Cardiac and neurogenic syndrome

Donald D. Purvis
University of Nebraska Medical Center

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CARDIAC AND NEUROGENIC SYNCOPE

DONALD F. PURVIS

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INTRODUCTION

There is not a more confusing and yet a more exacting situation than that which arises when a patient is brought to your attention with the one outstanding complaint of syncope. The sudden events which affect the integrity of the brain and heart strike at the very foundation of life itself and are necessarily associated with grave concern and apprehension by the patients, the relatives, and probably more so yourself. The only answer to a patient of this category is a rapid accurate diagnosis and immediate treatment to relieve the patient psychologically and physically in an effort to prevent future catastrophies.

In looking over the subject material of neurogenic syncope, it becomes quite evident that the subject is, as a whole, relatively new. Some types of syncope have been described centuries long past, but the majority of the material have appeared in the last two or three decades, some of it entirely new, some old, but with an entirely different interpretation. There still exists considerable confusion which is indicated by the numerous classifications advanced on the subject and the treatment as suggested in many cases is questionable.

It is impossible to cover the subject material on each syndrome or clinical manifestation. That is not the
purpose. The desire here is merely to simplify a few of the more common conditions causing syncope and to place them in an understandable category with the recognized correct symptomatology and treatment so that they may be recognized easily. With this in mind an attempt will be made to bring a little order out of the confusion as far as is possible.
ANATOMY AND PHYSIOLOGY

In the following discussion is a summary of the principle nervous mechanisms, reflexes, and actions accorded to said structures. It is necessary to have in mind the anatomy of the region in order to understand the physiological mechanisms that occur during an attack of syncope of the various types.

The nervous mechanism and control of the heart may be divided into the intrinsic and extrinsic groups. The intrinsic group includes the sinoauricular node, the atrioventricular node and the bundle of His. These structures show no definite nerve connections and do not present histologically true nervous tissue; but, instead, are specialized bundles of muscle tissue capable of exciting a rhythmical cardiac beat, and transmitting this impulse throughout the heart muscle making the heart independent of nerve elements if the need ever arose. This condition exists in embryonic life, in which the heart starts beating before the nerve connections ever establish connections.

The extrinsic nervous mechanism and control of the heart has its chief centers in a group of nerve cells located in the medulla; these are known as the cardiac centers. There are various afferent pathways along which impulses are transmitted from the heart from various re-
gions of the body, and efferent pathways which transmit impulses from the centers to the heart.

PARASYMPATHETIC SYSTEM.

Anatomically the heart is supplied extrinsically by the vagus (Xth cranial nerve) motor fibers. Its superficial origin is by 8-10 filaments from the groove between the olivary and restiform body below the glossopharyngeal; its deep origin may be traced through the fasciculi of the medulla to terminate in the nucleus of the grey matter, the nucleus vagi, at the lower part of the 4th ventricle beneath the ala cinerea below and continuous with the nucleus of origin of the glossopharyngeal. In addition a few fibers pass into the funiculus solitarius and others pass into the accessory vagal nucleus. The filaments become united and form a flat cord which passes out the jugular foramen. The nerve passes vertically down the neck, within the sheath of the carotid vessels, lying between the internal carotid artery and internal jugular vein as far as the thyroid cartilage. Here the paths of the nerves, on each side, assume a different course to form the cardiac plexus in which we are so much interested.

The cervical cardiac branches--two to three in number arise from the pneumogastric at the upper and lower part of the neck. The superior branches are communicative with
the cardiac branches of the sympathetic and can be traced to the great or deep cardiac plexus. The inferior branch-
es, one on each side, arise at the lower part of the neck. On the right side, this branch passes in front of the in-
nominate artery and communicates with one of the cardiac nerves proceeding to the great or deep cardiac plexus. On the left side, it passes in front of the arch of the aorta and joins the superficial cardiac plexus.

The thoracic cardiac branches--on the right side, arise from the trunk of the vagus as it lies by the side of the trachea and from its recurrent laryngeal branch, but on the left side from the recurrent nerve only, passing inward, they terminate in the deep cardiac plexus. Gray (26)

ACTIVITY OF THE PARASYMPATHETIC SYSTEM.

The vagus nerves are cardio-inhibitory as discovered by the Wever Brothers in 1845. They carry parasympathetic fibers from the cardio-inhibitory center in the medulla to the heart. Fibers derived from the right nerve terminate around ganglion cells in auricular tissue about the sino-auricular node. These cells serve as relay stations in the transmission of the vagal influence; their axons enter the node and are disposed in a plexus about muscle cells in the nodal tissues. The left nerve establishes similar relationships with the auriculoventricular node. Its terminations arborize around ganglion cells in the
interauricular septum which sends axons to the muscular elements of the nodal tissue. Each node, however, receives filaments from the opposite nerve.

Stimulation of the cardiac vagus causes pronounced slowing or complete stoppage of the heart with a consequent blood pressure drop due to a lengthening of the diastolic cycle. The heart will escape from under this inhibitory influence by setting up a ventricular beat after a period of asystole. The vagii have no direct action upon ventricular muscle, but the auricular effects are slowing and weakening of the auricular beat and shortening of the refractory period. The slowing or stopping of the ventricular beat is an indirect effect due to auricular slowing or arrest of auricular contractions or to arrest in conduction in auriculoventricular connections. In the latter instance, partial or complete heart block is induced. When the heart rate returns, it is due to impulses being initiated in regions below the lower part of the S-A node or A-V node or other parts of special tissue possessing lower rhythmical powers.

In stimulating the right vagus the results are slowing and weakening of the auricular beat and eventually a reduction in ventricular rate. Excitation of the left nerve which ends in the A-V node causes ventricular slowing by depressing A-V conduction and blocking auricular impulses, Best and Taylor (3).
SYMPATHETIC SYSTEM.

The sympathetic supply of the heart is made up in the following manner: the superior cardiac nerve arises by two or more branches from the superior cervical ganglion. It runs down the neck behind the common carotid artery and crosses in front of the inferior thyroid artery and recurrent nerve. The course from here differs on the two sides. The right nerve, at the root of the neck, passes before or behind the subclavian artery, back of the arch of the aorta joining the deep part of the cardiac plexus. The left nerve runs in front of the left common carotid artery and across to the left side of the aortic arch to the superficial part of the cardiac plexus.

The middle cardiac nerve, the largest, arises from the middle cervical ganglion. On the right side it follows the course of the superior cardiac nerve to its conclusion, but on the left side the middle cardiac nerve joins the left half of the deep part of the cardiac plexus.

The inferior cardiac nerve arises from either the lower cervical ganglion or first thoracic ganglion. It passes down to join the deep part of the cardiac plexus.

Grav (26)

ACTIVITY OF THE SYMPATHETIC SYSTEM.

The distribution of these nerves is similar to that of the vagus. The post ganglionic fibers on the right
side are distributed to the sinoauricular node, those on the left to the A-V node and bundle. These cells are a spinal cardioaccelerator center probably under the influence of higher centers probably the hypothalamus and medulla.

