ANALYSIS OF DEMOGRAPHY AND CLINICAL PROFILE OF SYNCOPE IN CHILDREN

Dissertation Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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M.D. (PAEDIATRICS)

BRANCH – VII

STANLEY MEDICAL COLLEGE

The Tamilnadu Dr. M.G.R. Medical University

Chennai, India

April 2011

CERTIFICATE

This is to certify that this Dissertation titled "**ANALYSIS OF DEMOGRAPHY AND CLINICAL PROFILE OF SYNCOPE IN CHILDREN**" is the bonafide original work of **Dr. G.SANGEETHA** in partial fulfillment of the requirement for **MD (Branch VII**) Paediatrics examination of the Tamilnadu Dr.M.G.R Medical University to be held in April 2011.

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DECLARTION

I, **Dr. G. SANGEETHA**, solemlnly declare that this dissertation "**ANALYSIS OF DEMOGRAPHY AND CLINICAL PROFILE OF SYNCOPE IN CHILDREN**" is a bonafide record of work done by me in the Department of Paediatrics,Government Stanley Medical College and Hospital, Chennai under the expert guidance of **Prof.Dr. P.CHANDRA MOHAN**, Professor and Head of the Department, Paediatric Neurology, Institute of Social Paediatrics, Government Stanley Medical College and Hospital, Chennai – 600 001.

This dissertation is submitted to the **Tamilnadu, Dr.M.G.R.Medical University, Chennai** in partial fulfillment of the University regulations for the award of MD Degree (Paediatrics) **Branch - VII, Paediatrics** Examination to be held in April 2011.

Place **:** Chennai

Date **:** **(Dr. G.SANGEETHA)**

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CONTENTS

INTRODUCTION

INTRODUCTION

Syncope[1] – derived from the Greek "Synkoptein", meaning "to cut" or "to break" - is defined as a sudden loss of consciousness and postural tone, because of transient cerebral hypoperfusion, followed by spontaneous recovery.

Transient interruption of cerebral blood flow is followed by loss of consciousness within 8 to 10 seconds. Less than 30 ml blood per 100 grams of brain tissue per minute results in syncope. The critical threshold of cerebral hypoperfusion at which syncope ensues is 50% below baseline mean cerebral flow velocity.(Njemanze, 1992)

Although syncope in children is usually benign and self-limiting, physical injury may result from unprotected falls. Older children and adolescents may suffer emotional trauma from embarrassment or fear of having epilepsy, cardiac disease or sudden death.

The earliest report of breath holding spells was published in1737 by Nicholas Culpepper, who gave the description:

There is a disease…. In children from anger or grief, when the spirits are much stirred and run from the heart to the diaphragms forceably, and

hinder or stop the breath…. But when the passion ceaseth, this symptom ceaseth.

Breath holding spells were described by Abt as occurring in "neuropathic children of neuropathic parents". Bridge and colleagues stated that children susceptible to breathholding are usually of active energetic types who react vigorously to situations and that episodes were precipitated by "spoiled child reactions". Breath holding spells were felt to be a sign of a disturbed parent-child relationship by Kanner (1935).

Laxdal[2] and his associates (1969)reported that 30%of children with breath holding spells had abnormal behaviour, including temper tantrums, hyperactivity and stubbornness.

Syncopal episodes occur in late childhood or adolescents in as many as 17% of patients with breath holding spells (Lombroso and Lerman,1967)[3]. Parenting a child with breath holding spells has been associated with more maternal stress than parenting a child with a convulsive seizure disorder,and parents of BHS are at risk for developing dysfunctional parenting behaviour. Referral of parents to professionals to help with stress and parenting skills should be considered.

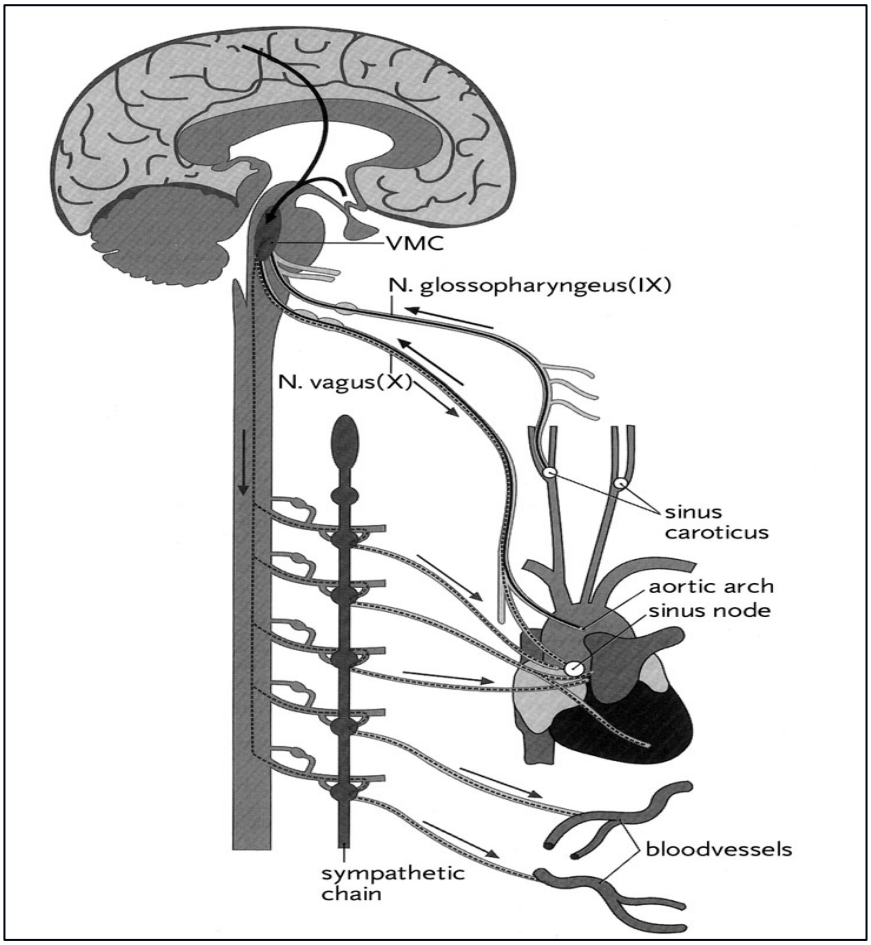
SYNCOPE

Definition:

Syncope[4] is defined as transient loss of consciousness and muscle tone that results from inadequate cerebral perfusion. Syncope follows an alteration in brain metabolism, the consequence of decreased cerebral blood flow, usually secondary to systemic hypotension.

Decreased blood flow causes loss of consciousness and the concomitant ischemia influences the higher cortical center to release their inhibiting influence on the reticular formation with in the brain stem. Neuronal discharges from the reticular formation then produce brief tonic contractions of the muscles of the face, trunk and extremities in approximately 50% of patients with syncope

AUTONOMIC NERVOUS SYSTEM



CAUSES OF SYNCOPE[5]

1. AUTONOMIC (NON CARDIAC) :

A.Orthostatic intolerance group:

Vasovagal syncope

Orthostatic syncope

Postural orthostatic tachycardia syndrome (POTS)

B.Exercise-related Syncope

C.Situational Syncope

Breath Holding Spells

Cough, micturition, defecation syncope

Deglution, diving, hair grooming syncope

Sneeze, trumpet playing, weight lifting syncope

Carotid sinus hypersensitivity

D. Excess vagal tone

2. CARDIAC:

A. Arrhythmia:

1) Tachycardia : SVT, atrial flutter & fibrillation, ventricular tachycardia (long QT syndrome)

2) Bradycardia: Sinus bradycardia, asystole, complete heart block, qpacemaker malfunction.

B. Obstructive Lesions:

Outflow obstruction: AS,PS, hypertrophic cardiomyopathy, pulmonary hypertension.

Inflow Obstruction: MS, Tamponade, constrictive pericarditis, atrial myxoma.

C. Myocardial Dysfunction:

Coronary artery anomalies, hypertrophic cardiomyopathy, MVPS, arrythmogenic RV dyplasia.

