MEDICAL GRAND ROUNDS

THE OUTPATIENT EVALUATION of SYNCOPE

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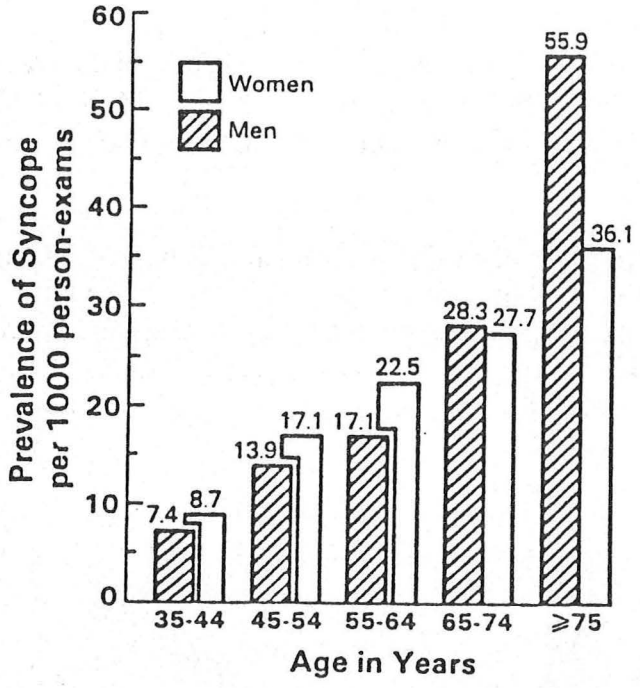
Syncope is a common clinical problem. It can be defined as a transient loss of consciousness with a loss of postural tone followed by spontaneous recovery not requiring specific resuscitative intervention (1,2). The evaluation of syncope spans several specialties and adult patients with this problem are frequently initially evaluated by a physician with broad training in Internal Medicine.

Syncope is a common symptom of an underlying disease or problem. It occurs in 3% to 30% of adults by the age of 75. (3,4) Many of these patients do not even seek medical attention. However syncope accounts for 1% of hospital admissions and approximately 3% of emergency room visits (5,6).

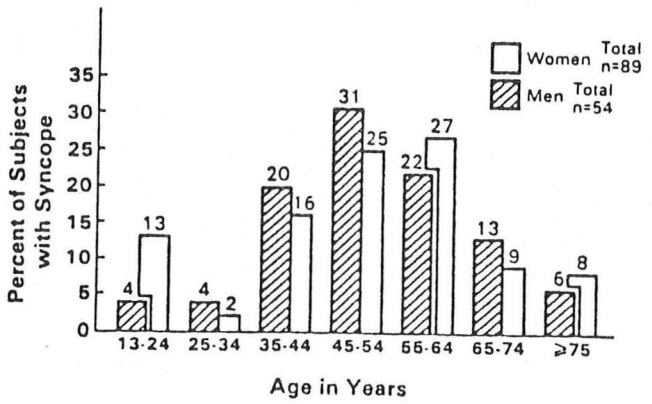
The Framingham study analyzed the epidemiologic information regarding syncope for 2336 men and 2873 women aged 30 to 62 years at entry, who were followed for 13 biennial examinations (7). During their lifetime, 71(3.0%) of the men and 101 (3.5%) of the women had experienced syncope. The mean age at initial episode for men was 52 years (15-78 years) and for women was 50 years (13-87 years). The distribution of ages at the initial episode of isolated syncope is shown in figure 1.

Figure 1.



Prevalence estimates for syncope by age and sex are shown in figure 2. As expected, prevalence increases markedly with age ranging from 7 per 1000 persons in men age 35 to 44 to 56 per 1000 among men age 75 and older. Men and women had similar prevalences except in those age 75 and over, where men were significantly more likely to give a history of syncope (56 versus 36 for men and women respectively).

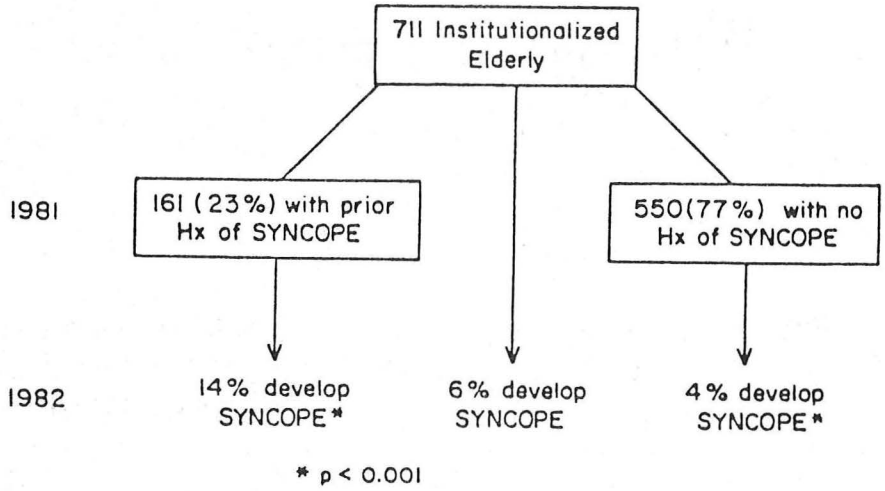
Figure 2.



Age at initial episode of isolated syncope, men and women, the Framingham Study. 26 year follow-up.

A group of people over the age of 75 residing in a long term care institution were found to have an annual incidence of syncope of 6% (6). Twenty-three percent of these people had a previous episode of syncope in their life.

Figure 3.



The spectrum of diseases presenting as syncope is wide. The etiologies can vary from benign problems for which the patient can be reassured to severe life-threatening disorders, for which the syncopal episode may be a prodrome to sudden death. Establishing a cause of syncope is frequently difficult. However, appropriate diagnosis and therapy can prevent recurrences and

possibly prolong life. It can also prevent associated complications, such as trauma from falls and loss of independence in the elderly. (9,10) The available diagnostic tests for the evaluation of syncope are imprecise and often expensive. The clinician evaluating a patient presenting with syncope must make decisions regarding the necessary workup, based on the patient's presentation.

This review describes the differential diagnosis of the etiologies of syncope and provides an analysis of an approach to patients presenting with this problem, focusing on the outpatient evaluation. The prognosis of the various entities that present with syncope will also be discussed.

PATHOPHYSIOLOGY

There are four major pathophysiologic mechanisms of transient loss of consciousness (6,11). The most common of these is a transient reduction of cerebral blood flow. This may result from four abnormalities. The first mechanism may be vasomotor instability developing from a variety of disorders which transiently decreases systemic vascular resistance or venous return or both, with subsequent hypotension and syncope. These disorders may have an associated absolute or relative bradycardia which contributes significantly to the reduction in cardiac output. A second mechanism is a critical reduction of the cardiac output due to obstruction to flow within the heart or pulmonary circulation. Thirdly, cardiac arrhythmias may temporarily reduce cardiac output and produce syncope. The fourth mechanism of reduction in cerebral blood flow is focal cerebrovascular disease. This is a more uncommon cause of syncope.

It is possible to have a normal blood flow to the brain but lose consciousness because of changes in the chemical composition in the blood. The lack of some essential substances necessary for cerebral metabolism can cause syncope.

Loss of consciousness may occur as a result of cerebral dysfunction secondary to seizures. Patients with seizures may present as unexplained syncope, especially if the seizure has occurred for the first time. However, cardiovascular syncope may be accompanied by a few myoclonic jerks of the extremities, a brief tonic extension of the body or even by a generalized seizure if the period of reduced cerebral blood flow is prolonged. Therefore seizure activity is a sensitive but not specific finding for generalized seizures as an etiology of syncope.