Stimulation of the accelerators causes a quickening in rate of both auricles and ventricles and an increase in contraction force. Thus, the accelerator fibers act directly on the ventricles, whereas, the inhibitory fibers act only indirectly on the ventricles. Ventricular fibrillation may be produced by stimulation of the accelerator fibers similar to the effect of adrenaline. Best and Taylor (3)

**AFFERENT CARDIAC SUPPLY.**

The afferent nerve endings in the heart and aorta are contained in the cardiac vagus itself. The receptors of these lie within the heart tissue and upon the aortic arch. Therefore, stimulation of the central end of a vagus will alter the cardiac rate. As a general rule, stimulation of peripheral nerves causes an acceleration of the afferent vagus inhibition. The latter depends on type and intensity of stimulus. There is an vagal afferent branch which terminates in the wall of the aortic arch and the heart itself. It is known as the aortic or cardiac depressor nerve because it produces cardiac slowing
and vasodilatation with a fall in blood pressure. Best and Taylor (3)

**CARDIAC REFLEXES.**

The cardiac reflexes are based on the activity of the cardio-accelerator and cardio-inhibitory centers which send continuous discharges of impulses along cardiac nerves. The maintenance of the tone of the centers, and so, of the resting rate of the heart and the alterations in rate which occur are either due to reflexes or to cerebral center impulses. Impulses may arise all over the body which may either exact or depress the cardiac rate.

The reciprocal actions of the cardiac centers produces heart rate changes as a result of variations in the tone of both centers. This is a reciprocal variation characterized by a decreased cardio-inhibitory center tone with an increased cardio-accelerator center tone or vice versa. This mechanism makes for a more rapid, smoother, and nicer adjustment of cardiac rate than would be possible otherwise. It has been found that the vagus or cardio-inhibitor element holds the more effective tone of the two systems. Best and Taylor (3)

**VASCULAR REFLEXES.**

**Vagopressor reflexes.** The pressor fibers of the vagus are stimulated by a fall in the pressure of the blood in the great veins feeding the heart. A fall in venous pressure
exerts an influence on the afferent vagal endings in the right auricle. As a result of this mechanism generalized vasoconstriction occurs as a compensatory mechanism. Best and Taylor (3)

The aortic or cardiac depressor nerves are purely afferent depressor nerves and when stimulated they produce vasodilatation and a decreased heart rate. The afferent is probably of vagal derivation, the efferents constitute the vagal fibers and the vasomotor pathways. The vasoconstrictor center is reduced and the vasodilator tone increased. The receptors for this reflex lie in the aortic wall proper. Best and Taylor (3)

The carotid sinus mechanism is a slight enlargement of the common carotid artery where it bifurcates into the internal and external carotid arteries. The carotid sinus was shown by Hering (29) in 1923 to play an important role in cardiac rate regulation and arterial blood pressure. Compression at the carotid junction (so as to raise the pressure within the sinus) caused a marked slowing of the heart rate, vasodilatation and a fall in blood pressure. Pressure on the common carotid, some distance below the sinus (so as to reduce intrasinal) pressure, causes cardiac acceleration, vasoconstriction, and a rise in arterial pressure together with the liberation of adrenaline.
The afferent fibers of this reflex arc are contained in the sinus nerve, a glossopharyngeal branch. The sinus nerve descends between the internal and external carotids to the sinus where its fibers terminate in sensory organs situated in the connective tissue fibers located in the adventitia of the sinus wall. Fibers also ramify the carotid body, a small structure situated behind the fork of the common carotid. Centrally the fibers of the sinus nerve make connections with the cardio-inhibitory and vasomotor centers. The efferent limb is the vagus. A nerve twig connects the sinus with the ganglion of the vagus; it also receives a filament from the superior cervical ganglion of the sympathetic.

The sinus and aortic nerves constitute a mechanism of utmost importance in controlling arterial blood supply and maintaining the circulation to the brain. Normally a rise in diastolic pressure and in the heart rate occurs when the body changes from the recumbent to the sitting position or from sitting to standing position; they therefore play an essential part in compensating for the effect of gravity upon the circulation.

Any increase in the tension exerted on the proprioceptors in the sinus or aortic wall causes a rise in afferent impulse frequency with consequent slowing of the heart and vasodilatation. A decreased tension causes the
reversed effect by lowering the tonic depressor effect. Best and Taylor (3).

**CONCLUSION.**

The pathological manifestations which are discussed further in this paper are based primarily on the nervous mechanisms just discussed. The activity of the vagus and glossopharyngeal as against the activity of the sympathetic system must be understood in order to correlate the reciprocal actions of vasodilatation and vasoconstriction. Furthermore the mechanism of the carotid sinus becomes more apparent at the pathological conditions involving this body as they occur.
The problem of syncope is extremely involved and it is my purpose to limit the discussion of syncopal attacks to those based on a cardiac or neurogenic origin. Several classifications have been advanced by various authors, but many are entirely too involved and massive. The fundamental purpose in this discussion is understanding and brevity. Included will be the etiology, symptomatology, physiopathology diagnosis and treatment of the various disorders found in clinical practice.

The following is a classification of neurogenic and cardiac syncope which include the most important syndromes.

1. **Extrinsic types**.
   1. Vasovagal reflex. (simple syncope)
   2. Vagovagal reflex.
   3. Postural hypotension and syncope.
   5. Nothnagel's syndrome.
   6. Carotid sinus syndrome.
      a. Vagal.
      b. Depressor.
      c. Cerebral.

II. **Intrinsic types**.
1. Adams-Stokes syndrome. (heartblock)
2. Abnormal cardiac rhythms.
   a. Paroxysmal tachycardia.
   b. Auricular flutter.
   c. Auricular fibrillation
   d. Ventricular flutter, fibrillation and standstill.
ETRINSIC SYNCOPE
VASOVAGAL REFLEX

DEFINITION.--"Vasovagal syncope is an acute and usually transient bodily state characterized primarily by a sudden and unexpected, partial or complete, suspension of the functions of locomotion, consciousness, and often of circulation and respiration." (68) This definition might be used to include the relative aspects of almost all syncopal attacks.

ETIOLOGY.--This is one of the most common types of syncope seen in clinical practice. The term was given to this syndrome by Lewis (36), although Gowers (24) used the term as early as 1907 designating an entirely different type of transient attack. The attacks occur in both sexes particularly at an early adult age. Predisposing factors of this manifestation are due to an unstable sympathetic-parasympathetic balance which, according to Parker (40), may be brought about in tall, rapidly growing adolescents who are suddenly subjected to such factors as pain, hunger, poor ventilation, and emotional disturbances. He believes these symptoms disappear as the patient grows older and increases in physical capacity. In the 18th and 19th centuries many essays were written on the subject, but until the disordered action of the heart was analyzed there were no differential characteristics between vasovagal and any other types.