3. **NEUROPSYCHIATRIC:**

Hyperventillation

Seizures

Migrane

Tumors

Hysterical

4. **Metabolic**

Hypogycemia

Electrolyte disorder

Anorexia nervosa

Drugs/Toxins

1) AUTONOMIC (NON CARDIAC) SYNCOPE

ORTHOSTATIC INTOLERANCE

Three entities of orthostatic intolerance are

1. Vasovagal syncope
2. Orthostatic syncope
3. Postural orthostatic tachycardia syndrome

Vasovagal syncope:

(Also called simple fainting or neurocardiogenic or neutrally mediated syncope)

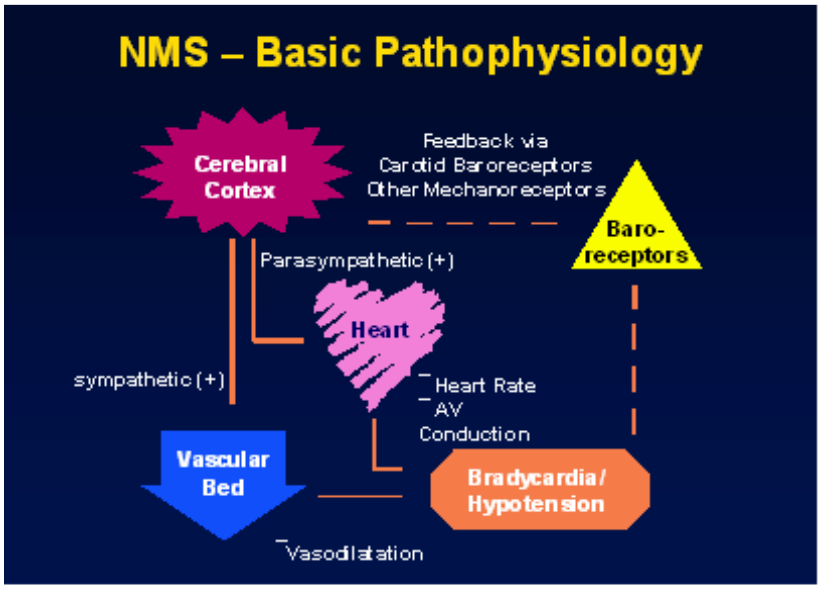
It is the most common type of syncope in otherwise healthy children and adolescents. It is characterised by a prodrome lasting a few seconds to minute. The prodrome may include dizziness, nausea, pallor, diaphoresis, palpitation, blurred vision, headache and/or hyperventilation.(manolis et al,1990;Feit [1]1996) The prodrome is followed by loss of consciousness and

muscle tone. The patient usually falls without injury, the unconsciousness does not last more than a minute and the patient gradually awakens. The syncope may occur after rising in the morning or in association with prolonged standing, anxiety or fright, pain, blood drawing or the sight of blood, fasting, hot and humid conditions, or crowded places.(Driscoll et al,1997[6])(Ganzeboom KS et al 2003[49])

Pathophysiology

In normal individuals, an erect posture without movement, shifts blood to the lower extremities and causes a decrease in venous return, thus decreasing stroke volume and blood pressure. This reduced filling of the ventricle places less stretch on the mechanoreceptors (i.e c fibers) and causes a decrease in afferent neural output to the brain stem, reflecting hypotension.(Aicardi et al.,1992). This decline in neural traffic from the mechanoreceptors and decreased arterial pressure elicit an increase in sympathetic output, resulting in an increase in heart rate and peripheral vasoconstriction to restore blood pressure to the normal range. Thus the normal responses to the assumption of an upright posture are a reduced cardiac output (by 25%), an increase in heart rate, an unchanged or slightly diminished systolic pressure and an increase in diastolic pressure to ensure coronary artery perfusion.

Cerebral blood flow decreases by approximately 6% with cerebrovascular auto regulation functioning near its maximal limit.[47]



In susceptible individual, however, a sudden decrease in venous return to the ventricle produces a large increase in the force of ventricular contraction. This causes activation of the left ventricular mechanoreceptors, which normally, respond only to a stretch. The resulting paroxysmal increase in neural traffic to the brain stem somehow mimics the conditions seen in hypertension and thereby produces a paradoxical withdrawal of sympathetic activity with subsequent peripheral vasodilatation, hypotension and bradycardia,(Grubb and Kosinski,1996)[8]. Characteristically, the reduction of blood pressure and especially the heart rate severe enough to decrease

cerebral perfusion and produce loss of consciousness. Persistence of neuro cardiogenic syncope in subjects who had cardiac transplants and therefore technically denervated hearts, as well as the finding of increased epinephrine levels during upright position and syncope,are strong evidence supporting a sympathetic withdrawal mechanism in syncope (Hannon and Knilans,1993)[9].Altered cerebral autoregulation may also be a contributory factor.Trans cranial Doppler studies have demonstrated cerebral vasoconstriction during tilt table induced syncope.

History is most important in establishing the diagnosis of vasovagal syncope. (Kapoor,2000; Lerman Sagie et al,-----1994)[3]

1. Placing the patient in a supine position until the circulatory crisis resolves may be all that is indicated. If the patient feels the prodrome of faint, he or she should be told to lie down with the feet raised above the chest. This usually aborts the syncope.
2. Increased salt and fluid intake is encouraged.
3. Elastic support hose are useful in patients with postural hypotension. (Milstein et al,1990)
4. Success in preventing syncope has been reported with medications, such as Fludrocortisone(0.1mg/kg orally), βblocker (1mg/kg/day

orally), Pseudoephedrine (60mg orally twice daily). (Grubb Kosinski,1996)[8]

Orthostatic Hypotension (Dysautonomia)

The normal response to standing is reflex arterial and venous constriction and a slight increase in heart rate. In orthostatic hypotension, the normal adrenergic vasoconstriction of the arterioles and veins in the upright position is absent or inadequate, resulting in hypotension without a reflex increase in heart rate. In contrast to the prodrome seen with vasovagal syncope, in orthostatic hypotension, patients experience only lightheadedness.(Steinberg et al,2005[10], Zhang et al 2005[11]) Orthostatic hypotension is usually related to medications or dehydration,but it can be precipitated by prolonged bed rest,prolonged standing,and conditions that decrease the circulating blood volume (eg. bleeding, dehydration). Drugs that interfere with the sympathetic vasomotor reponse (eg.calcium channel blockers, antihy- pertensives, vasodilators, phenothiazines) and diuretics may exacerbate orthostatic hypotension. Dysautonomia may also be seen during an acute infectious disease or in peripheral neuropathies such as Guillain-Barre syndrome.

In patients suspected of having orthostatic hypotension blood pressures should be measured in the supine and standing positions. The American Autonomic Society has defined orthostatic hypotension as a persistent fall in systolic / diastolic pressure of more than 20/10 mmHg within 3 minutes of assuming the upright position without moving the arms or legs, with no increase in the heart rate but without fainting. Patients with orthostatic hypotension also have a positive tilt test but do not display the autonomic nervous system signs of vasovagal syncope such as pallor,diaphoresis,and hyperventilation.(Stewart et al,2002)[12]

Elastic stockings, a high salt diet, sympathomimetic amines and corticosteroids have been used. The patient should be told to move to an upright position slowly[13].

Postural Orthostatic Tachycardia Syndrome[14](POTS)

It is a form of autonomic neuropathy that predominantly affects the lower extremities. The findings may be related to chronic fatigue syndrome, such as chronic fatigue, exercise intolerance, palpitations, light headedness, nausea and recurrent near syncope and sometimes syncope.

For the diagnosis of POTS, heart rate and blood pressure are measured in the supine, sitting and standing positions. POTS is defined as the

development of orthostatic symptoms that are associated with atleast a 30 beats/min increase in heart rate (or a HR >120 beats/min) that occurs within the first 10 minutes of standing.

The patient is advised to avoid extreme heat and dehydration and to increase salt and fluid intake. Fluducortisone, Midodrine or Venlaflaxine are useful in many patients.

Exercise related syncope *:*

It is due to a combination of venous pooling in vasodilated leg muscles, inadequate hydration and high ambient temperature.(Pratt et al,1989)[5]

Cough syncope :

It follows paroxysmal coughing in asthmatic children. The patients face becomes plethoric and cyanotic and the child perspires, become agitated and is frightened. Loss of consciousness is associated with muscle contractions lasting for several seconds. Urinary incontinence is frequent. Consciousness is regained within few minutes(Bleasel et al,1995)[15]Paroxysmal coughing produces a marked increase in intraplueral pressure with a reduced venous return and reduced cardiac output, resulting in altered cerebral blood flow and loss of consciousness. Treatment is aimed at preventing

bronchoconstriction with aggressive asthma treatment plans. (Feit et al,1996)

BREATH HOLDING SPELLS :

Breath holding spells with the loss of consciousness occur in almost 5% of infants. The cause is a disturbance in central autonomic regulation probably transmitted by autosomal dominant inheritance with incomplete penetrance.

The spells are involuntary responses to adverse stimuli.

Cyanotic Syncope :

It is three times more common than pallid spells. The usual provoking stimulus for cyanotic spells is anger, frustration or fear. Cyanosis develops rapidly, followed quickly by limpness and loss of consciousness. Most spells, especially the ones referred for neurological evaluation, are longer and are associated with the tonic posturing of the body and trembling movements of the hands and arms. The eyes may roll upward. These movements are probably a brain stem release phenomenon. Concurrent EEG shows flattening of the record, not epileptiform activity. The typical

sequence of cyanosis, apnoea and loss of consciousness is crucial for diagnosis. Between the attacks, the EEG is normal.

Pathophysiology of Cyanotic spells:

During the spell, spasm of the glottis and respiratory muscles with increased intrathoracic pressure occurs during expiration. Increased intrathoracic pressure reduces cardiac output, causing a decrease in cerebral perfusion. Violent crying could lead to hypocapnia, which would also impair cerebral circulation. (Lombrosco and Lerman 1967)[16]

Prolonged expiration usually triggered by noxious stimuli will cause the arterial oxygen saturation to fall below 20mmHg within 20 seconds. Loss of consciousness will occur after 30 seconds.(South all and associates 1985)

Pallid syncope :

The provocation of pallid syncope is usually a sudden, unexpected painful event, such as a bump on the head. The child rarely cries but instead becomes white and limp and loses consciousness. After the initial limpness, the body may stiffen and tonic movements of the arms may occur(Bridge et

al,1943)[17] As in cyanotic syncope, these movements represent a brainstem release phenomenon, not seizure activity. (Laxdal et al,1969)[2]

Pallid syncope is the result of reflex asystole. Bradycardia with periods of asystole of > 2 seconds may be recorded.(Lombrosco and Lerman, 1967, Stephenson,1978)[18]

Interictal EEG is normal. Pressure on the eyeballs to initiate a vagal refex provokes an attack.