Rarely psychiatric or functional disorders may produce an apparent loss of consciousness. The duration of syncope in these individuals is often more prolonged than cardiovascular syncope or seizures. These patients may have unexpected responsiveness to sensory stimuli during the period of apparent unconsciousness.
The pathophysiologic mechanisms are summarized in Table 1.

Table 1.

Pathophysiologic Mechanisms of Syncope
Decreased Cerebral Blood Flow
Vasomotor instability
Decreased cardiac output due to obstruction
Cardiac arrhythmias
Cerebrovascular disease
Altered Chemical Composition of the Blood
Cerebral Dysfunction Secondary to Seizures
Psychiatric or Functional Disorders

DIFFERENTIAL DIAGNOSIS

Syncope can represent abnormalities in several body systems including cardiac, central nervous system, and peripheral vascular. Table 2 summarizes the etiologies of syncope in several large studies. A discussion of each problem follows.

Table 2.

Frequency of Causes of Syncope					
	Cause (percent)				
	Vasomotor Instability	*Cardiac*	*Neurologic*	*Other*	*Unknown*
Wayne 1961 Outpatients & Inpatients, N=510	63.5	11	10.6	10.5	4.5
Day, et. al. 1982 Outpatients, N=198	33.0	8.6	38	8.1	12.5
Silverstein, et. al. 1982 MICU Patients, N=108	4.8	39.0	4.5	4.7	47.0
Kapoor, et. al. 1983 Outpatients, N=123 Inpatients, N=81	18.0	26.0	5.9	2.6	47.5
Martin, et. al. 1984 Outpatients, N-170	46.8	4.1	8.8	2.7	37.6
Lipsitz, et. al. 1985 Nursing Home Residents, N=170	37.0	21.0	4.4	6.6	31.0
Eagle, et. al. 1985 Outpatients, N=176	44.9	8.5	1.1	6.2	39.3

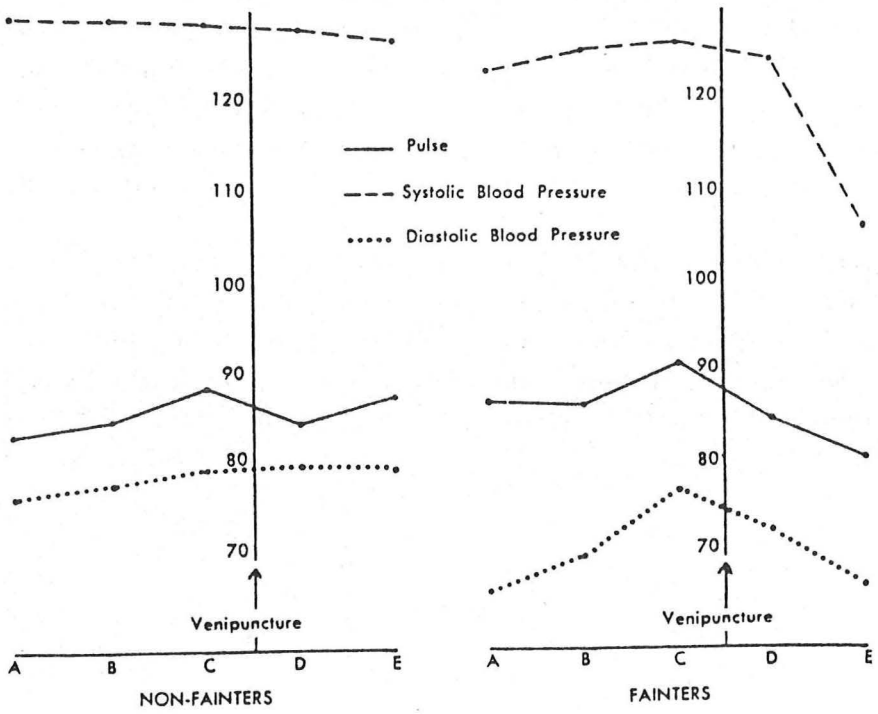
Vasodepressor Syncope

Vasovagal or vasodepressor syncope is the most common type of syncope seen in medical outpatients (5,11). The mechanism of these fainting spells is a transient inadequacy of cerebral perfusion due to inappropriate vasodilatation with pooling of blood in the extremities. Vasovagal episodes are brief and rarely occur while the patient is recumbent.

There are several factors that distinguish vasovagal syncope from other causes of transient loss of consciousness. Vasovagal syncope most often follows a stressful event (11,12). It is most likely to occur when two conditions exist: first the physical injury or threat is one in which the person is relatively unfamiliar or with which he has not been able to cope in the past; second the injury or threat occurs under circumstances that he feels he is expected to face with courage (13). Other spells with a vasodepressor mechanism are related to prolonged standing or assuming the upright position. This may occur while waiting in lines, standing in warm crowded rooms, or rising from bed to empty a full bladder. Factors which predispose to this include large meals, warm baths, pregnancy, malnutrition, fatigue, and physical deconditioning. Hyperventilation may also predispose to vasodepressor syncope.

The sequence of circulatory changes during vasovagal syncope is biphasic. There is an initial period of a minute or two during which heart rate, blood pressure, total systemic resistance and cardiac output all increase (13-15). During this phase the individual becomes apprehensive. The physiologic pattern then abruptly reverses. Heart rate, systemic and pulmonary arterial blood pressures, total systemic and pulmonary vascular resistance, cardiac output and atrial pressure all fall (16).

Figure 4.



Muscle blood flow increases and systemic vascular resistance falls (17). The fall in blood pressure and heart rate is most pronounced in an upright position. Atropine prevents the bradycardia but not the depressor response. Loss of venous tone probably occurs (12). This second phase is associated with characteristic premonitory symptoms which precede vasovagal syncope. These include weakness, nausea, diaphoresis, generalized warmth or numbness culminating in lightheadedness or unconsciousness if recumbency is not achieved before the systolic blood pressure has fallen below 70 to 80 mm Hg. The loss of consciousness is abrupt and brief. Alertness resumes soon after the patient is recumbent. The patient may experience a few brief clonic movements or incontinence of urine due to cerebral hypoperfusion but post event confusion does not follow vasovagal syncope.

Some persons experience vasovagal spells recurrently. The history of preceding typical episodes supports but is not necessary to make the diagnosis. Symptoms similar to vasovagal syncope may occur in carotid sinus sensitivity. Estimates of the incidence of vasovagal syncope vary according to how strictly the criteria for this diagnosis are applied. In two large studies of patients presenting for medical care as the result of syncope, vasovagal syncope was diagnosed in 13 to 48% of the cases (3,11).

Orthostatic Hypotension

Orthostatic hypotension is implicated as the cause of syncope if there is a decrease in systolic blood pressure of more than 25 mm Hg on standing or any reproducible decrease in blood pressure that produces symptoms (1,5,9,18). Decreases in systolic blood pressure of 10 to 25 mm Hg when associated with a systolic pressure decreased to less than 90 mg Hg are also considered diagnostic for orthostatic hypotension.

Orthostatic hypotension may be caused by volume depletion, adrenal insufficiency, profound deconditioning, phenothiazine, antidepressant or antihypertensive drugs, or peripheral neuropathy. Primary disorders of sympathetic tone that cause orthostatic hypotension include those with central nervous system involvement, Shy-Drager syndrome (an intact peripheral nervous system) and those with peripheral autonomic dysfunction. Symptoms of the disorders of sympathetic tone can be incapacitating (19).