-14-
Lewis (36) states that many of the first cases studied were in young soldiers invalided into hospitals for a condition known as an "effort syndrome." This was the state of ill health which quickly responded to exercise with syncope. He also found the condition existed in civilian population as well as those who were troubled with fatigue, fasting problems, overheated or overcrowded rooms and in certain true cardiac pathological conditions.

Lewis (36) stated a typical case which is presented here: A soldier, age 21, was invalided from active service. He had been unable to play strenuous games since childhood owing to breathlessness and an early onset of exhaustion. He was invalided for these symptoms and because he had fainted while on guard duty when attempting to salute an officer of high rank of whom he became abruptly aware. Physically he was undersized and poorly developed man presenting no clear signs of heart disease or other organic pathology. While he was sitting watching blood being withdrawn from his arm he complained of dizziness, queerness, and facial pallor. His head fell forward to his knees. By that time the pallor was intense, the pulse imperceptible, heart sounds distant, and regular bradycardia. The patient was limp and mentally confused and actually unconscious for several minutes. A heavy sweat broke out over the body and head, the pallor re-
mained extreme, respirations were slow and cyanotic, systolic blood pressure was 60-55, the pulse rate varied between 50-60. Nine minutes after the onset the patient was able to respond and shortly thereafter to get up and walk about. He was tired and nervous for 36 hours thereafter. His average blood pressure was 118, pulse rate 80-90.

The above case is a typical type of vasovagal syncope.

The majority of the fainting attacks in this category are due to prolonged standing, particularly in persons with poor muscular tonus, poor physical health, chronic undernutrition, anemia and similar circumstances. Fatigue, fasting, mental worry or conflict, emotional tension, straining during defecation or the administration of an enema often cause the collapse. A hot bath or hot shower may bring on an attack as does gastrointestinal disturbances, pregnancy, vomiting, mountain sickness, and instrumentation of various types. In other words, this type may be found in all phases of life and under all conditions.

Physiologically the attack is brought about by a diminished blood return to the heart probably due to a vasodilatation in the splanchnic and constriction in the peripheral areas causing a pooling of blood with a resultant cerebral ischemia. In these afflicted individuals the vasomotor system under control of sympathetic and
parasympathetic balances is physiologically unstable and the delicate balance is easily upset between these reciprocal elements. Proof of this mechanism is suggested by Blitz (6) who reported a case of a patient with marked generalized urticaria who suddenly fainted. The urticaria disappeared during the syncopal attack and reappeared a short time after recovery. He concludes that splanchnic dilatation occurred with peripheral vasoconstriction. Thus, an interruption of the blood supply to the skin occurred destroying the wheals which are formed by a local dilatation of capillaries, arterioles, and venules and increased permeability of vessel walls.

Vasovagal syncope occurs also in patients with organic disease. Some of the more common causes are infectious diseases, mitral and aortic stenosis, congenital heart disease, arterial hypertension and certain other pathological manifestations. Certain vasodilator substances, such as the nitrites, will cause a syncope identical in every respect to vasovagal syncope. Therefore, it must be assumed that spinal anesthesias will produce similar attacks in certain instances.

Shillito (53) discovered that in treating one of his cases with asthma, by using the insulin-glucose method, a vasovagal attack resulted. Because of this, he believed that some connection existed between hypoglycemia and
vasovagal syncope. Shillito suggested the existence of a relationship between chronic hypoglycemia and convulsive attacks. These attacks were preventable by glucose administration.

Gumpert (25) advances the rather rabid belief that all syncopal attacks are vasovagal in origin except the attacks due to postural hypotension and heart block.

Silberberg (54) concluded that the innocent or benign types of vasovagal syncope were entirely dependent on the disturbance of the nervous mechanism, a disharmony between the sympathetic and parasympathetic systems. The disturbance, he states, "acts through the vagal mechanism and vasomotor systems producing a cerebral anemia." This might be brought about in various ways such as the overactivity of the vagus and vasomotor pooling of blood in the large abdominal vessels.

SYMPTOMS.—The clinical manifestations may show considerable variation, depending on the rate of onset of the attack. In the severe type, the patient collapses instantly without warning, the body lies crumpled and motionless. The face and body surfaces are ghastly pale. The pupils are dilated and the corneal reflex is absent. Respiratory movements are quite variable, but the heart sounds are very distant and faint. Naturally the pulse is practically imperceptible giving rise to the extreme alarm felt
by relatives and often times by the doctor. If syncope endures slow long enough, clonic convulsions are to be seen over the facial region and other parts of the body. Weiss (68) makes a startling statement, "there is no other condition including the deepest coma, which so closely resembles death. No wonder that a simple, benign syncope is often described as 'an attack in which the patient almost died,' and, contrariwise, that some cases of instantaneous death are believed at first to be but fainting." The duration of unconsciousness varies from a few seconds to several minutes, but the rate of recovery is much slower than the onset. Gradually muscular power and coordination return, but, oftentimes, the patient lies weak and limp for one-half to one hour. Headache, anorexia, and weakness may last from twelve to twenty-four hours or longer.

In those milder attacks which come on gradually there are usually some important premonitory signs, such as, epigastric or precordial distress, giddiness, and light-headedness. Many experience a rather sudden dimming of vision and a throbbing headache. Yawning, belching, abdominal cramps and periodic waves of nausea and vomiting occur. The remarkably consistent reports show a progressive lowering of blood pressure, decrease in the heart rate, and a small imperceptible pulse with increasing pallor just pre-
cedes the actual syncopal attack. Muscular relaxation immediately precedes the syncopal attack and sounds recede in the distance just before unconsciousness. Patients oftentimes experience a sensation of coldness and numbness starting at the lips and extremities moving toward the heart just as syncope overtakes them.

The usual after effects of a vasovagal attack are chiefly headache, weakness, and trembling; increased perspiration, nausea, and vomiting. These effects are quite brief in duration after the more mild attacks lasting only from one-half to two minutes.

One point that is a definite signal toward a vasovagal attack is the fact that syncope of vasovagal origin never occurs while the patient is lying down--the patient must be either sitting or standing. Richardson (45) in 1854 suggested that fainting is a mechanical reflex capable of supplying more blood to the heart and finally to the brain. He discovered that if a phlebotomy were done on a patient in the erect position syncope would soon result due to the brain anoxemia; therefore, he concluded that if a body were placed in the horizontal position the mechanical factors of the reflex would be abolished.

It is commonly believed that vasovagal syncope is a benign manifestation, and it usually is, but there are
certain cases in which a fatal outcome is produced. Such is the case in sudden deaths occurring in elderly patients with coronary arteriosclerosis who collapse on getting out of bed after a prolonged rest period. The combination of coronary arteriosclerotic vagal slowing and a weakened vasomotor system results in severe cerebral anoxemia without recovery. Some patients who have been sitting in a wheelchair or armchair after a prolonged rest "pass out" because the body is unable to assume a horizontal position which is so vital to cerebral recovery. As early as 1740 Hoffman (68) described patients suffering from otherwise benign diseases who developed a fatal collapse on standing.