Treatment :

The most important aspect of the treatment is to reassure the family about the benign nature of the spells. It is important to emphasis that the episodes do not lead to mental retardation. Parents should be instructed to place the child in a lateral recumbent position so as not to prolong the period of cerebral anoxia. Although anger and frustration are often precipitants for BHS, parents should be encouraged not to alter customary disciplines for fear of triggering an episode.

Treatment with iron therapy should be initiated in any child who has iron deficiency anemia and should be considered in any child with breath holding spells because children without anemia may have improvement in their BHS.(Holowach and Thurston 1963, Graft et al 2000)[19]The convulsive

movements seen during BHS are reflex anoxic seizures (Emery, 1990) , which are not epileptic and donot require antiepileptic treatment (Stephenson 1990) .Atropine(0.01mg/kg two to three times daily)is effective for pallid BHS,but its use is rarely warranted (Stephenson 1980)[20]

Piracetam,(40mg/kg/day) which has a chemical structure similar to gamma amino butyric acid can be used. (Donma 1998).Levitracetam is similar and equally effective.

2) CARDIOVASCULAR MEDIATED SYNCOPE :

It has a higher mortality and a higher incidence of sudden death. Causes include aortic stenosis, hypertrophic obstructive cardiomyopathy, primary pulmonary hypertension, sick sinus syndrome, long QT syndrome, Supraventricular tachycardia, ventricular tachycardia and heart block.(Ritter et al 2000)[21]

Peripheral vasodilation secondary to exercise is not accompanied by an adequate increase in cardiac output because of obstructive lesions, which results in diminished perfusion to the brain.(Sarasin et al2002)[22]

Stimulation of the upper respiratory tract, GI tract, eyes or skeletal muscles may induce a vagal response and therefore cause bradycardia,

decreased peripheral resistance, reduced blood pressure and also cause syncope

Obstructive Lesions:

Patients with severe obstructive lesions such as aortic stenosis, pulmonic stenosis, HOCM, as well as those with hypertension, may have syncope. Peripheral vasodilation secondary to exercise is not accompanied by an adequate increase in cardiac output because of obstructive lesion, which results in diminished perfusion to the brain. Patient may also complain of chest pain, dyspnoea and palpitation.

It can be diagnosed by careful physical examination, ECG, X-Ray studies and ECHO. Surgery is indicated for most of these conditions, with the exception of irreversible forms of pulmonary hypertension.

Myocardial Dysfunction:

Myocardial ischemia secondary to congenital anomalies of the coronary arteries or acquired disease of the coronary artery (such as Kawasaki disease) may cause syncope. Patients with dilated cardiomyopathy may have episodes of syncope associated with self-terminating episodes of ventricular tachycardia, which can lead to cardiac arrest. Syncope is a major

risk factor for subsequent sudden cardiac death in HOCM, particularly if it is repetitive and occurs with exertion.

Arrhythmias:

Either extreme tachycardia or bradycardia can decrease cardiac output and lower the cerebral blood flow below the critical level, causing syncope.Commonly encountered rhythm disturbances include supraventricular tachycardia, ventricular tachycardia, sick sinus syndrome and complete heart block. Simple bradycardia is usually well tolerated in children, but the combination of tachycardia followed by bradycardia is more likely to produce syncope.

No identifiable Structural Defects:

Syncope from arrhythmias in children with structurally normal hearts may be seen in

1. Long QT syndrome :

It is charecterised by syncope caused by ventricular arrhythmias, prolongation of the QT interval on the ECG, and occasionally a family history of sudden death. Congenital deafness is also a component of Jervell and Lange Nielsen syndrome but not that of Romano-ward syndrome.

2. Wolff-Parkinson-White (WPW) pre-excitation may cause SVT
3. RV dysplasia (RV cardiomyopathy) is associated with repeated episodes of ventricular tachycardia.
4. Brugada Syndrome: May cause sudden death by ventricular arrhythmias. ECG typically shows RBBB with J point elevation and concave ST elevation in V1.

Structural Heart defects:

Ebstein's anomaly, mitral stenosis, mitral regurgitation, mitral valve prolapse and congenitally corrected transposition of great arteries may cause arrhythmias.

3) NEURO PSYCHIATRIC

Hyperventilation Syncope:

This type of syncope occurs due to cerebral vasoconstriction induced by hypocapnia. It is commonly related to anxiety and by their reproducibility during voluntary hyperventilation. During this attacks, tachypnoea, anxiety, breathlessness, lightheadedness, paresthesias are predominant(Brignol et al,2006)[23,24]. Diagnosis requires a high index of suspicion and reproduction of the symptoms during voluntary hyperventilation. Emperic treatment

consisting of breathholding, slow breathing or breathing into a paper bag may be worthwhile.(Herman et al,1981) [25] (Evans RW et al 1995)[26]

BASILAR MIGRAINE

The term basilar (artery) migraine characterizes recurrent attacks of brainstem or cerebellar dysfunction that occur as symptoms of a migraine attack. Girls are affected more often than boys. The peak incidence is during adolescence, but attacks may occur at any age. Infant onset cases are more likely to present as benign paroxysmal vertigo.(Chen et al 2007)[27]

Clinical Features :

Gait ataxia occurs in approximately 50% of patients. Other symptoms include visual loss , vertigo, tinnitus, alternating hemiparesis and paresthesia of the fingers, toes, and corners of mouth. An abrupt loss of consciousness may occur, usually lasting for only a few minutes. Cardiac arrhythmia and brainstem stroke are rare life threatening complications. A severe, throbbing, occipital headache usually follows the neurological disturbances. Nausea and vomiting occur in less than one third of cases. Children may have repeated basilar migraine attacks, but with time, the episodes evolve into a pattern of classic migraine. Even during attacks of

classic migraine, the patient may continue to complain of vertigo and even ataxia.

Diagnosis

EEG distinguishes basilar migraine from benign occipital epilepsy. The EEG shows occipital intermittent delta activity during and just after an attack in basilar migraine and occipital discharges in epilepsy. (Sneddon JF,1993)[28]

Management

Treatment of basilar artery migraine is the same as for other forms of migraine. Frequent attacks require a prophylactic agent.

EVALUATION OF A CHILD WITH SYNCOPE

The goal of evaluation of a patient with syncope is to identify high risk patients with underlying heart disease, which may include ECG abnormalities, cardiomyopathy, or structural heart disease. ECG should be inspected for heart rate, arrhythmias, WPW pre excitation, heart block and long QT interval as well as abnormalities suggestive of cardiomyopathies and myocarditis.(Rea R et al 1993)[29], (Strickberger SA et al)[48], (Louis simonet et al 2001)[40]

Holter monitoring usually records ECG for upto 24 hours. External loop recorders with extended monitoring (usually for a month) may increase the diagnostic yield. The implantable loop recorder (implanted in the left pectoral region) is a device that can be used to record ECGs for a period much longer than one month.(Fogoros RN et al,1993)[30], (Pratt JL et al 1989)

Cardiac catheterization and electrophysiological testing may be indicated in some equivocal cases.(Linzer M,Yang et al 1997)[31,32]

Echocardiographic studies to identify structural abnormalities.

Serum glucose and electrolytes are of limited value because the patients are seen hours or days after the episode. (Manolis as et al,1990)[33]

- Hb level, peripheral smear study- to assess the anemia (Colina et al,1995)[34],(Lombrosco et al 1967)

- Blood sugar and electrolytes has to be checked.(About et al,1993)[35]
- EEG to look for any seizure activity(Benditt DG,1996)[36]
- CT has to be taken to rule out any structural anomalies (Farewell OJ 2004)[37]
- X Ray cervical spine to rule out any cervical vertebral anomalies. (Soteriades EJ et al 2002)[38]

Head up tilt table test:

Patients lie supine on an electric tilt table and have an i.v line established.ECG monitoring and automated BP measurements are performed. Patients are tilted to 60 to 80 degree headup position for a period of 30 minutes. These patients remain upright with recording of BP every one or two minutes with HR monitoring.Positive response commonly include light headedness, dizziness, nausea,visual changes and frank syncope(Dijane et al., 1996)[39]

Several distinct abnormal patterns have been identified following head-up tilt table test

1. Vasovagal:an abrupt decrease in BP, usually with bradycardia.
2. Dysautonomia:a gradual decrease in BP leading to syncope.
3. POTS : An excessive HR increase to maintain an adequate BP to prevent syncope.

More controlled studies are needed to use this test in children.(Victor 1996)

CLINICAL PROFILE OF SYNCOPE IN CHILDREN

VARIOUS STUDIES IN THE REVIEW OF LITERATURE

Syncope represents a common & challenging complex of symptoms for the presenting physician. The syncope spectrum differs distinctly between children & adults and also the causes of syncope in adults are rarely seen in children. Since many years various studies have been undertaken to analyse syncope in children, which includes large population based prospective study,studies based on etiology,studies regarding management and studies about associated risk factors .