The mechanism of the fall in blood pressure with its resulting cerebral ischemia is passive loss of peripheral resistance as opposed to reflex loss of peripheral resistance with vasodepressor syncope. Certain hemodynamic changes take place with assuming the upright position which are necessary to counteract the effects of gravity (11). Contraction of muscles in the lower extremities and their increase in tone aid in venous return. Prolonged strapping of healthy individuals to a tilt table will result in spontaneous loss of consciousness in a certain number of subjects. Immobilization in the erect position following strenuous exertion predisposes to orthostatic syncope (20). Patients with postural hypotension do not pool more blood, but normal pooling causes an abnormal fall in blood pressure due to absence of reflex vasoconstriction (21).

Decreased blood pressure on standing of greater than or equal to 20 mm Hg is reported to occur in as many of 24% of persons over 65 years of age. This is accounted for by numerous age related physiologic changes (2). Baroreflex sensitivity decreases with age. Drug induced in-

creases in blood pressure result in less heart rate reduction in old persons than in young (22). In addition elderly persons are more sensitive to the blood pressure lowering effects of drugs and show a marked blunting of reflex tachycardia in response to blood pressure lowering (23,24). Elderly people are less able to compensate for sudden drops in blood pressure.

Age related stiffening of the carotid arteries and the aortic arch may reset baroreflex sensitivity by producing less tonic input to the vasomotor center for any given level of blood pressure (25). Aged hearts tend to lose their sensitivity to adrenergic stimulation (25), thus they appear to have a decreased heart rate response to upright posture. Data indicate that there is a postreceptor defect in sympathetic responsiveness in the aged heart (26,27).

Adaptive mechanisms responsible for maintaining extracellular fluid volume are also impaired in elderly persons. The aged kidney takes longer to conserve sodium when salt intake is restricted (25). This contributes to rapid volume depletion in acutely ill elderly patients who discontinue oral intake. In addition plasma renin and aldosterone concentrations are diminished by 30 to 50% in elderly persons. Salt restriction, diuretic administration and upright posture, which augment renin secretion in the young, produce less of a response in elderly persons. All of these mechanisms of aging make an older person especially vulnerable to syncope. When the effects of a drug or disease are added to a system that is already marginally balanced, syncope often results.

Orthostatic hypotension accounts for 3 to 7% of the cases of syncope that have presented for medical evaluation in large studies.

Carotid Sinus Syncope

The carotid sinus has an important role in the normal reflex regulation of heart rate and peripheral vascular tone. In patients with hypersensitive carotid sinus syndrome, symptoms result from transiently diminished cerebral perfusion due to an exaggerated cardiovascular response to carotid sinus baroreceptor stimulation (28). Mechanical stimulation of the carotid sinus in normal subjects results in a mild slowing of the heart rate and an insignificant decrease in blood pressure (29). A few otherwise healthy subjects have a hypersensitive carotid baroreflex with an exaggerated response to stimulation of the carotid sinus (30). Three types of responses have been described: 1) a cardioinhibitory response in which marked sinus bradycardia and/or transient atrial ventricular block results in systemic hypotension; 2) a vasodepressor response in which arterial hypotension occurs in the absence of marked bradycardia; 3) a mixed response in which both cardioinhibitory and vasodepressor factors contribute to hypotension (29,31).

The carotid sinus baroreceptors consist of extensively branched and myelinated nerve endings located in the adventitia of a small dilatation of the internal carotid artery just above the division of the common carotid artery into its internal and external branches. Afferent impulses from the carotid sinus ascend in the glossopharyngeal and vagus nerves to the sensory nucleus of the vagus. An increase in intrasinus tension results in the activation of the dorsal motor nucleus of the vagus (cardioinhibitory center) and inhibition of tonic discharge from the vasomotor center with concomitant slowing of the heart rate and decrease in blood pressure. The efferent limb of

the reflex arc passes through the vagus nerve and sympathetic outflow (30).
The pathogenesis of the hypersensitive carotid sinus reflex is unknown. Enlarged cervical lymph nodes, local tumors, previous neck radiation, and aneurysmal dilation of the internal carotid have been implicated in some patients. However, most patients have no demonstrable pathologic abnormalities in the carotid sinus (18).

Carotid sinus sensitivity is not a common cause of syncope, occurring in less than 3% of individuals who present for medical evaluation of syncope. However, it is a potentially treatable cause of recurrent neurologic symptoms, so if the history is at all suggestive the diagnosis should be pursued (32). The test of choice for confirming the presence of carotid sinus sensitivity is carotid sinus massage. Auscultation of the carotid arteries prior to this maneuver is mandatory. Patients with known carotid or intracranial arterial disease should not be studied because of the risk of cerebral arterial embolism after carotid sinus massage (33). Carotid sinus massage should be performed with continuous electrocardiographic and blood pressure monitoring. Firm pressure is applied to the carotid below the angle of the mandible for five seconds. At least a thirty second pause should elapse between massage of each of the carotid sinuses. The test should be undertaken on both carotid sinuses in that the hypersensitivity may be unilateral. Ideally the temporal arteries should be palpated simultaneously to ensure that carotid occlusion has not occurred. If carotid sinus massage in the supine position does not yield a positive result, it should be repeated in the upright position since a vasodepressor response may only be detected in this position (34). An abnormal response is considered to be cardiac asystole lasting three seconds or more and/or a decrease of systolic and diastolic blood pressure of 50 mm Hg. A borderline abnormal response is defined as a slowing of the heart rate by 30 to 50%, cardiac asystole lasting two seconds, or a decrease of 30 mm Hg in systolic blood pressure (18,32,35). Responses fitting these criteria allow a diagnosis of carotid sinus hypersensitivity to be made as an etiology of syncope. Carotid sinus hypersensitivity may not be consistently reproducible in a given patient (36). This may contribute to difficulty in making a diagnosis. Although it is the rarest of the responses to carotid sinus hypersensitivity, patients with pure vasodepressor response have been described (34), therefore monitoring of the blood pressure is an important component of this test.

Situational Syncope

Syncope can be precipitated by several specific situations. These include cough, micturition, defecation and deglutition.

Tussive or cough syncope is an uncommon but easily recognized syndrome. Three mechanisms have been proposed to explain the pathophysiology of cough syncope (37). Coughing in these patients produces extremely high intrathoracic pressures, resulting in an exaggerated Valsalva response with decreased venous return and decreased cardiac output (38,39). In addition the intrathoracic pressures during cough are transmitted to the subarachnoid space. The resulting extremely high pressure in the cranium reduces blood flow and predisposes to syncope (40). The rapid rise in intracranial pressure can also result in a concussion effect that contributes to syncope (41).

The typical case of cough syncope is benign and does not require extensive evaluation. However, several patients with co-existing reversible problems have been described. These include stenosis of the carotid artery (37), heart block (42,43), hypertrophic obstructive cardiomyopathy (44), hypersensitive carotid sinus (45), and herniation of the cerebellar tonsils (46). Generally the patient with cough syncope should be evaluated with a history and physical examination. If the patient smokes, he or she should stop and any airway obstruction should be treated aggressively. If these measures are unsuccessful in preventing recurrent cough syncope, further work up to exclude co-existing treatable cardiovascular or neurologic disease contributing to the syncope should be undertaken.

Micturition syncope was originally described as occurring in healthy men who, after rising from bed in the early morning hours, experienced sudden loss of consciousness during or immediately after urination (47,48). Predisposing factors include reduced food intake, recent upper respiratory tract infection, fatigue, and ingestion of alcohol.

