood flow due to CHB, LVOT and/or pulmonary embolism.
- Cardiac arrhythmia may reduce cardiac output and leads to syncope.
- Vasomotor instability may transiently decrease systemic vascular resistance and /or venous return may leads to hypotension & syncope.
- Drugs: Variety of drugs may cause orthostatic hypotension and /or arrhythmia reduces cerebral blood flow and may cause syncope.

DIAGNOSTICS TEST IN EVALUATION OF SYNCOPE

- History and physical examination.
- Non- invasive: ECG, Echocardiography, Holter monitoring, Signal average ECG (SAECG);
Loop recorder/ event recorder, Autonomic function test (Carotid sinus massage, Pharmacological test, Upright -tilt table test).
- Invasive test: SNRT (Sinus node reactivation time), SACT (Sinoatrial conducted time), EP study (Electrophysiological study).

**HISTORY AND PHYSICAL EXAMINATION**
Careful history taking and physical examination may determine the cause of syncope in about 50% of the cases[3,9,10] orthostatic hypertension, cardioinhibitory reaction and situational syncope are diagnosed only by history taking and/or by physical examination[3,9,10]. Dizziness should not be considered as syncope. Eyewitness is helpful for evaluation for pre-syncope, syncopal and post syncopal episodes[8,9]. Physical examination is important when the patient is not able to describe the event or no eyewitness is available. Following physical note should be taken.

- Carotid burit.
- Neurological deficits.
- Cardiac murmurs.
- Extra heart sound as tumor plop.
- Peripheral pluses for evidence of peripheral vascular disease.
- Dermatological examination for collagen vascular diseases or vasculitis.
- BP shout be checked in both arms and also in standing position for orthostatic hypotension.
- Funduscopic examination of eye for an embolic source.

**ECG**
- Sinus bradycardia with rate of  40 BPM in the awoken state.
- Inappropriate bradycardia- heart rate does not increase with exercise.
- Sinoatrial exit block : SA node depolarize but fail to exit. Pause is multiple of sinus PP interval.
- Sinus pause or sinus arrest : Fail to depolarize SA node in time, >3 second pause is significant. Pause is not multiple of sinus PP interval.
- 1st degree SA block: Progressive prolongation of SA node conduction and intermittent failure of impulse formation not apparent on the ECG.
- 2nd SA block: Intermittent absence of P wave resulting irregularly irregular atrial rhythm.
- 3rd degree SA block: No P wave in the ECG.
- A-Vdissociation (3rd degree A-V block), 2:1 block, second-degree A-V block (Mobitz type-II)
- Bifascicular /trifascicular block, prolong QT interval  400msec. WPW syndrome.

In asymptomatic patient level of A-V block evaluation:
- Block in A-V node: Carotid sinus massage and vagal stimulation slow heart rate. Exercise, Atropine and isoproterenol increase heart rate.

- Infranodal block: Carotid sinus massage Vagal stimulation exercise and atropine have less effect on heart rate.
- Congenital heart block: Narrow QRS complex and increase heart rate with exercise.
- Acquired heart block: Wide QRS complex and heart rate does not increase with exercise.

**ECHOCARDIOGRAPHY**
It may exclude organic heart disease especially aortic stenosis or hypertrophic Cardiomyopathy.

**HOLTER MONITORING**
Holter monitoring is one of the most commonly used test of evaluation of syncope: It may reveal significant sinus pause≥3 sec., 2:1 AV block/intermittent complete AV block/arhythmia such as AF/non sustained VT etc. for cause of syncope.

**EVALUATION OF AUTOMATIC DYSFUNCTION**
- **Carotid sinus massage:** unilateral massage of carotid sinus for 5 second may lead to sinus pause /sinus arrest for ≥ 3 second and/or fall of BP ≥ 50 mmHg in autonomic dysfunction.
- **Tilt-table test:** This is a provocation test to evaluate vasovagal syndrome. The patient is maintained in upright position on a tilt- table for defined periods to induce vasovagal episodes. West minister tilt-table protocol:
  - Test is perform in morning in the fasted state.
  - Vasoactive drugs to be stopped for 2 days before test.
  - Patient is kept in supine state for 30 minutes. BP and heart rate are recorded.
  - Table is tilted to 60° upright angle for up to 20 to 45 minutes and patient is asked to report any symptoms.
  - Test is terminated if bradycardia and /or hypotension occur in association with symptoms.
  - Three types of response may be noted.
    a) Vasopressor response : Fall of BP due to vasodilatation
    b) Cardio inhibitory: Bradycardia may be followed by hypotension.
    c) Mixed response: Bradycardia and hypotension.

- **Pharmacological Testing**
  It will differentiate between sinus node dysfunction and autonomic dysfunction.

Total autonomic blockade: Injection Atropine 0.04 mg/kg and Propranolol 0.2 mg/kg, I.V to be given. This will blocked autonomic influence on S A node resulting intrinsic heart rate intact. Intrinsic heart rate= 117.2-(0.53 x age). Intrinsic heart rate lower than predicted heart rate infers sinus node
dysfunction. If close to normal then autonomic dysfunction.