**PHYSIOPATHOLOGY.**—This condition of vasovagal syncope is best understood after visualizing the nervous mechanisms associated with the heart and vasomotor system. The vagus, with its inhibitory powers on the heart, exerts an abnormal tone through the superficial and deep cardiac plexuses; thereby slowing the rate. The afferent stimuli for this effect are brought about as distance, position, aural, visual, and emotional impressions on unstable vasomotor mechanism. Proof of the fact, according to Lewis (36) that the heart rate decrease is due to vagal slowing is evidenced on the administration of atropine. An immediate increase in the heart rate occurs as the atropine destroys vagal inhibition, but the associated vascular factors of
vasodilatation with a marked blood pressure drop do not respond to atropine and syncope results in spite of vagal elimination. The vasodilator center probably through the aortic or cardiac depressor nerve stimulates the vasodilator center in the medulla producing the splanchnic pooling with the associated drop in blood pressure.

Weiss (68) noted there always occurred a lowered systolic pressure and an emptiness of the superficial veins of the neck and skin indicating a lack of return blood flow. Cardiac output is actually decreased during syncope, because of the decreased systolic and rising diastolic pressures which resulted in a low pulse pressure. This is not a definitely proved concept as yet and much work is being carried out in an effort to discover the exact nervous mechanisms and physiological changes produced in these patients.

Thus, it must be assumed that in the mechanism of vasovagal syncope the most significant and most constant change is the progressive decrease in pulse pressure caused by a slowly rising diastolic and decreasing systolic pressures. The vasomotor center exerting an increased vasodilator tone causes a stagnation of blood in the splanchnic region. Simultaneously with these changes, there is a decreased venous return of blood. At first the heart rate is oftentimes elevated, but as the pulse
pressure becomes less and less syncope occurs with a sudden appearance of vagal inhibition on the cardiac muscle. In other words, a number of functional changes occur, all of which, are not, as yet, understood, but it must be unquestioned that the vagal inhibitory center is extremely susceptible to sudden cerebral anoxemia secondary to a vasomotor collapse.

DIAGNOSIS.--This syndrome is easily diagnosed when a careful history has been elicited! Usually by the time the doctor arrives the patient has recovered sufficiently to give an adequate description of preceding occurrences, premonitory signs and symptoms, the attack and recovery. Any upsetting situation, prolonged standing, visual or aural impressions and shock, are frequently found as precursors to the situation.

If one is called, during an attack, the picture of extreme pallor, imperceptible pulse, low blood pressure, slow heart rate, perspiration, and a cold and clammy feeling of the skin all point to the diagnosis of a vasovagal attack.

According to Gumpert (25), who was fortunate enough to secure EKG studies during an attack, the following changes were found: No P wave was discernible and the RT segment is longer than normal. Since the P wave is lost, it must be buried in the ventricular complex and the impulse thus, must have arisen low down in the
AV node producing a retrograde spread through the auricle. This information is practically without value, because the attack is usually too brief. It is of value from the academic point of view, but the combination of history, symptoms, signs, and physical findings is much more accurate.

**TREATMENT.**--The treatment of vasovagal syncope is comparatively simple. Richardson (45) found the horizontal position was nature's way of caring for the attacks, consequently he believed it was sufficient to lay the body in a horizontal line and leave it alone.

Effective preventive measures depend entirely on the predisposing causes in the individual cases. If emotional factors are the basis of such attacks proper psychotherapy should be instituted to overcome weaknesses according to Fantus (14), Ferris (15), and Weiss (68). The vasomotor system instability is the chief causative factor in most instances and the outline of prophylaxis as advocated by Ferris (15) is quite adequate to cover most situations. He suggests, 1. proper psychotherapy, as mentioned previously, 2. routine morning and physical exercises, 3. hydrotherapy to improve the vasomotor tone, 4. the use of abdominal binders to prevent abdominal congestion, 5. proper breathing habits, using thoracic musculature to an advantage, and 6. cor-
rective postural conditions including the periodic shifting from one foot to the other when standing for a comparatively lengthy period. Fantus (14) suggests anemia as a common associated condition and advises antianemic therapy. This view is supported strongly by Weiss (58).

Active treatment of vasovagal syncopal attacks resolves itself into a few important considerations: 1. The patient should be placed at once in a horizontal position with the head lowered, 2. The clothing should be loosened to insure adequate breathing, 3. Reflex stimulation by use of cold towels or ice to the forehead and face, 4. Olfactory stimulants such as spirits of ammonia, 5. Massage of legs and abdomen, and 6. 1 cc. of 1:10,000 adrenaline solution intravenously as an immediate beneficial therapeutic agent. In extreme cases artificial respiration and cardiac massage may be an indicated procedure. Oxygen therapy is used by many clinicians and is of doubtless value in many of these individuals.

For post syncopal conditions analgesics may be used for headaches. The patient should be maintained in the horizontal position for a period of at least one-half to two hours after a recovery from the syncopal attack to insure stabilization.

PROGNOSIS.--The prognosis is excellent in regard to the syncopal attacks proper, but recurrences of such conditions
is usually the case. Treatment, if carried out vigorously may tend to prevent the frequency of the same, but no assurances can be given the patient as to positive recovery.

VAGO-VAGAL SYNCOPE

**DEFINITION.**--This syndrome may be defined as a syncopal attack of reflex origin which has the entire reflex in the vagal nerve afferent and efferent pathways. Many clinicians consider this as the Adams-Stokes type of syndrome on a reflex origin. It is not as common by far as the vasovagal type of syncope.

**ETIOLOGY.**--The causative agent in this syndrome is the stimulated vagus nerve, which acts reflexly on the heart producing the typical attacks. Because of the wide distribution of the sensory fibers of the vagus, the chances are that in some pathological condition these will be irritated, "setting the spark to ignite the reflex chain."

The chief sensory endings of the xth cranial nerve related to cardiac regulation are those existing within the heart tissue and aortic arch proper. Coexisting afferent pathways also supply the other mediastinal and thoracic contents as well as abdominal viscera. As one can see the distribution of the vagus is protean, and consequently, is it of no wonder that sometimes a rather
aberrant stimulus should cross the reflex arc?

Best and Taylor (3) state the aortic or cardiac depressor nerve was first discovered in 1866. Stimulation of this aortic or cardiac depressor nerve produced vasodilatation and cardiac slowing. The stimulus then passes to the cerebral cortex and out by way of the vagal efferent fibers which supply the heart as the superficial and deep cardiac plexuses. These fibers terminate about the sinoauricular and atrioventricular nodes. Thus, they are closely associated with cardiac function and rhythmicity.

The end result of such a stimulation is not always the same. In some instances there may exist, 1. a slowing of the heart, 2. sinoauricular block, 3. partial or complete heart block, or 4. in total cardiac standstill. Because of these varied effects, this syndrome is often referred to as the Adams-Stokes' attack of reflex origin.