As a part of a large study of individuals presenting with syncope, Kapoor and colleagues prospectively evaluated thirty-three patients who had syncope in association with micturition (49). Eight of these patients were young healthy individuals similar to the patients described previously. All of these patients had normal physical exams and laboratory findings. However, the majority were older with a mean age of 60. They had multiple concurrent medical problems with an average of 3.8 illnesses per patient. Nine of the twenty-five patients had recurrent episodes of syncope. The physical examination in these patients revealed orthostatic hypotension in twenty-two of the twenty-five patients. Of the patients who had orthostatic hypotension, fourteen had been taking diuretics, two had been at prolonged bedrest, two had recent gastrointestinal bleeding, one had autonomic neuropathy, one had nausea and vomiting, and one had orthostatic hypotension with paroxysmal supraventricular tachycardia.

Table 3.

Patient Characteristics

	No. of Patients	
	Group I (N=I8)	Group 2 (N=25)
Mean age, yr (range)	*25(I8-34)*	*60(39-88)*
Underlying illnesses	I	49
Posture preceeding syncope		
Recumbent	5	21*
Sitting	3	2
Sleeping	4	14
Phase of Urination		
Beginning	I	2**
During	5	9
Termination or just after completion	2	II

**In two patients, not known*
***In one patient, not known*

The incidence of micturition syncope in the general population has been reported to be very low. However, in Kapoor's group, nine percent of 400 patients who presented for syncope had a history consistent with micturition syncope. Many mechanisms may contribute to micturition syncope. These include physiologic changes during sleep or micturition, such as decompression of the bladder leading to hypotension (45), a decline of blood pressure and heart rate during sleep (51), vagal stimuli associated with micturition (22) and Valsalva maneuver leading to decreased venous return. In addition, as noted in Kapoor's older patients, baseline orthostatic hypotension may be exacerbated by one or more of these physiologic changes.

Defecation syncope is a symptom with multiple etiologies. Kapoor evaluated twenty patients in whom syncope occurred in association with defecation (53). Nine of these twenty patients were found to have underlying diseases that may have contributed to their syncope. Two patients had underlying gastrointestinal tract disease. One of these had a Meckel's diverticulum and the second a ruptured appendix. Three patients had underlying cardiovascular disease-one with complete heart block, one with ventricular tachycardia, and one with myocardial infarction and bradycardia. One patient had cerebrovascular disease and three patients were found to have orthostatic hypotension. No identifiable cause could be found in the other eleven patients.

Table 4.

Categories of Defecation Syncope

Potential Causes	*No.*
Gastrointestinal tract	
Meckel's diverticulum	I
Ruptured appendix	I
Cardiac	
Complete heart block due to digoxin toxicity	I
Ventricular tachycardia	I
Myocardial infarction-bradycardia	I
Cerebrovascular disedase	I
Possibly related to orthostatic hypotension	3
No identifiable cause	II

The mechanism of defecation syncope is not well understood. Perhaps an interaction of physiological changes secondary to defecation combined with underlying acute or chronic medical disorders may lead to hypotension and/or bradycardia resulting in syncope. Forty-five percent of patients in Kapoor's evaluation had significant underlying disease. Defecation syncope is probably not a single distinct clinical entity. Patients with these symptoms should be evaluated for underlying disease causing or contributing to syncope.

Swallowing is a complex neuromuscular activity that depends on an interaction of many reflex activities. Esophageal peristalsis is mainly dependent on vagal stimulation (54). Distension of the esophagus with an inflated balloon in some patients with swallow syncope has been reported to produce transient bradyarrhythmias (55).

Swallow syncope seems to be relatively rare with only 34 cases reported in the literature (56-64). However, swallow syncope may be underreported in that a careful history to associate syncopal episodes with the act of swallowing may not be obtained. In one review of twenty-two cases of swallow syncope, sixteen had demonstrable esophageal disease (58). These included spasm, achalasia, diverticulum, spasmodic stricture, hiatal hernia and cancer. Another evaluation of five patients with swallowing syncope revealed two to have underlying esophageal disease and two to have underlying cardiovascular disease (64). While the majority of patients with swallow syncope have a bradyarrhythmic response, rare patients have clinically been felt to have a vasodepressor response. Those patients with a bradyarrhythmic response tend to respond well to pacemaker placement.

Intracardiac Obstruction and Other Cardiovascular Disorders

Cerebral blood flow may be critically reduced by obstruction to blood flow within the heart. Lesions which may obstruct flow enough to cause syncope include aortic or pulmonic valve stenosis, hypertrophic cardiomyopathy, prosthetic valve malfunction, and atrial myxoma (6). Syncope classically occurs with exertion when obstruction is an etiology.

Syncope is a common complication of aortic stenosis. Its incidence with other valvular lesions is rare. Syncope is noted in from twelve to twenty-five percent of patients with aortic stenosis (11,65). There are many hypotheses regarding the pathogenesis of syncope in aortic stenosis. These include ventricular arrhythmias (66), abrupt left ventricular failure (67), and baroreceptor malfunction with excessive vasodilation triggered by intracardiac reflexes (68). This last theory is best supported by the current literature (65,69).

Structural disorders of the heart accounted for twenty-eight of twenty-nine cases of sudden death of young athletes (70). Hypertrophic cardiomyopathy was the most common etiology (l4 patients). Patients with hypertrophic cardiomyopathy are not a homogenous group regarding mechanisms of syncope. High grade ventricular arrhythmias have been reported to cause syncope in these individuals (71). In addition bradyarrhythmias responding to pacemaker therapy have been described in patients with hypertrophic cardiomyopathy (72).

Syncope during or immediately following physical exertion suggests a potential cardiac disorder. Heart murmurs on examination of patients with this presentation should be carefully evaluated as representing a lesion that may be a potentially treatable cause of syncope.

Ischemic heart disease is a common coexisting problem in patients presenting with syncope who have other medical illnesses (73). There are several mechanisms for syncope in association with ischemic heart disease including: bradyarrhythmias, tachyarrhythmias, decreased left ventricular output due to acute ischemia, and orthostatic hypotension due to drugs used to treat ischemic heart disease.

Intrapulmonary Obstruction to Flow

Syncope occurs in more than ten percent of patients with pulmonary embolism (74,75). Syncope is more likely to occur in patients with massive pulmonary embolism. It is generally believed that the majority of these cases of syncope occur because of occlusion of the pulmonary artery with decrease in blood flow to the left side of the heart and the systemic circulation. This is generally accompanied by increased sympathetic tone and tachycardia. However, bradycardia during pulmonary embolism has been reported (74). This suggests that in some cases the syncope accompanying pulmonary embolism may be due to a vagal mechanism.

Generally syncope associated with pulmonary embolism will occur in the settings of other symptoms suggestive of pulmonary embolism such as dyspnea, weakness, pleurisy or hemoptysis. However, occasionally syncope has been reported as the primary symptom with few other signs of pulmonary embolus (5). In this case diagnosis may be overlooked in the initial presentation.

Pulmonary hypertension, because of its hemodynamic effects on cardiac output may also be an intrapulmonary cause of syncope.

Cardiac Arrhythmias

Both bradyarrhythmias and tachyarrhythmias can cause syncope because of diminished cardiac output (76). It is estimated that 25,000 new cases of syncopal attacks due to arrhythmias occur per year (77,78). Syncopal spells due to arrhythmias typically occur without warning or with only brief lightheadedness and occasionally palpitations (1). They occur in any position, including recumbency.

Supraventricular tachycardia, ventricular tachycardia, and bradyarrhythmias of all types are about equally common as causes of syncope (79). Elderly persons may faint due to arrhythmias that would be tolerated by younger persons. Sustained palpitations may occur prior to a spell of syncope, with the syncope being related to a prolonged sinus pause following spontaneous reversion of a supraventricular tachycardia to sinus rhythm (80). With complete heart block, most patients will recover spontaneously from syncope because of restoration of cerebral blood flow by an idioventricular rhythm (81). However, only 50% of these patients will survive for one year without a pacemaker (82).