**SIGNAL-AVERAGE ECG**

A device which records 100-300 single QRS complexes which are amplified and filtered for noise and averaged to determine the presence of late potentials which are low amplitude, high frequency signal in the terminal segment of the amplified QRS. It predicts life threatening VT in future.

**LOOP RECORDER**

Loop recorders effectively couple arrhythmia and syncope. They are used in patients with recurrent frequent syncope and can monitor for weeks to months.

**External loop recorder**

The recorder can capture previous arrhythmia for 4 to 5 minutes of rhythm data during syncopal episodes if the patient activates it after regaining consciousness.

**Implantable loop recorder**

This is an implantable device similar to pacemaker. It continuously records a single lead ECG and has the capacity to store for 15 minutes segment of cardiac rhythm when activated during or after syncopal episode. The physician can later retrieve this information. It is almost 3 times as likely to provide a diagnosis by prolonged monitoring in the patients with unexplained syncope of arrhythmic origin as other standard testing such as tilt-table test, EP study and external loop recorder.

**INVASIVE TESTS**

- **SNRT** (Sinus node recovery time): Right atrium paced faster than sinus for 30 second all pacing complex abruptly stopped pacing spontaneous sinus beat recorded. If recovery time ≥ 15,000 msc is abnormal. Corrected sinus node recovery time = recovery time – sinus cycle length ≥ 550 msc. Suggestive of sinus node dysfunction.

- **SACT** (Sinoatrial node conduction time) Atrial rate is determined (A1-A1 interval i.e time between P wave). Right atrium is paced and PAC is produced and marked as A2 (without p-wave) pacing is stopped and spontaneous atrial response noted A3.

  SACT = A2-A3 interval-(A1-A1 interval)

  Or SACT = Spontaneous cycle length –return cycle length

  Normal range 50-125 msc. Prolonged SACT indicates susceptibility to exit block.

  If SACT = A1-A1 interval then automaticity is normal.

- **EP Study** (Electrophysiological Study): An invasive study. Following are the indications of EP study

  **Evaluation of bradycardia:** Sinus node dysfunction: Symptomatic patients with suspected sinus node. Disease, where the link between arrhythmia and symptoms is not established non-invasively. It can detect sinus node recovery time (SNRT) and sinoatrial conduction time (SACT). Known sinus node dysfunction to evaluate A-V node function during permanent Pacemaker implantation.

  **SITE OF BLOCK**


  In the A-V node: Normal QRS complex and A-H interval ≥ 125 msc.

  Infranodal block (His Purkinje): Wider QRS complex and H-V interval ≥ 55 msc.

  Both side block: ECG bundle branch block and both A-H and H-V interval are prolonged.

  **Evaluation of Tachyarrhythmias**

  It is useful in evaluation of reentrant tachyarrhythmia. It detects anatomic or physiologic substrate responsible for arrhythmia and defines electrical mechanism, Hemodynamic response and guides therapy.

  **TACHYCARDIA WITH NARROW QRS COMPLEX**

  Frequent symptoms, where EP characterization will determine the treatment and where drugs are ineffective/poorly tolerated/not wanted.

  **TACHYCARDIA WITH BROAD QRS COMPLEX**

  Where diagnosis of tachycardia are not determined by surface ECG, where will determine by line of treatment as well as diagnosis.

  **WOLFF-PARKINSON-WHITE SYNDROME (WPW SYNDROME)**

  - Evaluation for RF ablation.
  - Patients with unexplained syncope or resuscitated Cardiac arrest
  - Patients in whom knowledge of accessory Pathway properties may determine requirement for treatment (e.g high risk occupation).

  **EVALUATION OF UNEXPLAINED SYNCOPE**

  - Suspected or know structural heart disease in which non invasive testing fails to establish diagnosis especially elderly patients with symptomatic bradycardia.
  - Recurrent unexplained syncope after negative tilt-table testing

  **EVALUATION OF CARDIAC ARREST SURVIVORS**

  - Cardiac arrest in absence of myocardial infarction.
- Cardiac arrest ≥ 48 hours after acute MI, in absence of recurrent myocardial ischemia.
- Cardiac arrest causes by bradycardia.

**EVALUATION OF PALPITATIONS**
- Palpitation preceding syncopal episode
- Documented inappropriately rapid pulse rate in absence of adequate ECG in documentation of arrhythmia

**EP GUIDELINE OF DRUG THERAPY**
- In VT/VF cardiac arrest, sustained VT
- Guiding drug therapy in narrow QRS complex tachycardia (rarely used) as RF ablation is the treatment of choice.

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**CONCLUSION**

Syncope is a common but complex clinical symptom. Its etiology is not always easily identified. Careful history taking and meticulously related clinical examination is very important and may pick up the cause of syncope in about 50% of cases. Stepwise following of the Algorithm is important in order to identify the cause of syncope without unnecessary costly investigations.
REFERENCES


8. Stults BM, Gandolfi RJ; Diagnostic evaluation of syncope. Western Journal of Medicine, 1986; 144(2): 234.