Lerman –sagie(1994) studied about the prospective evaluation of paediatric patients with syncope which showed that increased prevalence of syncope as the age advances. 58% were in the age group of > 8 yrs. Driscoll et al (1997) studied about various types of syncope in children. Study population included 240 children. He concluded that the most common age group affected was those between 9 – 12 yrs. (54%)

Abboud et al (1993) conducted a study which showed the result as increased female predilection in neurocardiogenic syncope (54%). Linzer et al (1997) in his study of clinical profile & diagnostic approach in syncope , with a study population of 320 patients, showed females (52.7%) were slightly in higher range compared to males (47.3%).

Steinberg et al (2005) studied the various etiologies of syncope with a study population of 220 children. Vasovagal syncope was the most common type followed by breath holding spells, cardiac syncope & others. Snedden et al (1993) & sarasin et al (2001) analysed the precipitating factors in vasovagal syncope and found prolonged standing (52 %) and staying in crowded places (30.6%) as the common causes followed by others like sight of blood and fasting. Sarasin et al also ,from his study , concluded that the occurrence of syncope was more common in the morning.

Many number of studies were conducted to analyse Breath holding spells. OESIL study (2000) with study group of 320 and SEEDS study (2004) with study group of 210 concluded cyanotic BHS to be more common (2/3) than pallid BHS (1/3). Lerman et al (1994) & graf et al (2000) studied about the prevalence of anemia in BHS. Holoworth & Thurston et al (1963) found that 102 children with BHS had a Hb level less than 8 g% compared with 7% and 2.6% in the two control groups.

There are reports of children with BHS and concomitant anemia who had resolution of their spells with the correction of anemia. Bhatia et al, colina & Abelson (1995) , Dinario (1992) , Tom & Rash (1997), described a child with pallid BHS associated with transient erythroblastopenia of childhood. The spells resolved with iron therapy before the correction of anemia. Daoud & colleagues (1997) studied the effect of iron therapy. 51.5% of the children treated with ferrous sulphate had complete remission of spells & an additional 36.4% experienced greater than 50% reduction. No children in the placebo group had total remission of spells.

Laxdal et al (1969) , Lombroso & Lerman (1967) evaluated the family pedigree in syncope and found that 27% of 114 proband parents & 21% of proband siblings had a history of BHS, suggesting a genetic influence.

AIM & OBJECTIVES

AIM OF THE STUDY

To study the epidemiology, etiological factor and Clinical profile of syncope in children.

MATERIALS & METHODS

MATERIALS AND METHODS

STUDY PLACE:

Institute of social paediatrics,

Stanley medical college and hospital,

Chennai.

STUDY DESIGN:

Descriptive and cross sectional study

STUDY PERIOD:

September 2009 to august 2010

INCLUSION CRITERIA:

- Children in the age group of 6 monthsto 12years presenting with syncope for the first time in the OPD.

EXCLUSION CRITERIA:

- Less than 6months of age.
- Known developmental delay
- Mental retardation
- Seizure disorder children.
- Children who were already evaluated and on treatment.

METHODOLOGY:

- All the children fulfilling inclusion & exclusion criteria of about 120 cases attending the paediatric outpatient Department were selected for our study.
- Informed and written consent has obtained.
- Lower limit of 6 months was selected because it was expected that breath holding spells were peak after the age of 6months.
- Demographic data including the educational status of the parents, occupation of the father and income of the father were recorded. These were used as proxy Measures to assess the socio economic status of the Children.
- Age of onset, duration of syncope, patient position, precipitating factors, associated symptoms,family history were recorded.

- The systemic and neurological examination results and the results of laboratory tests including blood counts, peripheral smear study, blood sugar, Electrolytes, ECG, X-ray chest, Echo, EEG, CT brain were taken for all the children.
- Distribution of anemia and nutrional status in various syncope were studied.
- All the details were analysed using appropriate statistical methods.

STATISTICAL ANALSIS:

- The various etiologies of syncope were expressed in Percentage.
- Socioeconomic & demographic variables, distribution of Anemia, nutri- tional status in various syncope were given in Percentage, the association was studied by pearson chi Square test.
- P value<0.05 was taken as significant.

OBSERVATION & RESULTS

OBSERVATION AND RESULTS

In this study conducted between September 2009 and August 2010, about 120 cases were included. All the children were divided into 4 groups based on their age. Also sex of the child, educational status of the parents and socioeconomic status of the family were recorded.

All the datas were tabulated and also graphically represented. All datas were analysed for statistical significance using P value.

Syncope was also divided into groups based on etiology. The two major type namely Vasovagal syncope and breath holding syncope were analysed separately. The association of anemia and the influence of nutritional status were studied too.

Children in this study were in the age group of 6months to 12 years. Mean age was 9.5yrs. They were divided into 4 groups as follows

Group 1 : 6 months – 3 years

Group 2 : 3 years – 6 years

Group 3 : 6 years – 9 years

Group 4 : 9 years – 12 years

During this 1 year period, OP census was 2,10,078.

Peadiatric neurology census was 41,787.

Prevalence of syncope was 57.12/lakhs/year

Table - I

DEMOGRAPHIC FACTORS IN SYNCOPE

Demographics(overall)		**No of children(n)**	**%**
Age	6m-3yr	36	30
	3yr-6yr	8	6.7
	6yr-9yr	19	15.8
	9yr-12yr	57	47.5
Sex	Male	47	39.2
	Female	73	60.8
Educational status of the parents	Literate	79	66
	Illiterate	41	34
Socio economic status	Class IV	53	44.2
	Class V	67	55.8

The above table shows the overall demographic profile of the study.

Syncope in children was common in the age group of 9 to 12 years – group 4 (47.5%), which was mainly of vasovagal type, followed by 6 months to 3 years (group 1) which was due to Breath holding spells. About 60 % of children were female. Majority of the parents (66%) were literate and all the parents belonged to low socioeconomic status- Class IV (44.2%) ; Class V (55.8%).

Table - II

THE VARIOUS ETIOLOGIES FOR SYNCOPE IN CHILDREN

IN THIS STUDY IS GIVEN BELOW

CAUSES OF SYNCOPE IN CHILDREN			
S.No	**Types Of Syncope**	**NO.(n)**	**%**
1	Vasovagal	48	40
2	Breath Holding Spells	40	33.3
3	Cardiac	8	6.7
4	Orthostatic Hypotension	4	3
5	Cough	2	1.7
6	Hyperventilation	2	1.7
7	Migraine	1	0.8
8	Others	15	12.5
	Total	120	100

The table reveals that Vasovagal syncope was the most common type where 48 children out of 120 belonged to this group accounting for 40%.

Breath holding spells was the second most common cause accounting for 33.3%, followed by other types.