On the basis of data on electrocardiographic monitoring of large groups of symptomatic and asymptomatic individuals, the following findings are considered indicators for potential causes of syncope.

1.Symptomatic sinus pauses (symptoms include dizziness or syncope)
2.Symptomatic sinus bradycardia.
3.Sinus pauses of more than two seconds.
4. Atrial fibrillation with a slow ventricular response (R R interval greater than 2 seconds).

5.Symptomatic supraventricular tachycardia.
6.Supraventricular tachycardia leading to hypotension (systolic blood pressure less than 90).
7.Complete atrial ventricular block.
8.Mobitz Type II atrial ventricular block.
9.Ventricular tachycardia that is symptomatic, sustained, or accompanied by hypotension.
10. Asymptomatic ventricular tachycardia of more than five beats.

Cerebrovascular Disease

Transient Cerebral Ischemic Attacks (TIAs) - As a rule TIAs produce focal neurologic deficits rather than alterations of consciousness. Except for the association with cerebral emboli, transient focal neurologic deficits induced by arrhythmias are rare, even in patients with underlying carotid or vertebrobasilar disease. There are some situations in which cerebrovascular disease may lead to altered consciousness (1). These include bilateral extracranial carotid occlusive disease, migraine headaches (possibly induced by vertebral basilar arterial spasm) and transient brain stem ischemia. Syncope due to transient ischemic attacks may be accompanied by focal neurologic deficits, incoherence, and confusion. The diagnosis of this etiology of syncope may be suspected because of the above accompanying symptoms in addition to vertigo, ataxia, and alteration of vision in an elderly person or someone with probable underlying atherosclerosis.

Subclavian Steal Syndrome - Syncope can occur with subclavian steal syndrome. It is diagnosed by the presence of the typical clinical and radiologic manifestations of this syndrome (93). The common coexisting symptoms of subclavian steal are outlined below.

Table 5.

Frequency of Symptoms in 168 Patients

Symptoms	*No. of Patients*	*Frequency, %*
Diplopia	32	19.0
Vertigo	87	52.0
Syncope	30	18.0
Monocular visual	27	16.0
Bionocular visual	52	31.0
Limb paresis	58	34.0
Ataxia	43	26.0
Intermittent claudication	22	13.0
Dysarthria	21	12.5
Paresthesias	56	33.0
Mental changes	25	15.0
Dysphasia	17	10.0

Seizures

While strictly speaking seizures are not considered a form of syncope, a first time seizure is a consideration in a patient who presents with loss of consciousness. Even if a sudden loss of consciousness is transient and unwitnessed, the diagnosis of seizure can frequently be made from the clinical features. Seizures typically occur without warning. When an aura precedes a seizure it consists of a localized neurologic occurrence such as an olfactory sensation, a complex visual or auditory phenomenon, or a sinking feeling. The generalized feeling and other premonitory symptoms that often precede syncope from a vasovagal etiology do not occur with seizures.

Seizures can occur in any position and at any time including during sleep. Tonic clonic contractions occur with seizures but may also occur in patients who faint from decreased cerebral blood flow. Experiments in the 1940's (84) induced rapid cessation of blood flow to the brain by means of an occlusive cuff around the neck of humans. Fixation of the eyes, constriction of visual fields, paresthesia of the extremities and tonic clonic convulsions occurred in most subjects. Urinary and fecal incontinence also occurred.

The most specific feature that differentiates seizures from other causes is postictal confusion.

Metabolic and Drug Induced Alterations of Consciousness

It is possible to lose consciousness because of the lack of some essential substance necessary for cerebral metabolism. In addition many toxic agents can cause unconsciousness. The majority of these changes in the blood composition are more likely to result in delirium or coma. However, occasionally only a transient loss of consciousness can occur.

Hypoglycemia is usually accompanied by palpitations, anxiety, sweating, and hunger sensation and only rarely progresses to actual loss of consciousness. It most frequently occurs in patients taking hypoglycemic drugs who have not eaten adequately.
Other metabolic parameters that can cause syncope or syncope like episodes include hypoxia, hypocapnia, hypercapnia and nutritional disturbances. Agents that depress the central nervous system such as alcohol, barbiturates, and other drugs can cause loss of consciousness, but this is frequently more prolonged than simple syncope.

Hysteria

Hysterical patients may appear unresponsive. Their waxing and waning unresponsiveness generally persists longer than syncope. Blood pressure and pulse are normal.

Syncope of Unknown Origin

There are always some patients in whom the etiology of the syncope cannot be determined despite extensive evaluation, 12.5 to 47.5% of these patients fall into this category (3,8,9,85-87). The number of patients in this category seems to be related to the rigidity of critieria for establishing the cause of syncope and the setting in which the patients are evaluated. Patients presenting to ambulatory settings for evaluation of syncope (5) are less likely to be diagnosed as syncope of unknown origin than are patients who are admitted to the hospital for extensive work up after recurrences as syncope (88).

Several factors may contribute to the diagnostic difficulty of syncope. Premonitory symptoms of predisposing factors characteristic of a given disorder may be absent, and the syncopal episode may have not been witnessed. Many causes of syncope, such as cardiac arrhythmias, occur episodically and are rarely evident at the time of physical examination. The physician infrequently has the opportunity to observe or directly monitor the patient during a syncopal episode. Even if abnormalities are found, they may be difficult to link with certainty to a patients syncopal episode.

A summary of the etiologies of syncope organized by major system is presented below in Table 6.

Table 6.

Etiologies of Syncope

Peripheral Vascular	Cardiopulmonary
1. Vasovagal	1. Obstruction
2. Orthostatic hypotension	Aortic stenosis
3. Carotid sinus sensitivity	Pulmonic stenosis
4. Situational syncope	Hypertrophic cardiomyopathy
Tussive	Atrial myxoma
Micturition	Prosthetic Valve Malfunction
Defecation	2. Ischemic heart disease
Deglutation	3. Arrhythmias
	4. Pulmonary embolus
Neurologic	5. Pulmonary hypertension
1. Seizures	
2. Cerebrovascular disease	Toxic/Metabolic
3. Cerebral vasospasm	1. Hypoglycemia
4. Subclavian steal	2. Hypoxia
	3. Hypocapnia
Psychogenic	4. Hypercapnia
1. Hysteria	5. Nutritional deficiencies
2. Conversion reaction	6. Drugs

DAGNOSTIC EVALUATION

The diagnostic evaluation of syncope can be very expensive. In one study of patients who were hospitalized for evaluation of recurrent syncope, the cost of each diagnosis made was $23,000 (88). The major task of the primary care physician is to evaluate syncope using a cost effective strategy that maximizes diagnostic efficiency while limiting the use of unnecessary tests (89).

In those patients in whom a diagnosis of the etiology of syncope can be established, the history and physical examination have the highest yield for making that diagnosis. In a study of patients evaluated in an emergency room for syncope over a period of a year, the history and physical exam were sufficient for making the correct diagnosis in 85% of the patients (5). Other studies which have more selected patient populations, such as hospitalized patients or patients referred for specialty evaluation, reveal a diagnostic yield of the history and physical examination of approximately 50% (6).

History

The most pertinent component of the history is a minute by minute account of the patient's syncopal episode (85). These can be divided into three periods of time: presyncope; syncope; and postsyncope.

Information on the presyncopal period must include the patient's activity and position just prior to syncope. Data regarding this activity should include whether the patient was at rest or exertion, if there was a change in posture or if the syncope occurred following coughing, urination, defecation, or swallowing. It should be noted whether the patient was standing, sitting, or supine at the time of the syncope.