Common etiological factors, according to Weiss (68), which produce vagal stimulation are inflamed tonsils, esophageal diverticulations, carcinomatous growths of the mediastinum, or mechanical irritation of the nerves of the bronchial mucosa. Visceral factors may be gastric distension, the effect of drinking cold fluids in the overheated or fatigued state as seen in mountain climbing, threshing "spike pitchers" and the like. Some of the
drowning cases following ingestion of a heavy meal may be vago-vagal in origin, but the vasovagal influence cannot be disregarded.

Two cases which will be presented, demonstrate beautifully the etiological factors that may produce a vago-vagal attack. Weiss, Ferris and Capps (7) reported a case of a white male, age 64, who was admitted to the hospital in a stuporous state. He attempted to take his life with gas, because he had been troubled with fainting spells for ten years. These attacks manifest themselves by sudden dizziness and fainting lasting one to two minutes, usually precipitated by swallowing food, particularly sticky food. Sometimes water would cause this condition. The dizziness and fainting were associated with pains along the lower part of the sternum, descending toward the stomach. X-Ray examination showed a small diverticulation in the esophagus at the level of the fourth costochondral junction. The physical examination was essentially normal except for a slight systolic murmur. The pulse was normal, except for being slowed to about 48 per minute. The arterial pressure varied from 94-140 mms. systolic and 56-80 mms. diastolic.

Spontaneous attacks were produced with the following picture of pallor, hoarseness, decreased heartrate and unconsciousness. Induced attacks were produced by means
of inflating the esophagus. The EKG showed complete AV dissociation.

BaCl₂ was administered, but had no effect in stopping the attacks; epinephrine caused a cessation of the attacks, but the EKG showed complete AV dissociation with the ventricle taking up the ectopic beat. Ephedrine and atropine also abolished the attacks, but did not stop the AV dissociation.

The conclusions derived from this case were that the vagus sensory nerves were stimulated. This was proved by novocainizing the vagus in the neck; and thus, stopping the attacks. All other reflexes were normal in this patient. Simple sinus slowing then, was due to vagal stimulation producing a vagal slowing.

Iglauer and Schwartz (33) reported the second case, herein presented, of a woman, age 55, who came to them complaining of occasional fainting spells while partaking of food. This began at 9 years of age which ceased at 14 for a time. A few years later she experienced difficulty in deglutition and would have a sense of weight and fullness under the sternum while partaking of food. X-Ray showed a spastic esophagus. Five years later she began having peculiar fainting spells occurring usually while taking food or water. The attacks were sometimes preceded by a full sensation in the pit of the stomach ascending.
toward the throat. The patient's associates noticed that her face would flush and then grow pale at the time of the attack. The duration of the faintness was momentary. On several occasions, she noticed that her pulse had stopped and then began to beat slowly and intermittently. She had been taking ephedrine gr. ¼ before each meal for a period of four months and was almost free of syncopal attacks. During the past year she had lost considerable weight, (24 Pounds).

Physical examination showed nothing. The vascular system examination was normal. Pressure on the right carotid sinus caused a slowing of the pulse from 70-40; pressure on the left carotid sinus reduced the pulse from 76-60. X-Ray presented a dilated esophagus with cardiospasm.

Ingested food stimulated the sensory nerve endings in the esophageal wall, thus, exciting the vago-vagal reflex.

Treatment consisted of successive bouginage treatments and ephedrine sulfate. Improvement was rapidly noted.

In commenting, these authors, Iglauer and Schwartz, make the poignant suggestion that many cases of heartblock are probably due to a cardiospasm, but are unrecognized as such.
PHYSIOPATHOLOGY.-- The entire mechanism is tied up in the vagus nerve ramifications involving both the sensory and motor branches. There can be no doubt that the vasomotor system is involved just as well, but the definite relationships are not yet established. On stimulation of vagal nerve, afferent nerve endings usually caused by some pathological entity impinging on these nerve fibers, a reflex mechanism is set up which passes to the cortex and out along the motor nerve trunks to the heart musculature. Cardiac rhythm is definitely and immediately disturbed, always slower and perhaps irregular.

SYMPTOMS.--Clinically, this syndrome must be differentiated from the carotid sinus syndrome which it resembles so closely. Obviously the history of some previously existing condition or action immediately preceding an attack will point toward a vago-vagal syncope. Usually these patients have had several attacks during their lifetime and can give a comparatively accurate description of a typical attack.

The chief symptoms are a preceding aura of short duration and sudden collapse. The eyes roll up and the patient looks as if he were dying. The heart sounds may be slowed, or there may be a complete block, or even a cardiac standstill. The pulse is descriptive in itself—being very slow or absent altogether. The patient usually recovers rapidly and with very little after effect in most
Tocantins (63) fortunately was taking the blood pressure on such a patient who had experienced a sudden, apparent vago-vagal attack. At first, the blood pressure was normal, but upon the onset of syncope the blood pressure dropped way out of sight until a definite rhythm was established. Nevertheless, this was much lower than normal.

Rowland and Johns (49) discovered, in a male patient under observation who suddenly fainted, a decreased ocular tone bilaterally. This was verified by a Maclean tonometer. It is questionable whether this was a vasovagal or vago-vagal attack; however, no definite conclusions can be drawn from the observation.

Clinically, therefore, the matter is one differential diagnosis after eliciting a careful history of a typical attack.

**DIAGNOSIS.**—The diagnosis of this syndrome is based chiefly on the history of preceding attacks because, as a rule, the patient afflicted with this manifestation has had several such attacks before. The combination of low blood pressure, very slow or no pulse, heart sounds of normal intensity but extremely slow or even absent in some cases, quick recovery and the general physical conditions should point toward either a carotid sinus syndrome or a vago-vagal
syndrome. The vagal type of carotid sinus syncope is very similar to vago-vagal syncope, but the differentiation according to Horine (31) may be made by a carotid sinus pressure test to determine if the syncope is on this basis. If no syncope occurs when the sinus is stimulated, the chances are that it is of vago-vagal origin.

Never should a case of syncope pass through the clinicians hands without a careful physical examination of the auriculotemporal branch distribution of the vagus, nasopharyngeal examination including reflexes, and of the lungs and mediastinum. Even the abdomen should be suspected of harboring some abnormal pathological condition which produces a vagal stimulation. Common pathological entities capable of producing syncope of this type are:
1. acute otitis media or external canal irritation involving the auricular branch of the Xth cranial nerve,
2. bronchial irritation, 3. bronchogenic neoplasm,
4. esophageal stricture, neoplasm, or dilatation, 5. hypersensitive "touch points" in the nasopharynx or pharynx which produce vagal stimulation and occasionally other abnormal physiological conditions.