Figure1

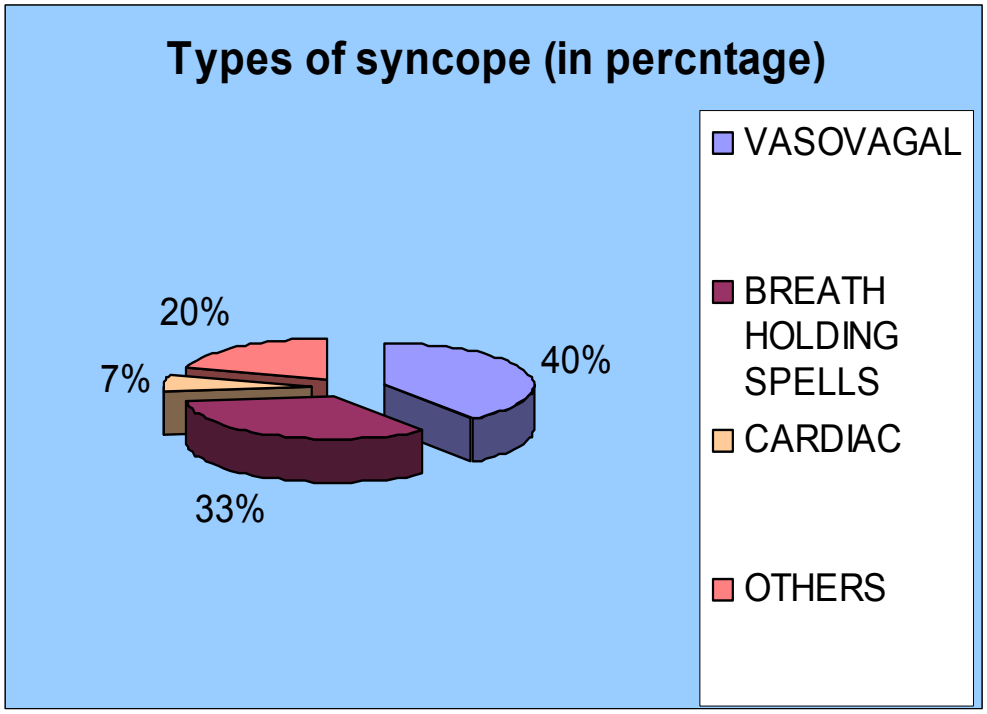


Figure2

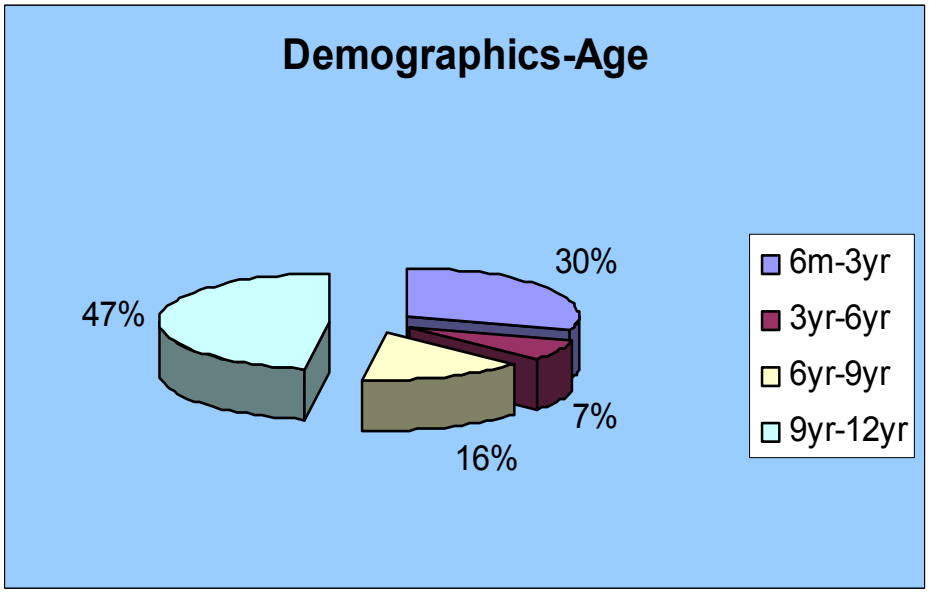


Table - III

ANALYSIS OF OVERALL DEMOGRAPHIC PROFILE IN SYNCOPE

AGEWISE DISTRIUTION OF SYNCOPE

Age	Vasovagal Syncope		Breath Holding Spells		Others		Total		Chi Square	P Value
	N	%	N	%	N	%	N	%		
< 6 yrs	2	4.7	40	93	1	2.3	43	35.8	0.000	<0.001
>6yrs	46	59.7	0	0	31	40.3	77	64.2		

Syncope was common in children more than 6 years of age comprising of 64.2% (n=77). The difference between these two groups was found to be statistically significant; P value was less than 0.001.

Table - IV

DISTRIBUTION OF SYNCOPE BY GENDER

Gender	Vasovagal syncope		Breath holding spells		Others		Total		Chi square	P value
	N	%	N	%	N	%	N	%		
Male	16	34	21	44.7	10	21.3	47	39.17	4.512	0.105
Female	32	43.8	19	26	22	30.1	73	60.83		

Though the above table shows that the majority were female children (60.83%), the difference was not statistically significant; p value being 0.105.

Figure3

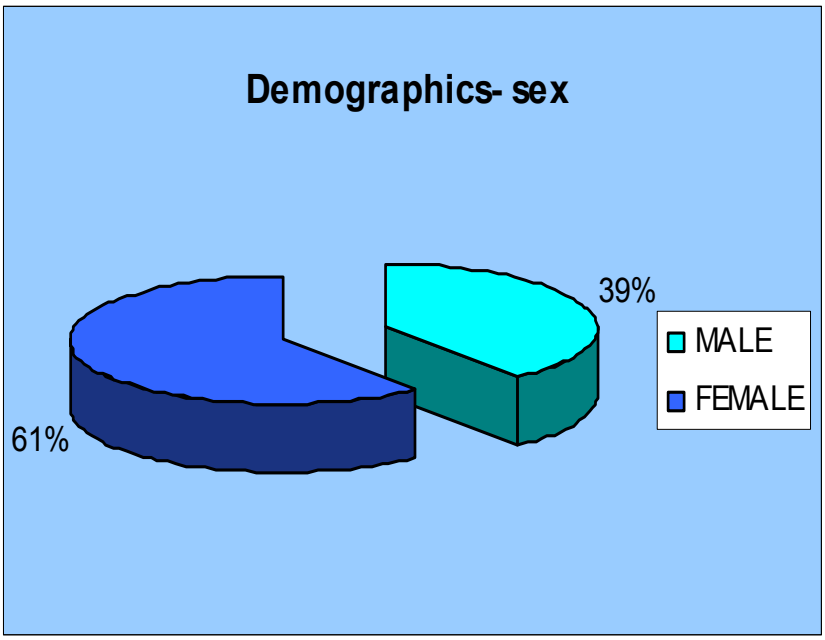


Figure4

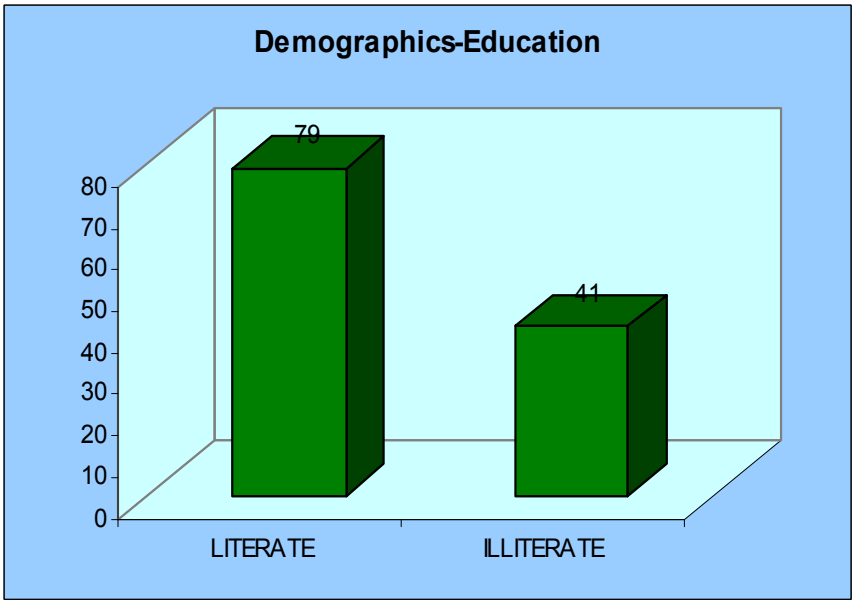


Table - V

DISTRIBUTION OF SYNCOPE BASED ON

EDUCATIONAL STATUS OF THE PARENTS

Educational Status of the parents	**Vasovagal syncope**		**Breath holding spells**		**Others**		**Total**		**Chi square**	**P value**
	N	%	N	%	N	%	N	%		
Literate	35	43.8	28	35	16	21.3	79	66	0.158	>0.05
Illiterate	13	32.5	12	30	16	37.5	41	34		

Statistical significance was not there between the two groups even when 66% of the parents were literate. Table reveals that p value was more than 0.05.

Table – VI

DISTRIBUTION OF SYNCOPE IN DIFFERENT

SOCIO - ECONOMIC STATUS GROUPS

Socio economic status	Vasovagal syncope		Breath holding spells		Others		Total		Chi square	P value
	N	%	N	%	N	%	N	%		
Class IV	22	41.5	11	20.8	20	37.7	53	44.17	0.012	<0.05
Class V	26	38.8	29	43.3	12	17.9	67	55.83		

All the patients were from low socioeconomic status, of which statistically significant group ($p< 0.05$) belongs to class V (55.83%).