Associated symptoms immediately prior to the loss of consciousness are important. Particular attention should be paid to discerning symptoms of weakness, sweating, epigastric discomfort, nausea, dizziness, lightheadedness, blurring or fading of vision, change in hearing or tinnitus, feeling of generalized numbness or warmth, paresthesia, pallor, shortness of breath, chest pain, aura, palpitations, or an awareness of being about to faint. Have the patient estimate the duration of the presyncopal symptoms.

In order to establish a diagnosis of vasovagal syncope, precipitating factors should be elicited. These include emotional tension, fear, apprehension, fatigue, lack of food or sleep, a hot environment, pain, and the sight of blood. With syncope of a neurologic etiology, presyncopal focal neurologic symptoms may be identified. These include vertigo, diplopia, ataxia, dysarthria, hemiparesis, and unilateral numbness.

Data regarding the syncopal episode itself may be best collected from witnesses. Important information includes an estimated duration of the loss of consciousness and whether the patient fell. In addition, symptoms exhibited during the loss of consciousness such as convulsions, automatisms, and loss of urine or stool are very helpful.

Residual symptoms that persist in the postsyncopal period are important. These include postictal confusion or lethargy, amnesia, muscle soreness, and focal neurologic symptoms. Additional medical history focusing on the presence or risk of illness known to predispose to syncope, medications or other ingestions, and previous syncopal episodes will be of great help in establishing a diagnosis of the etiology of syncope.

Physical

There are several components of the physical exam that must be documented in evaluating a patient with syncope. These include orthostatic blood pressure and heart rate. These measurements should be taken with the patient in each position for three minutes and include the standing position if possible. The cardiovascular exam should be attentive to the carotid upstroke, presence of carotid bruits and murmurs compatible with obstructive cardiac lesions. A neurologic exam should detail any alterations in mental status or focal neurologic findings. If the initial neurologic exam is abnormal, serial neurologic examination should be undertaken to document a clearing postictal state. In patients in whom there are no contraindications, carotid sinus massage can be performed with appropriate monitoring and precautions. A stool hemoccult should be documented and is imperative if the patient has orthostatic changes in vital signs or signs and symptoms suggestive of anemia. Other components of the physical exam can be included if indicated by data from the history or a limited physical exam.

Laboratory Tests

Routine laboratory tests in the evaluation of syncope have an extremely low yield. Tests frequently performed include complete blood counts, serum electrolytes and/or blood glucose determinations, and toxicology screens. These tests may be important in confirming an abnormality suspected by history and physical exam such as hypoglycemia. Rarely will they reveal an otherwise unsuspected etiology of syncope.

In a study of 204 patients presenting with syncope, Kapoor found that the initial basic laboratory evaluation did not suggest a cause of syncope in any patients (9). In an evaluation of 198 patients presenting to an emergency room with transient loss of consciousness, Day found that diagnostic laboratory tests were frequently ordered (5). Of 130 complete blood counts that were ordered, no occult bleeding was identified. The CBC did confirm a known vaginal bleed in one patient. The diagnosis had been made on the basis of the history and physical exam. The 130 serum electrolyte and/or blood glucose determinations revealed abnormalities which proved helpful in five patients. Two patients had hypoglycemia confirmed by the blood glucose level; however, it had already been diagnosed and treated. A third patient had seizures secondary to hypocalcemia. This patient was known to be hypoparathyroid and off medications. A fourth patient with known uncontrolled chronic renal failure had uremia on evaluation. One unexpected metabolic abnormality was a serum sodium of 110 mEq/L in a psychotic patient who presented with a seizure several days after starting therapy with a thiazide diuretic.

In general initial laboratory evaluations should only be ordered to document suspected abnormalities contributing to syncope. If on the basis of history and physical exam a diagnosis of nonmetabolic etiology of syncope is made, routine laboratory tests can be omitted as the yield is extremely low.

Electrocardiogram

An electrocardiogram done at the time of evaluation of a syncopal episode is frequently abnormal. However, abnormalities that make the diagnosis of the cause of syncope are only present approximately five percent of the time. Diagnostic findings on electrocardiogram include bradyarrhythmias, tachyarrhythmias, or acute myocardial infarctions that precipitated the episode of syncope. Electrocardiographic abnormalities may be present that are not diagnostic of the cause of syncope but may suggest the presence of disorders such as Wolf Parkinson's White Syndrome or structural heart disease and help guide the selection of other tests to confirm the etiology of the syncope (6). Patients who have an abnormal cardiovascular exam, have a history suggesting cardiovascular disease or risk thereof, or who are over 45 years of age should have an electrocardiogram as a part of the initial evaluation of syncope.

Prolonged electrocardiographic Monitoring

Prolonged electrocardiographic monitoring has assumed an important role in the evaluation of patients with syncope when a diagnosis has not been established initially (9,90). However arrhythmias frequently occur in asymptomatic healthy subjects undergoing prolonged monitoring (91-99). In addition, asymptomatic arrhythmias are frequently found during monitoring of patients evaluated for syncope (100,101). Abnormalities noted in healthy individuals include sinus bradycardia down to a rate of 42 beats per minutes in twenty-five percent of patients; brief runs of supraventricular tachycardia in fifty percent of patients; PAC's or PVC's in fifty to seventy-five percent of subjects; and frequent, multiform, or paired PVC's in fifteen percent of subjects (102). Even sinus pauses of two to three seconds and brief unsustained runs of ventricular tachycardia have been reported to occur in two to four percent of normal healthy subjects. Therefore the occurrence of these arrhythmias in patients being evaluated for a prior syncopal episode does not necessarily establish a cause for the loss of consciousness. However, prolonged electrocardiography can provide significant information in determining the cause of syncope if strict criteria are applied.

The most diagnostic finding is a temporal correlation between symptoms of syncope or dizziness and electrocardiographic findings. However, syncope rarely occurs during monitoring, so strict adherence to this approach greatly underestimates the prevalence of arrhythmias potentially leading to syncope. Another helpful factor if syncope occurs during monitoring is that if arrhythmias are not present during the episode, they may be ruled out and other causes of the syncope investigated. However, temporal correlation between syncope and electrocardiographic findings occur in only two to three percent of patients (103).

Criteria based on asymptomatic findings on prolonged electrocardiographic monitoring have not been widely agreed upon. Some investigators have accepted liberal criteria such as bradycardia less than fifty-five beats per minute, sinus pauses of more than one and one half seconds, supraventricular tachycardia of six or more beats, any atrial ventricular block except first degree, and PVC's as potentially diagnosed arrhythmias (100,101). This approach may not be restrictive enough and may lead to misdiagnosis of the etiology of syncope and overtreatment of arrhythmias.

It is generally accepted that runs of asymptomatic ventricular tachycardia of any length implicate ventricular tachycardia as the cause of syncope (5,9,85,104-106). These criteria will identify a group of patients at high risk for sudden death (5,9).
Kapoor and his group attempted to related arrhythmias detected on monitoring with ultimate outcomes to help determine the clinical importance of the arrhythmias (102). They found that the incidence of sudden death in patients with frequent or paired PVC's was higher than that in patients with rare PVC's. In addition, the overall mortality rate in patients with frequent or paired PVC's was higher than that in patients with rare PVC's. These incidences of sudden death and mortality rate in patients with frequent or paired PVC's were similar to those in patients with ventricular tachycardia.

Table 7.