The clue to the syndrome is the heart rate, and this is one of the most important points in differential diagnosis of syncope.
TREATMENT.--Here again the resemblance to carotid sinus attacks is very similar. Preventive measures should include a complete physical check-up to rule out any irritating foci which may produce the vagal irritation. The continued use of .5 mg. (1/20 grain) of atropine two or three times a day, or 1 cc. (15 m.) of tincture of Belladonnae two or three times a day are quite effective. Ephedrine may be given in addition to atropine in dosages of 15-30 mg. two or three times a day if atropine alone fails to relieve the situation. Unfortunately patients develop a rapid tolerance to ephedrine, and the dosages become prohibitive. All irritating foci should be removed surgically if at all feasible, or if this is not possible, novocainization of the vagus trunk in the neck should be performed as a diagnostic test, first on one side and then on the other. If such a test abolishes the attack, unilateral section of the vagus trunk may be achieved.

In the active treatment the methods used are standardized. The Trendelenberg position should be assumed by the patient immediately. Clothing should be loosened to insure adequate breathing. Specific drugs, such as atropine in 1/120 grain doses or epinephrine .5-1 cc. subcutaneously are of value. The former acts by eliminating the vagal effect on the myocardium,
the latter by overcoming vagal inhibition and increasing the irritability of the myocardium to sympathetic tone effect.

**POSTURAL HYPOTENSION**

**DEFINITION.**—This syndrome is one in which certain patients exhibit fainting particularly following an abrupt change from the horizontal to the upright position.

**ETIOLOGY.**—Because great quantities have been written about this clinical entity, it is not the purpose of this paper to extensively exhaust the subject material on postural hypotension.

Most authors place the hypotension group at or below 110 mm. systolic pressure, but others believe this to be too high by 10 mms. pressure.

In general, in both men and women, the normal blood pressure response upon assuming the erect from the supine position is a slight drop in systolic pressure, a slight rise in diastolic pressure, and a slight rise in the pulse rate; the pulse pressure of course falling. Since Bradbury and Eggleston (8) reviewed the literature in combination with their own clinical studies in 1925, very little has been added of importance to their conclusions. They concluded that a marked drop occurred in both the systolic and diastolic pressures. Christ and Brown (21) carrying on similar investigations found results which
coincided with Bradbury and Eggleston's in regard to the systolic and diastolic pressures. They believed the essential disturbance in this affliction is due to the lack of resistance in the splanchnic vessels to sudden shifts in the blood mass and to absent or diminished vagus regulation of the heart rate to changes in blood pressure. A hypotonic state of the myoneural structures of the sympathetic and parasympathetic nervous system exists which allows such a collapse.

Gilchrist (22) explained the situation on the basis of the decreased activity of the autonomic nervous system amounting to virtual paralysis of the vasomotor constrictor fibers. Horton, Eaton, and Meriwether (32) consider the condition due only to the inadequate vasomotor control of the arterial system when the patient arises from the horizontal to the standing position. Ferris (16) attributed the syncopal attacks to a lacking of the blood in the peripheral vessels on the basis of a venous collapse.

Mayerson and Burch (37) discovered under closely controlled experiments that venous foot and intramuscular gastrocnemius pressures in the resting horizontal position are lower in individuals who develop postural syncope than in those who do not. When these individuals are tilted from the horizontal to the upright position, an
Immediate and simultaneous rise in venous, subcutaneous, and intramuscular pressure occurs, but this rise is much lower than in those who show no circulatory embarrassment. Syncope does not occur if any significant amount of tonus exists to assist in venous return and thereby preventing vasomotor collapse.

From the data available, it is impossible to state, at present, whether the immediate cause of syncope is cerebral anoxemia, sudden loss of intracerebral pressure resulting from cardiovascular changes, or reflex action of these changes on certain centers. Gowers (24) in 1907 has suggested that decreased intracranial pressure may be an important cause of syncope in general, but at the present time this is considered rather unlikely, because spinal and cistern punctures seldom cause a syncopal attack.

The etiological factors may be considered under two classes, 1. the benign physiological and, 2. the pathological conditions. The benign physiological postural hypotensive attacks are those which occur when a person who is suddenly awakened, jumps up practically before he or she is awake. A sudden attack of dizziness and syncope often results. All of us have experienced such an attack.

The pathological conditions predisposing to postural
hypotension are numerous. Bradbury and Eggleston (8) considered disease of the suprarenals as the causative factor in hypotension and asthenia in these individuals; however, this has not been borne out. Addison's disease has been eliminated because of the lack of typical features and by the fact that the condition did not progress during the several years over which these cases have been observed. Weiss (68) suggested tabes dorsalis and other spinal cord lesions in which the sympathetic system and tracts were involved as a possible cause in some instances. Intense pain and chronic disability is often associated with hypotension and syncope. This fact led physicians, of the remote past, to use it as a means of temporary anesthesia. Before volatile anesthetics were introduced, it was the usual custom in Baron Larey's Hospital in Paris to lay the patient on his back and then raise him suddenly to the standing posture. This was especially effective in dislocation therapy work.

The present air age presents many cases of syncopal attacks in sudden maneuvers, namely; climbing too high or falling to low altitudes rapidly. Therapeutic measures have been taken to avoid the splanchnic pooling of blood in these instances by the use of abdominal binders, effective training and the like.

Parasympathetic tonus increase can be disregarded
in these cases because of the clinical findings which all point to sympathetic pathology.

Weiss (68) reports a case of a woman, age 68, who suffered severe attacks of weakness, dizziness, and faintness, particularly when changing her position from horizontal to upright. She had mild diabetes. Exudates were present in the retina. A systolic murmur was heard over the precordium particularly over the pulmonic area. EKG showed left ventricular preponderance, otherwise, the laboratory studies revealed nothing pertinent. The blood pressure showed such variations as 144/86 to 84/58, 142/76 to 76/60 or 152/84 to 65/55 on changing from the horizontal to the standing position on different occasions. No change was reported in the heart rate in spite of marked changes in blood pressure.

SYMPTOMS.--The clinical picture in postural hypotension is quite clear cut. The history is again exceedingly important. If a patient gives the story of dizziness and syncope on suddenly arising from a horizontal position, then the entity is fairly well established. The patients usually complain of dizziness, headache, faintness, and actual syncope in many cases. There is usually a local or generalized deficiency of sweating according to Bradbury and Eggleston (8). The heart rate remains quite constant, but the blood pressure drops rapidly in both
systolic and diastolic elements. The patient usually finds quick relief upon reassuming a horizontal position if syncope has not developed spontaneously.

PHYSIOPATHOLOGY.--The pathological, or more correctly, the abnormal syndrome herein mentioned is due to a faulty vasomotor center control. The center probably lies in the hypothalamus or medulla, and normally exerts an equilibrated vasoconstrictor-vasodilator tone. In this syndrome, whether to a pathological condition such as adrenal hemorrhage, traumatic shock, anesthesia, tuberculosis and debilitating diseases; or to an inherent, lowered, sympathetic tone with an increased vasodilator mechanism. The blood "pools" itself in the abdominal viscera producing a diminished venous return and syncope because of cerebral anoxemia (3) and (26).