Figure5

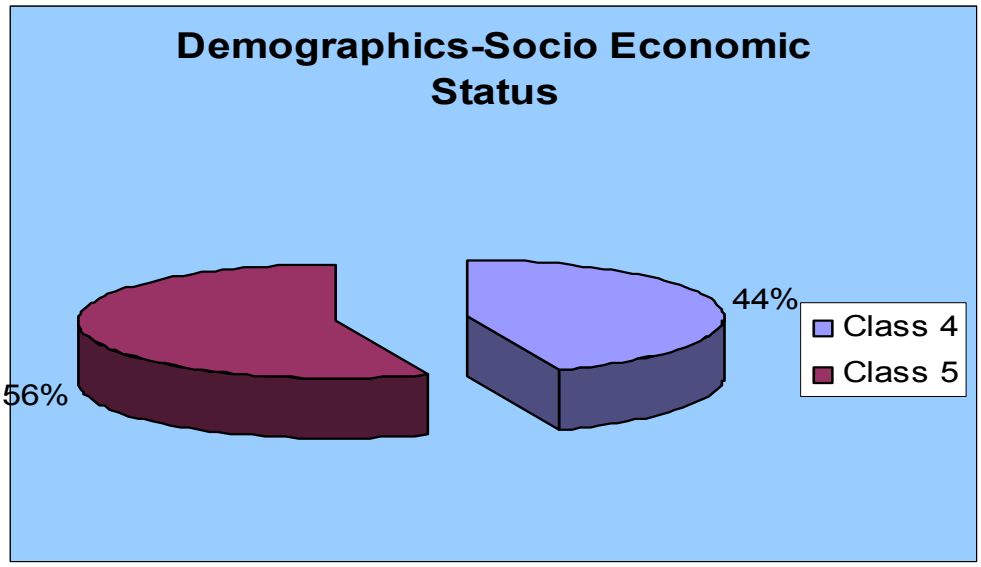


Figure6

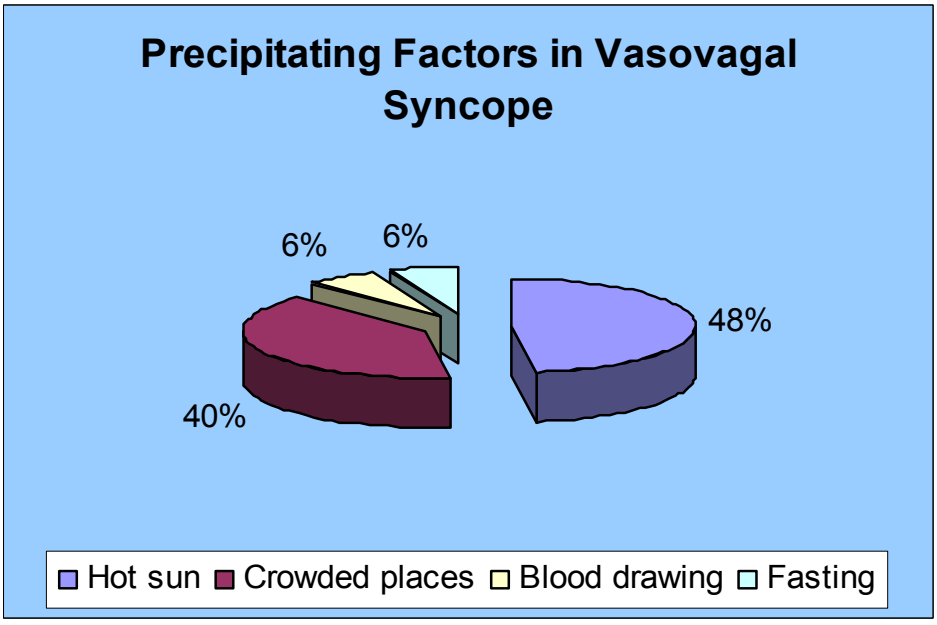


Table - VII

VASOVAGAL SYNCOPE

DEMOGRAPHIC FACTORS IN VASOVAGAL SYNCOPE			
Demographics		No. Of children	%
Age	6m-3yr	NIL	-
	3m-6yr	2	4.1
	6yr-9yr	9	18.8
	9yr-12yr	37	77.1
	Total	48	100
Sex	Male	16	33.3
	Female	32	66.7
	Total	48	100
Parents Literacy	Literate	35	72.9
	Illiterate	13	27.1
	Total	48	100
Socio economic Status	U.lower	22	45.8
	L.lower	26	54.2
	Total	48	100

The demographic profile in Vasovagal syncope is the same as compared to the syncope overall. Majority of children belonged to the age group of 9 to 12 years accounting for 77.1%, common in females (66.7%). The literacy rate of the parents was 72.9% and 54.2% belonged to class V Socioeconomic status.

Table - VIII

PRECIPITATING FACTORS IN VASOVAGAL SYNCOPE			
FACTORS		No.(n)	%
Hot sun		23	47.9
Crowded places	Temple(3)	19	39.6
	Bus(11)		
	Festival (5)		
Blood drawing / Sight of blood		3	6.25
Fasting		3	6.25
Total		48	100

Standing in the hot sun is the most common precipitating factor for Vasovagal syncope (47.9%) followed by crowded places (39.6%) like festival, temples, buses etc. Fasting and sight of blood formed the rest.

Figure7

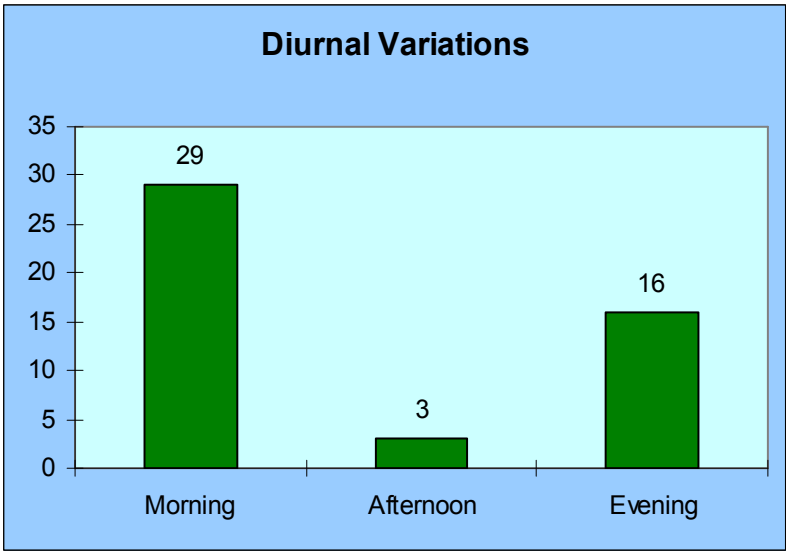


Figure8

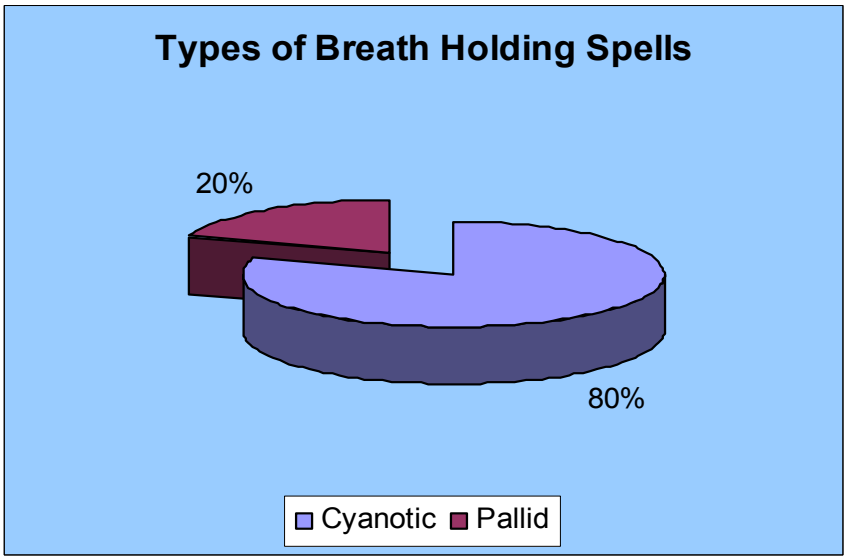


Table - IX

EFFECT OF STANDING IN HOT SUN		
Duration of standing in Hot Sun causing Syncope	**No. (n)**	**%**
Up to 5 min	2	8.7
5 to 10 min	17	73.9
> 10 min	4	17.4
TOTAL	23	100

Of the children who had vasovagal syncope due to standing in hot sun, about 74% were affected when the duration of exposure to sun was between 5 to 10 minutes.

Table - X

DIURNAL VARIATIONS IN VASOVAGAL SYNCOPE		
Timing	**No.(n)**	**%**
Morning	29	60.41
Afternoon	3	6.25
Evening	16	33.34
Total	48	100

Majority suffered of vasovagal syncope in the morning (60.41%), evening time (33.3%) being the next common.

Table - XI

EFFECT OF FASTING IN SYNCOPE		
Time of fasting	**NO**	**%**
Overnight	NIL	-
Morning	3	6.25
No fasting	45	93.75
Total	48	100

In our study fasting had no major influence in the occurrence of syncope as nearly 94% of the children were not fasting during the attack.