Relation of Findings on Initial Monitoring and Outcome In Subsequent Follow-up

	No. of Pts	*Cardiac Arrest*	*Sudden Death*	*Total Deaths*
PVC's				
Rare (<10/hr)	133	0	4	8
Frequent (>10/hr) or paired	32	0	5	9
Ventricular tachycardia				
3-9 beats	23	3	3	8
10-30 beats	7	0	3	4
31-210 beats	5	0	0	1
Sinus pauses	7	0	1	2
Mobitz II complete heart block	3	2	0	2
Other	25	1	0	3
Total	235	6	16	37

Other findings on electrocardiographic monitoring that are considered indicators for potential causes of syncope based on reviews of monitoring in asymptomatic persons and persons with syncope include: atrial fibrillation with a slow ventricular response (R-R interval greater than two seconds); symptomatic supraventricular tachycardia or supraventricular tachycardia leading to hypotension (systolic blood pressure lower than 90 mm Hg); third degree atrioventricular block; and Mobitz II second degree atrioventricular block (102).

Prolonged monitoring is certainly indicated for patients suspected of having cardiac syncope due to arrhythmias. Those patients at high risk for suspected cardiac syncope would include older patients, patients with known or potential underlying cardiac disease, patients presenting with syncopal spells without warning, or in recumbency or accompanied by palpitations, chest pain, or dyspnea. The monitoring of patients at low risk for underlying cardiac etiologies of syncope is unlikely to produce findings that correlate with the syncope. This would include those under the age of thirty with no history or suspicion of cardiac disease and those under the age of seventy with suspected vasovagal syncope. The cost of prolonged monitoring is not warranted in these individuals.

Electrophysiologic Study

A detailed discussion of electrophysiologic testing in the evaluation of syncope is beyond the scope of this review. However, it is important to know which patients benefit from this specialized intervention. A summary of several studies of electrophysiologic testing in patients with syncope is outlined in the table below (105).

Table 8.

Comparison of Studies of Patients with SUO					
Study	*Patients*	*Multiple SUO*	*OHD*	*Abnormal ECG*	*Abnormal EPS*
		(%=% of patients tested)			
DiMarco,et.al.	25	25 (100%)	21 (84%)	22 (88%)	17 (68%)
Brandenberg,et.al.	92	N/A	N/A	N/A	59 (64%)
Hess,et.al.	32	26 (81%)	17 (53%)	18 (56%)	18 (56%)
Akhtar,et.al.	3	31 (100%)	18 (60%)	17 (57%)	16 (53%)
Morady,et.al.	53	53 (100%)	38	23 (79%)	38
Gulamhusein,et.al.	34	N/A	0	0	4 (12%)
Westveer,et.al.	31	31 (100%)	21 (68%)	15 (48%)	20 (64%)
Olshansky,et.al.	105	N/A	57 (54%)	N/A	47 (45%)
Teichman,et.al.	105	32 (30%)	57 (54%)	77 (73%)	83 (79%)
Abbreviations: *EPS = electrophysiologic studies; OHD = organic heart disease*					

Electrophysiologic testing is generally undertaken in patients who have syncope of unknown origin despite thorough evaluation. Those patients at higher risk of dysarrhythmias include those with underlying heart disease or an abnormal electrocardiogram. Because the subsequent risk of recurrent syncope and/or significant morbidity or mortality in patients with underlying arrhythmias is high, if the etiology of syncope can not be determined by noninvasive studies, electrophysiologic testing is indicated.

Other Cardiovascular Testing

Exercise tolerance tests and cardiac catheterizations can be helpful in evaluating patients with syncope. The yield of these tests is related to the suspicion of underlying cardiac ischemic disease in the case of exercise tolerance tests or structural cardiovascular disease in the case of cardiac catheterization. These studies should be undertaken only in patients in whom a high suspicion of these problems exist on the basis of the history, physical exam, and electrocardiogram. The signal averaged electrocardiogram is a new technique in electrocardiography designed to aid and detect susceptibility to malignant ventricular arrhythmias (114). There have been some studies applying these tests to patients with syncope (114,115). On the basis of these

small studies the signal averaged electrocardiogram may be a sensitive and specific noninvasive test for detecting a high risk subset of patients with syncope prone to ventricular tachyarrhythmias. Further information regarding the efficacy of this test should be available in the literature in the near future.

Neurologic Tests

Neurologic tests undertaken in the evaluation of the patient with syncope frequently include electroencephalograms and CT scans. The diagnostic impact of both of these tests is closely related to the suspicion of neurologic disease based on the history and physical exam. For example in Day's study of emergency room patients, sixty-three electroencephalograms were performed (5). Twenty-six of these were performed in patients who were not thought to have a central nervous system cause for transient loss of consciousness. Three (12%) of these electroencephalograms were abnormal. However, studies of electroencephalograms in normal individuals have revealed up to fourteen percent being abnormal. Of the thirty-seven patients in Day's study in whom a central nervous system diagnosis was likely, fifteen had an abnormal electroencephalogram for a yield of forty-one percent. A summary of the contribution of various investations to the final diagnosis in two large studies is outlined below in Table 9.

Table 9.

Yield of Diagnostic Studies

Study	No. of Patients (%) Kapoor, et.al.(8)	Day, et.al.(5)
History and Physical	52 (49)	147 (85)
Serum Electrolytes		1 (0.6)
Electrocardiogram	12 (7.4)	4 (2.3)
Electrocardiographic Monitoring	29 (17.8)	6 (3.5)
Cardiac Catheteterization	7 (4)	
Electrophysiology	3 (1.7)	
Electroencephalography	1 (0.6)	3 (1.7)
CT Scan		4 (2.3)
Cerebral Angiography	2 (1.2)	

CT scans did not reveal any abnormalities in seventeen patients in Day's study who did not have focal neurologic findings or seizures. In seven of twenty patients who had evidence of neurologic disease by history and physical exam the CT scans demonstrated important new abnormalities. Sophisticated neurologic testing is only indicated in individuals in whom the history and physical exam suggests a central nervous system etiology of their syncope.

THERAPEUTIC CONSIDERATIONS

Syncope is a symptom having many etiologies and therapy is aimed at the underlying problem. The goal of this therapy is to optimally treat that problem to prevent episodes of syncope and subsequent mortality and morbidity. Most people with vasovagal syncope, a relatively benign etiology, can be reassured and counselled in methods to avoid syncope in the future. This would include assuming a supine position when presyncopal symptoms occur to avoid loss of consciousness to due to decreased cerebral blood flow.

In patients found to have orthostatic hypotension as a cause of syncope, the etiology of the orthostatic hypotension should be treated if at all possible. This would include volume repletion in patients who are volume depleted, adjusting medications in drug related orthostatic hypotension, and symptomatic precautions in people who have neuropathies or idiopathic orthostatic hypotension.

The bradycardia associated with carotid sinus syncope may require pacemaker implantation to prevent further episodes of syncope. The vasodepressor component, if a significant factor, may respond to vasoconstricting drugs (18).

Pacemakers in general are indicated when heart block or symptomatic bradycardia or sinus pauses are demonstrated by monitoring in a patient who presents with syncope. Pacemakers do prolong survival of patients with complete heart block (82). However, it is important to carefully evaluate patients suspected to have heart block to exclude other causes of syncope. Even patients with bifasciular block on electrocardiogram are more likely to have syncope because of factors other than heart block (96). After pacemaker implantation, patients with sick sinus syndrome may continue to experience dizziness related to concurrent conditions such as ischemic heart disease or congestive heart failure (116).

Antiarrhythmic therapy is not indicated for patients with syncope until a cardiac etiology is established by correlating symptoms with dysarrthymias demonstrated by monitoring. It is difficult to interpret empiric trials of antiarrhythmic therapy. In addition the potential side effects of antiarrhythmic drugs are great. When dysarrhythmias are determined to be the cause of syncope, treatment should be instituted under controlled circumstances allowing selection of the empirically determined optimal antiarrhythmic therapy for each patient.