DIAGNOSIS.--The diagnosis is made by eliciting a history of recurrent syncopal attacks on suddenly arising from a horizontal position which are characterized by dizziness, faintness, and syncope. A lowered blood pressure and a normal heart rate and pulse are found on physical examination.

Anhidrosis so frequently found as an accompanying complaint in these cases, more than ever, suggests some decreased sympathetic capacity because the sweat glands normally secrete chiefly under sympathetic control.
Vagal effects may be ruled out by the use of atropine which fails to produce a cardiac acceleration in these cases.

The chief diagnostic points are then the history of previous attacks brought about by the sudden change in the position from a horizontal to an upright position, the blood pressure, and correlated physical findings during syncopal attacks.

**TREATMENT.**—The treatment of postural hypotension is relatively simple. Richardson (45), in 1854, suggested that mechanical factors of syncope may be eliminated by assuming the horizontal position, and this observation has been proven through the years to be an excellent therapeutic agent. Goodman (23) instructs the patients in the following manner: 1. reasonable hours for retiring and arising; 2. morning exercises; 3. corrective breathing habits; 4. hot and cold shower baths; 5. periods of relaxation through the day; 6. Tincture of Nux Vomica 15 gtt. three times a day and increase until the physiologic action of the drug is achieved; and 7. avoid excessive indulgence of tobacco, alcohol, tea or coffee. It has been found that a small rise in blood pressure occurs when this schedule is closely adhered to.

Abdominal binders and corsets are of value in supporting the abdomen by increasing the intraabdominal pressure.
In most instances the best therapeutic measures are the adrenergic drugs, namely; epinephrine and ephedrine. Epinephrine may be used in loc. doses of 1:10,000 solution subcutaneously. Ephedrine should be given in large doses of 15-30 mg. (¼-½ grain) three or four times a day.

**PROGNOSIS.**--The prognosis is excellent in these cases, but this depends entirely on the amount of corrective exercises which are undertaken by the patients. No matter what is done, some recurrence of postural hypotension are bound to recur.

**GOWER'S SYNDROME**

**DEFINITION.**--A type of prolonged seizure, the symptoms of which consist chiefly in the disturbance of some of the functions of the vagus for the most part sensory and subjective. They are probably due to functional changes in the vasomotor and vagus nerves. Gowers (24) used the terms "vagal" and "vasovagal" in referring to this syndrome which produces a great deal of confusion in the nomenclature of some of the various syncopal attacks. The reference here is made to a previous subject "vasovagal syncope" which has already been discussed.

**ETIOLOGY.**--It is essentially a disturbance in the function of the vagus and sympathetic system manifesting itself in both the sensory and subjective manner. Women suffer
far more frequently than men, but the latter occasionally show such a syndrome. This, and the fact that the vagus and vasomotor systems are readily influenced by emotion, has probably submerged this manifestation in the group of "Hysterias." It is probable that the syncopal attacks are chiefly on an emotional basis.

Controversial issues arise concerning this syndrome because Gowers (24) used the term "vasovagal" in describing his clinical studies on these patients. He neglected to study the cardiovascular system during such attacks, consequently, the whole issue is a questionable matter. It was suggested by Gowers that these cases were closely akin to epilepsy actually existing in the borderland between epilepsy and neurogenic syncope.

Lewis (36) raised an active objection to this syndrome as a true clinical entity because Gowers used, misconceivably, the term "vasovagal syncope." Therefore, Lewis believes his descriptions should be considered of very little value since there is insufficient cardiovascular study.

Weiss and Wilkins (72) place the syndrome in the category of a psychic manifestation and not as a separate distinct entity.

In concluding the etiological discussion it must be viewed with an open mind and any interpretations formed
should be supported by further careful study before de-
definite statements can be made. Little evidence, at
present, can be found in the literature which would
support such a syndrome.

SYMPTOMS.—The attacks are moderate in length, lasting
about ten to thirty minutes. With the vagal symptoms
there is often a slight mental change and some distur-
ance in the vasomotor system causing constriction of
peripheral vessels and coldness of the extremities.

The attacks usually come on gradually with a
sensation referred to the epigastrium, described as a
sense of oppression or fulness, but often indescribable.
This sensation ascends to the chest. There is seldom
nausea and never vomiting. There is a sense of re-
piratory distress and difficulty in breathing which may
become actually orthopneic in character. Cardiac symptoms
such as discomfort and acute pain are elicited in some
cases characterized by a feeling of rapid action and
sudden stoppage of the heart. The attacks may be so
severe the sense of impending death presents itself to
the patient's alarm. Although, there is no impairment
of consciousness a peculiar slight mental state is common.

The vasomotor spasm sometimes attains a high degree;
producing a symmetrical coldness, pallor of the face,
shivering, numbness, and tingling of the extremities.
Gowers (24) recognized these attacks as relatively different from simple cardiac fainted, but Lewis (36) considered his differentiation of a definite entity as probably false and possibly a borderland type of epileptic attack.

A fairly typical case was described by Gowers (24) of a single woman, age 28, whose cousin was epileptic. She had suffered for a period of occipital headaches on waking in the morning. Suddenly in the forenoon she found she couldn't fix her attention on any subject. After this had lasted for an hour or so, a sense of sleepiness came on—then suddenly she felt wide awake with an intense sense of fear, extreme coldness of the hands, face, and feet and unable to move. Presently, an indescribable sensation was felt in the epigastrium and back, sometimes with nausea. After a few moments cardiac palpitation, orthopnea, and a flush would come on accompanied by teeth chattering. During the attack there was a curious sense of unreality in everything about her. The coldness was usually extreme making her feel as if she were made of stone. The feeling of impending death always accompanied these attacks.

PHYSIOPATHOLOGY.--It is difficult to attempt a description of the underlying mechanism in such a condition as this when it is quite possible there is no real physio-
logical basis other than a psychic disposition. It is all very well that the vagus has such a close relationship to cardiac rhythmicity of beat, but if one assumes the vagus does act in an inhibitory manner one should also expect a vasodilatation mechanism in conjunction with the vagal activity. Instead, however, a marked vasospasm with numbness, tingling and coldness follows in the wake of the so-called Gowers syndrome.

I believe it would be adequate to consider the psyche at fault in such a protean condition as this, and allow the future to determine the fallacy or the truth of the syndrome. Weiss (68) is inclined toward placing the Gower's syncopal syndrome under the cerebral type of carotid sinus syncope. Several later writers have reported as Gower's syndromes attacks entirely different in nature from those presented by Gowers.

**DIAGNOSIS.**—Assuming, for purely academic purposes the existence of such a syndrome, the combination of blanching of the skin, numbness, and tingling of the hands and feet, anxiety, fear, palpitation, and giddiness, and attacks lasting from 10-30 minutes with possible syncope then the diagnosis of a Gower's syndrome might be made.

**TREATMENT.**—It is essential to discover any defect in the general health and anything that places a strain on the nervous system and attempt to put these right.
fatigue should be avoided and constipation adequately relieved. Bromides are of value. Amylnitrite or nitroglycerine for relief of the vasospasm are very useful agents. Nitroglycerine is excellent in regular daily doses in preventing such attacks and in steadying the vasomotor center.