Figure 9

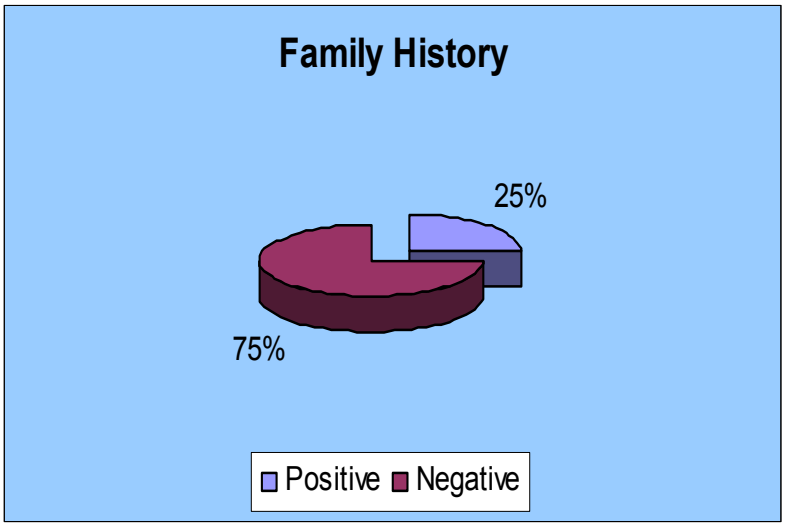


Figure10

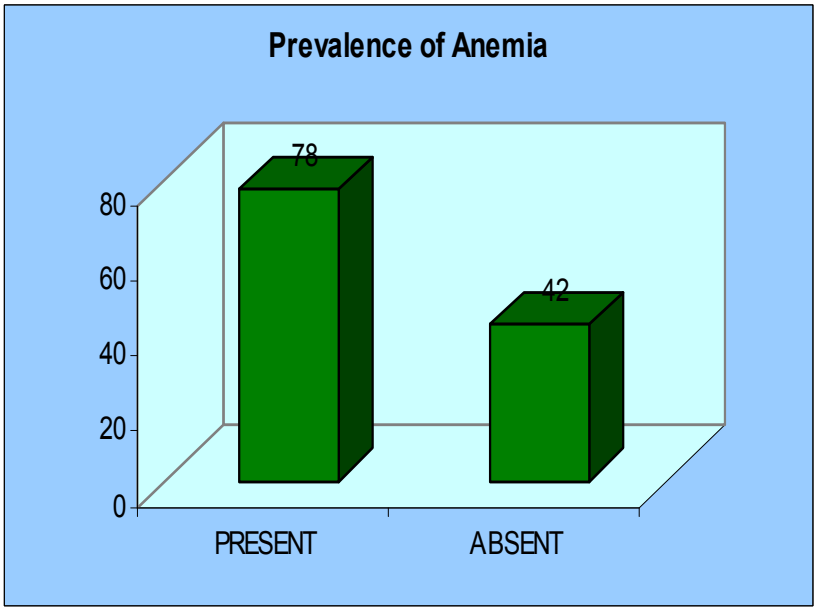


Table - XII
BREATH HOLDING SPELLS

DEMOGRAPHIC FACTORS IN BREATH HOLDING SPELLS			
Demographics		No. Of children	%
Age	6m-3yr	36	90
	3yr-6yr	4	10
	6yr-9yr	NIL	-
	9Yr-12yr	NIL	
	Total	40	100
Sex	Male	21	52.5
	Female	19	47.5
	Total	40	100
Literacy	Literate	28	70
	Illiterate	12	30
	Total	40	100
Socio economic status	U.lower	11	27.5
	L.lower	29	72.5
	Total	40	100

In contrast to the overall demographic profile, in breath holding spells, 90% of the children (n=36) belonged to the age group of 6 months to 3 years (group 1) and majority were male (52.5%).

Only 4 children were under the age group of 3 years to 6 years, this accounting for 10%. As the age increases, number of children affected by BHS was decreased. No children came under the age group of 6 to 12 years.

Figure11

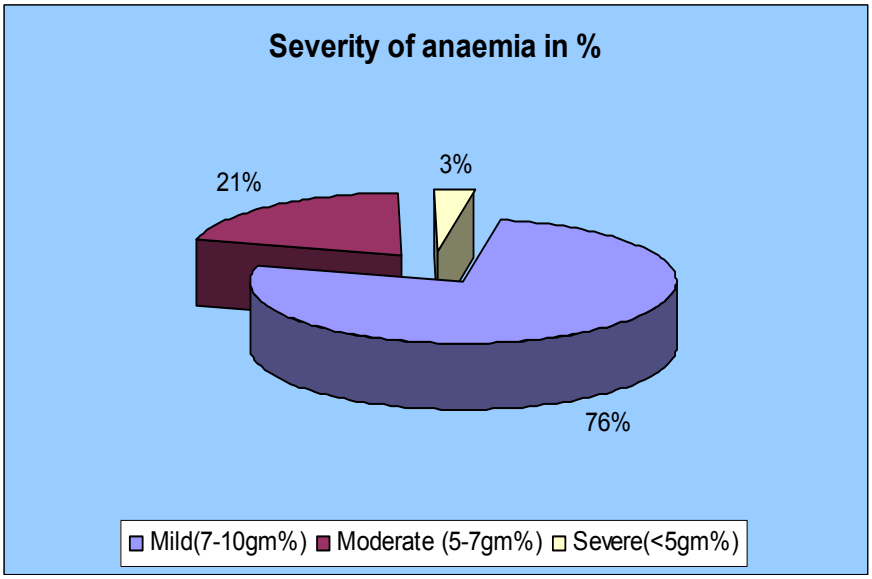


Figure 12

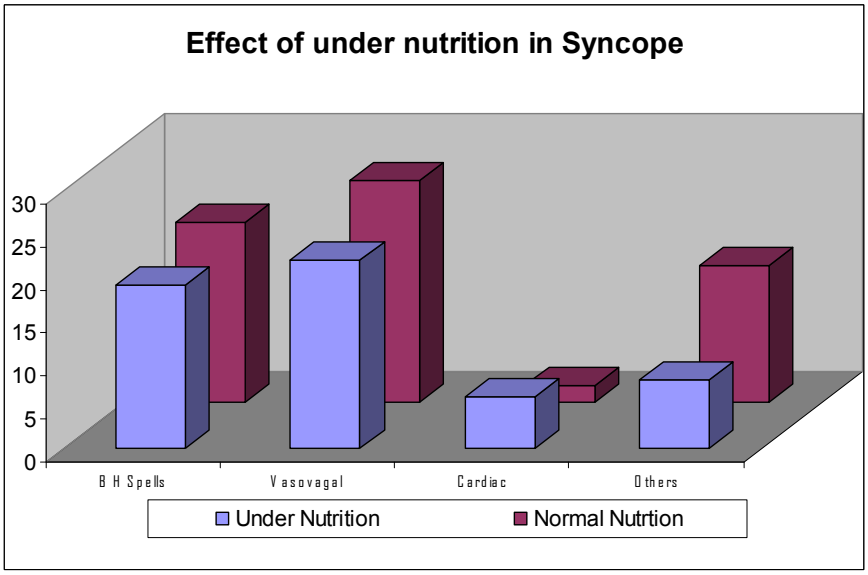


Table - XIII

TYPES OF BREATH HOLDING SPELLS		
Types	**No.(n)**	**%**
Cyanotic	32	80
Pallid	8	20
Total	40	100

Cyanotic BHS were more common compared to pallid BHS. 32 patients (80%) fall under cyanotic BHS whereas 8 (20%) patients were under the group of pallid.

Table - XIV

ASSOCIATION OF FAMILY HISTORY		
Family h/o in breath holding spells	**No.(n)**	**%**
Positive	10	25
Negative	30	75
Total	40	100

Only 10 (25%) patients had family history of breath holding spells. 30 (75%) patients had no family history

Table - XV

PREVALENCE OF ANEMIA IN SYNCOPE		
Anaemia in syncope	**No.(n)**	**%**
Present	78	65
Absent	42	35
Total	120	100

Out of 120 cases, 78 children were anemic (65%), 42 children (35%)were having their Hb in the normal range.

Table - XVI

DISTRIBUTION OF ANAEMIA IN CHILDREN WITH SYNCOPE										
Anaemia	**Vasovagal syncope**		**Breath holding spells**		**Others**		**Total**		**Chi square**	**P value**
	N	%	N	%	N	%	N	%		
Present	29	37.7	33	42.9	15	19.5	77	64.17	0.003	<0.001
Absent	19	45.2	6	14.3	18	40.5	43	35.83		

Majority of the children with syncopal attacks were anaemic (64%) and the difference was statistically significant.P value was <0.001.

Table - XVII

DISTRIBUTION OF ANEMIA IN VARIOUS SYNCOPES					
S.no	**Causes**	**No**	**Anemia (no.of cases)**	**No anemia (n)**	**%**
1	Breath holding spells	40	34	6	85
2	Vasovagal	48	29	19	60.4
3	Cardiac	8	3	5	37.5
4	Others	24	12	12	50

Table - XVIII

SEVERITY OF ANAEMIA IN B.H.S[52]		
Hb level	**NO.(n)**	**%**
Mild (7-10 gm %)	26	76.5
Moderate (5-7 gm %)	7	20.5
Severe (<5 gm %)	1	3
Total	34	100

Among the anaemic children in BHS, 26 (76.5%) had mild anemia, 7 (20.5%) had moderate anemia, 1 (3%) had severe anemia.

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Table - XIX

DISTRIBUTION OF UNDERNUTRION IN VARIOUS SYNCOPES				
S.No	**Causes**	**Nutrition**		%
		Under(n)	**Normal(n)**	
1	Breath holding spells	19	21	47.5
2	Vasovagal	22	26	45.8
3	Orthostatic Hypotension	2	2	50
4	Cardiac	6	2	75
5	Cough	1	1	50
6	Hyperventilation	--	2	--
7	Migraine	--	1	--
8	Unclassified	5	10	33.3
	Total	55	65	

Table - XX

NUTRITIONAL STATUS IN CHILDREN WITH SYNCOPE										
Nutritional status	Vasovagal syncope		Breath holding spells		Others		Total		Chi square	P value
	N	%	N	%	N	%	N	%		
Under nourished	22	39.3	22	39.3	12	21.4	56	46.67	0.331	>0.05
Well nourished	26	40.6	18	28.1	20	31.3	64	53.33		

Among the 120 cases, 55 (45.8%) were undernourished, 65 (54.2%) were normal nourished. This difference was not found to be statistically significant.

DISCUSSION

DISCUSSION

Total number of cases analysed in our study was 120 during the period of septemder 2009 to august 2010.Incidence of syncope in our study was 57.12cases/lakh/yr compared to Illario bo et al where it was 86.5/lakhs/yr. Mean age of children in our study was 9.5 years with the minimum of 6 months and maximum of 12 yrs.