PROGNOSIS

Soma Weiss once referred to sudden death as "irreversible syncope" (117). One of the major goals in evaluating and treating episodes of syncope is to identify serious underlying disease which may predispose to sudden death and in which that outcome may be prevented by appropriate therapy.

Patients with cardiac causes of syncope are at highest risk for subsequent death or significant morbidity related to their cause of syncope. In various series the mortality rate in these patients varies from eighteen to thirty-three percent (5,9,85-87). Nearly eighty percent of these deaths occur suddenly (6). Because of this significant mortality, patients in whom a cardiovascular cause is suspected usually require admission to the hospital to undergo appropriate tests to confirm the diagnosis and begin treatment.

A medium risk group of patients was identified in Day's study (5). These included patients over age 70 years with vasovagal, psychogenic, or unknown causes of syncope and patients over the age of 30 with a central nervous system or drug/metabolic cause for syncope (5,87). Of patients in this category, seventeen percent had a poor outcome at one year which included mortality and significant morbidity. Kapoor defined a category with an intermediate risk of outcome as all patients with non cardiovascular etiologies of syncope (9). The one year mortality rate of patients with this category was twelve percent. Similarly, Eagle studied a large group of patients at Yale and found only one percent poor outcome of lll patients in this category (87).

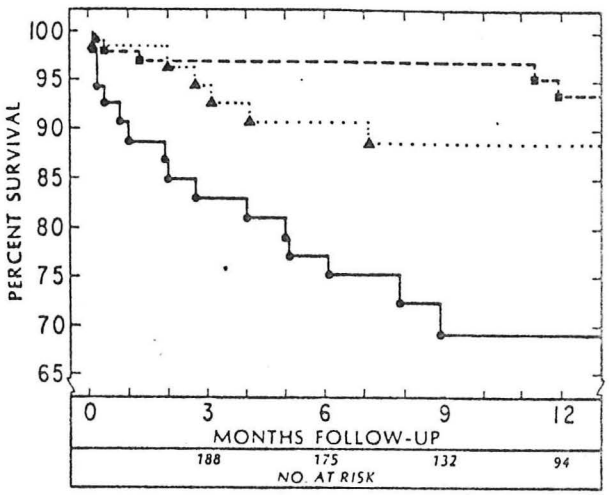
Table l0.

Poor Outcome* Related to Syncope Using Criteria of Day et. al.

	Yale Patients		*Brigham Patients*		
	Number	*Poor Outcome at One Year*	*Number*	*Poor Outcome at one Year*	
Low Risk	lll	l%	ll4	l%	p=NS
Medium Risk	50	6%	59	l7%	p=0.07
High Risk	l5	30%	l2	20%	p=NS

**Mortality or major subsequent morbidity from the cause of the syncope.*

Kapoor's study had a large group of patients in whom the etiology of syncope could not be defined (9). This made up 97 patients, 47% of his study group. Of the 97 patients in this category six percent had died within one year.



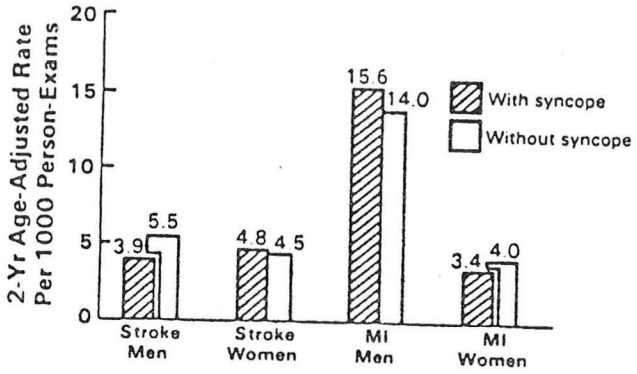
The Yale study identified 113 patients with syncope of unknown origin (87). Again, six percent of these patients died within a year. Three of these patients in Eagle's study were over the age of 70 and died of cerebrovascular disease.

Table II.

Mortality Using Criteria of Kapoor et. al.					
	Yale Patients		*Pittsburgh Patients*		
Diagnostic Category	*Number*	*One-Year Mortality Rate*	*Number*	*One-Year Mortality Rate*	
Cardiovascular	13	21%	53	30%	p=NS
Noncardiovascular	50	0%	54	12%	p=<0.01
Unknown	113	6%	97	6%	p=NS

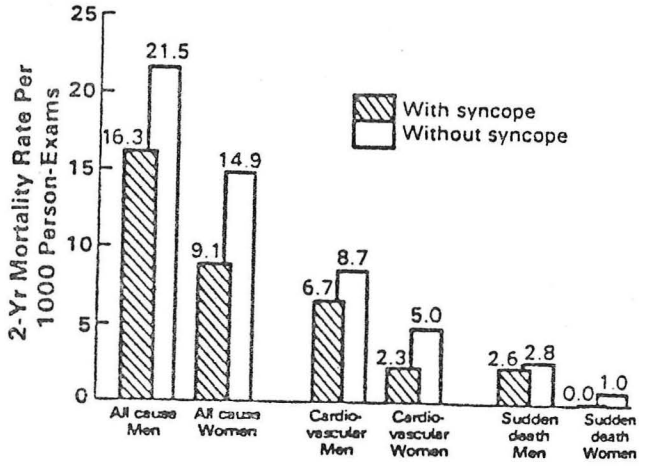
The Framingham study identified a group of patients with isolated syncope who had transient loss of consciousness in the absence of prior or concurrent neurologic, coronary, or other cardiovascular disease (7). During twenty-six years of follow up, this group of patients had no substantial or statistically significant difference in stroke or myocardial infarction rate compared to those who did not have syncope.

Figure 6.



In addition, age group and sex specific comparisons revealed no significant difference in mortality rate for all causes, cardiovascular causes, and sudden death between those with and without isolated syncope.

Figure 7.



Kapoor looked at the prognostic implications of recurrences of syncope in their group of 433 patients (118). Over a mean follow up of thirty months, 146 patients had recurrent syncope. Patients with an initial diagnosis of a cardiovascular cause of syncope had a recurrence rate of thirty-one percent, patients with noncardiovascular had a recurrence rate of thirty-six percent,

and patients with syncope of unknown origin had a recurrence rate of forty-three percent. In eight of 191 patients in whom a cause of syncope could not be found on an initial evaluation, a diagnosis was found in follow up after recurrent syncope.

Recurrent syncope lead to major morbidity in eight of 146 patients (five percent) and minor trauma in ten patients (seven percent). They found that recurrences of syncope were not a significant predictor of overall mortality or sudden death. In addition, they found that new diagnoses were rarely established on the basis of evaluation of recurrences of syncope.

SUMMARY

Syncope is a common symptom with diverse etiologies. The diagnoses underlying syncope may vary from a benign vasovagal faint for which the patient can be reassured to an underlying cardiac arrhythmia which carries a mortality rate of 33% in one year. The major component of the evaluation of syncope is a thoughtful history and physical exam keeping in mind all the possible etiologies of syncope.

Further evaluation of syncope can be difficult and costly. The tests performed should be carefully chosen based on the clinical suspicions for certain diseases from data collected in the history and physical exam. Patients in whom a cardiovascular etiology is suspected should be aggressively evaluated and treated.

The goals of the therapy of the clinical problems causing syncope are to prevent further episodes of syncope and to reduce the morbidity and mortality associated with that disease. The majority of patients who present with syncope do well. However, subsets of patients can be identified who are at major risk. These patients should be aggressively evaluated and treated as indicated.

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