Study	6mon–3yrs	3 – 6 yrs	6 – 9 yrs	9 – 12 yrs
Driscoll et al 1997	28%	4.7%	13.3%	54%
Present study	30%	6.7%	15.8%	47.5%

The percentage of syncope in each age group was compared with other studies. In the present study and in other studies it was clear that syncope was common as the age increases because of increased incidence of vasovagal syncope with increasing age and it was statistically significant.

Study	Male	Female
Linzer et al 2004[31]	47.3%	52.7%
Present study (2009 – 2010)	39.2%	60.8%

On comparing our study with that of Linzer et al, it was clear that females were commonly affected by syncope. But this difference was found to be statistically not significant in our study.

ETIOLOGY

Types	Steinberg et al[10] 2005	Zhang et al[27] 2005	Present study (2009 – 2010)
V.V.S	46.5%	60%	40%
B.H.S	30.7%	21.2%	33.3%
Cardiac syncope	10.6%	9.8%	6.7%
Others	12.2%	9%	20%

Syncope represents a common and challenging complex of symptoms for the practicing physician.The possible causes of such events are multiple. On comparing our study, with that of Steinberg et al, Zhang et al, there were differences in the percentage of various etiologies of syncope.

As comparable to previous studies, VVS was the most common cause followed by BHS and then others.

Precipitating factors in VVS	Sarasin et al 2001[40]	Present study (2009 – 2010)
Standing in hot sun	52%	47.9%
Crowded places	30.6%	39.6%
Sight of blood	7%	6.25%
fasting	10.4%	6.25

Standing in hot sun was the common precipitating factor for VVS followed by others.This is similar to the study of sarasin et al.

The human body has a remarkable ability to maintain a stable blood pressure in the presence of ever-changing forces that constantly shift and redistribute the circulating blood volume. To achieve this steady control, reflex mechanisms continuously adjust the cardiac output and vascular tone. In individuals susceptible to syncope a paradoxical reflex bradycardia and peripheral vasculsr dilataion occur.

Diurnal Variation	Sarasin et al2001[40]	Zhang et al2005[27]	Present study (2009 – 2010)
Morning	62.5%	65.2%	60.41%
Afternoon	9.6%	10.2%	6.25%
Evening	28.9%	24.6%	33.34%

Syncope was found to be more common in the morning time in all the studies. This may be due to the diurnal variation of autonomic nervous system function.

BHS types	OESIL study (2000)[41]	SEEDS study(2004)[42]	Present study (2009 – 2010)
Cyanotic	81%	78%	80%
Pallid	19%	22%	20%

Cyanotic BHS were common than pallid BHS in all the studies.More than 2/3rd of BHS were cyanotic BHS

Anemia	Lerman et al1994	Graf et al 2000	Present study (2009 – 2010)
Present	54%	52%	65%
No anemia	46%	48%	35%

Majority of children (65 %) were anemic in our study.In other studies it was found to be little less compared to the present study. The difference was found to be statistically significant in our study.This difference may be due to the increased prevalence of anemia in our population.

Family H/O BHS	Laxdal et al (1994)[2]	Lombrosco & Lerman(1967)[16]	Present study
Present	27%	21%	25%
Absent	73%	79%	75%

Family history was present in 25% patients. It was similar to the results found in other studies, suggesting the genetic influence on BHS.

Under nutrition	Graf et al 2000[19]	Present study (2009 – 2010)
Undernourished	25%	55%
Normal nourishment	75%	45%

Comparing to the other studies,percentage of undernourished children were more in our study. But this difference was found to be statistically insignificant.

CONCLUSION

CONCLUSION

1. Among 120 cases majority were coming under the age group of 6 to 12 years($p = 0.001$)
2. The mean age of presentation of syncope was 9.5yrs.
3. Female : male ratio was 1.5:1 ($p = 0.105$). Females were more commonly affected than males.
4. Educational status of the parents have no correlation with the syncopal episode as 66% of them were literate
5. 55.8% children were from lower socio economic status ($p = 0.05$)
6. Vasovagal syncope was the most common cause which accounts for 40% of cases (n=48).
7. Vasovagal syncope was more common in the morning time.
8. Breath holding spells were more common in children of age group 6mon to 3yrs (90%) & majority were cyanotic (80%).
9. Family history was present in 25% of patients with BHS.

10. Anaemia of mild grade was associated with majority of cases with B.H.S. (76.5%) with statistical significance ($p = 0.001$)

11. Nutritional status has no corelation . ($p > 0.05$) .

12. Blood sugar and electrolytes were normal in all patients with syncope.

13. CT brain and EEG showed no significant abnormalities in patients with syncope.

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ANNEXURES

APPENDIX-1

PROFORMA

Name age sex

Address Educational status of

the parents

Father :

Mother :

Family income

History

Complaints:

History:

Duration of symptoms

Time of the day:morning/afternoon/evening

Patient's position:supine/standing/sitting

Patient's appearance during &immediately following the

Episode:pallor/cyanosis

Abnormal movement or posturing

Confusion

Focal neurosymptoms

Amnesia

Muscle soreness

History of

- Fasting/no fasting
- Hot or humid conditions
- Crowded places:bus/temple/festival
- Standing in hot sun-duration:<5min/5-10min/>10min
- Cough duration:<10min/>10min
- Blood drawing or sight of blood

Associated symptoms:

- Palpitation
- Chest pain
- Shortness of breath
- Tingling or numbness of extremities
- Nausea
- Epigastric discomfort
- Diaphoresis
- Headache or visual changes
- Anxiety
- Panic attacks
- Fright
- Pain

Past history:

H/o

- Heart disease
- Neurologic diseases

Past treatment history:

H/o any chronic drug intake

Family history:

- Coronary heart disease
- H/o M.I. <30 years of age
- Family h/o seizures

Examination

Height : cm percentile :

Weight : kg percentile :

HR

PR

B.P : Supine

Sitting

Standing

Auscultation:

Neurological examinations:

Higher functions

Cranial nerves

Spino motor system

Sensory system

Cerebellar function

Investigations:

CBC

Peripheral smear study

Serum glucose

Electrolytes

ECG

X ray chest

Echo

EEG

CT brain

APPENDIX – 3

KEY TO MASTER CHART

KEY TO MASTER CHART

A – NAME

B - AGE

1 – 6m -3yrs
2 – 3yrs -6yrs
3 – 6yrs -9yrs
4 – 9yrs -12yrs

C - SEX

1 - MALE
2 – FEMALE

D – EDUCATION

1 – LITERATE
2 – ILLITERATE

E – SOCIO ECONOMIC STATUS

1 – CLASS IV
2 – CLASS V

F – ETIOLOGY

1 – VASOVAGAL SYNCOPE
2 – BREATH HOLDING SPELLS
a – PALLID
b – CYANOTIC
3 – CARDIAC SYNCOPE
4 – ORTHOSTATIC HYPOTENSION
5 – COUGH SYNCOPE
6 – HYPERVENTILATION SYNCOPE
7 – MIGRAINE
8 – OTHERS

G – PRECIPITATING FACTORS

1 – PRESENT
a – STANDING IN HOT SUN
b – CROWDED PLACES
c – FASTING
d – SIGHT OF BLOOD
e – COUGH

2 – ABSENT

H – FAMILY HISTORY

1 – PRESENT
2 – ABSENT

I – ANAEMIA

1 – PRESENT
2 – ABSENT

J – NUTRITIONAL STATUS

1 – UNDER NOURISHED
2 – WELL NOURISHED

APPENDIX - 5

ABBREVIATIONS

BHS	: Breath Holding Spells.
AS	: Aortic Stenosis.
PS	: Pulmonic Stenosis.
MS	: Mitral Stenosis.
MVPS	: Mitral Valve Prolapse Syndrome.
RV dysplasia	: Right Ventricular dysplasia.
POTS	: Postural Orthostatic Tachycardia Syndrome.
HOCM	: Hypertropic Obstructive Cardiomyopathy.
MI	: Myocardial Infarction

APPEDIX – 4

Stanley Medical College, Chennai – 1
Ethical Committee

CERTIFICATE FOR APPROVAL OF ETHICAL COMMITTEE

To
Dr.G.Sangeetha, PG in MD(Paed)

Dear **Dr.G.Sangeetha, PG in MD(Paed)**
The Institutional Ethics Committee reviewed and discussed your application for approval of the project entitled

"To study the Epidemiology Etiological factor and clinical profile of syncope in children"

The following members of the ethics committee were present at the meeting held on 25.06.2009 at the Modernised Seminar Hall, Stanley Medical College, Chennai-1 at 12.00Noon

Dr.C.B.Tharani, Director of Pharmacology,
Madras Medical College, Chennai-3 Chairman of the Ethics Committee
Dr.A.Sundaram, Vice-Principal,
Stanley Medical College, Chennai – 1 Member Secretary of the Ethics Committee

Members

Dr. Jayanthi
Prof. of Medical Gastroenterology
Dr. Usha Sadasivam
Prof. of Pharmacology
Dr. Lalitha
Prof. of Biochemistry
Dr. Madhan
Prof. of Aneasthesiology
Dr.Thenmozhivalli
Prof. of Microbiology
Dr.S. Ramasamy
Prof.of Medicine
Thiru.G. Karuppasamy
Administrative Officer
Thiru. A. Senthil Manoharan
Advocate

We approve the project to be conducted in its presented form.
The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.
Yours sincerely,

Member Secretary,
Ethical Committee

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.