It must be added that the above mentioned treatment is of value for conditions which are only classified as a Gower's syndrome. The entity must be proved before conclusive therapy is advocated.

**NOTHNAGEL'S SYNDROME**

**DEFINITION.**—The so-called Nothnagel's syndrome is the syndrome of "angina pectoris vasomotoria" as described by Nothnagel in 1867 (38). It is an attack of palpitation associated with a sense of anxiety, dizziness, and syncope.

**ETIOLOGY.**—Nothnagel (38) claimed that the attacks were due to a generalized spasm of the arterial system and probably no pathological condition of the heart existed accompanying these attacks.

Obviously, however, in the middle of the nineteenth century there was very little of the technic, as does exist today, which would make it possible for the scientific men of that time to verify results of this type of procedure. In other words it is difficult to definitely state whether the cases fall in the group of syncope.
anginosa or in some other group.

Weiss and Wilkins (72) placed Nothnagel's syndrome as a definite psychic entity along with Gower's and the vasovagal neurosis. In all probability this has much in its favor and is a means of classifying it. Weiss (68) on the other hand, states the symptoms resemble those of the vasovagal, carotid sinus, and vago-vagal syncopal attacks and that the syndrome should not be classified as a separate syndrome in regard to etiology, symptomatology, and the like.

Since the subject is a disputed one, it is merely the purpose here to mention it as one of the possible manifestations of syncope to be either proved or disproved in the future.

SYMPTOMS.--Nothnagel described a series of four cases, without heart disease, who developed the condition of severe palpitation associated with a sense of anxiety, dizziness, and syncope. The attacks usually start with a sensation of coldness, numbness, and tingling over the hands and feet. The entire extremities may be involved, and in some cases, even the trunk shows evidence of coldness. There may be a slight sensation of dyspnea, and definite pallor of the skin. There is pain over the precordium at times associated with a feeling of anxiety and fear of impending danger as occurs in angina. The
pulse is small, but consistent during the attack. Consciousness is disturbed, but not usually lost. The heart rate is unaltered or moderately diminished. The skin shows diminished sensation, and increased perspiration.

Predisposing factors in producing the onset of such symptoms according to Lewis (36) are cold and bouts of drunkenness oftentimes associated with an emotional state of some order.

The attacks themselves last from 15-60 minutes before subsiding completely, and there is seldom any aftermath.

PHYSIOPATHOLOGY.—It is rather difficult to attempt an explanation of something that isn't proven as yet. The situation here is similar to that of the Gowers syndrome—described, but unproved and unaccepted in general, although, Lewis (36) believes Nothnagel was more precise than Gower.

The circumstances probably occurring if an explanation can be attempted is probably on an emotional basis. The heart rate is usually unaltered, thus, indicating no excessive vagal or sympathetic activity on that organ. The vasomotor reciprocal tonus system is upset resulting in a vasoconstriction overbalance. The evidence of pallor of the skin associated with coldness, numbness, and tingling indicates a decreased blood flow which in the absence of any other explanation must be on a vasoconstriction
basis.

There is according to Lewis (36) and Weiss (68) insufficient clinical and experimental evidence to make this syndrome of any importance.

DIAGNOSIS.—The diagnosis of this syndrome has very seldom been made since Nothnagel (38) first described the manifestation. However, from the academic point of view, it may be made on: 1. the combination of a good history, (including predisposing factors such as low temperatures, emotional upsets, and habits of drunkenness), 2. clearcut symptoms of blanching of the skin, numbness and tingling of the extremities, small pulse, anxiety, fear, and palpitation, and, 3. syncopal attacks lasting 15-60 minutes.

In comparing Gowers' with Nothnagels' syndrome, however, the question arises as to which is which because of the marked similarity of the two conditions. Consequently we are faced with the puzzling question as to whether the two syndromes are different or alike and whether they are existant or non-existant.

TREATMENT.—Adequate psychotherapy may prove sufficient in most cases if any are discovered. Warm clothing, avoidance of exposure to cold and elimination of liquor from habits should tend to remove all predisposing factors, and bring about an excellent recovery.
INTRODUCTION.--Here is a syndrome of syncope that has been recognized since 1799, but until the last two decades very few observations had been made of importance. Beginning in 1923, there has flooded the literature reports of all varieties and nature concerning the carotid sinus syndrome. It would be impossible to touch a fragment of the literature printed, but an attempt will be made to present a generalized resume of the more salient reports to make it of value in a differential diagnosis procedure.

HISTORY.--In ancient times the Chinese were accustomed to produce a state of anesthesia by making pressure on the neck. The Assyrians compressed the veins of the neck apparently by tying a band about the neck before practicing circumcision.

Parry (42), a Welshman, made the original observation in 1799 that pressure over the carotid sinus area caused marked slowing of the heart. He made clinical use of this fact in some of his patients to slow down the heart rate. The observation was also made that carotid sinus pressure produced a respiratory distress characterized by longer and deeper respirations.

Waller (65) in 1862 noticed he could retard the heart rate 4-5 beats per minute by pressure on the carotid
sinus. He also ruled out the possibility of the compression of the carotid artery causing the cardiac slowing.

Czermak (9) in 1866 repeated Waller's experiments and found similar results. He attributed this to a probable stimulation of the vagus nerve, which had been proven to have such an effect on the heart by mechanical stimulation of the bulb-like dilatation of the carotid artery. This phenomenon was known as the vagus pressure test, "vagus Druckversuch." Most of Czermak's tests were conducted on himself.

Hering (29) in 1923 first made clinical observations on the matter, that light pressure over the carotid sinus produced a cardiac slowing. To him it seemed improbable that this could be due to vagus stimulation. Consequently experiments carried on by Hering showed that he could produce the same effect by pressure over the sinus even when the vagus is separated from the artery. This was in itself proof that a true reflex was involved, and that the conclusions drawn by Czermak (9) in 1866 were incorrect.

Since Hering's discovery anatomical investigation has isolated the nervous elements of the carotid sinus mechanism. There is a definite sinus nerve of Hering's and the intercarotid nerve of de'Castro, as described in the anatomical and physiological section of this paper.
Weiss and Baker (59) made the first study on a series of cases of carotid sinus syncope as a rather common cause of fainting.

During the last decade the literature carries many reports of work done on the carotid sinus syndrome of which a short resume will now be attempted.

ETIOLOGY.--The etiology of the syndrome is that of a hypersensitive carotid sinus resulting from the hyperirritability of the nerve endings within the sinus from hypersensitivity of the synapses of the center, or from irritability of the end organs of the reflex alone, or in combination.

The carotid sinus mechanism may be reviewed in the Anatomy and Physiology chapter of this paper. Briefly in a clearcut description given by Lewis (36) it exerts a powerful controlling influence upon the heart rate, blood pressure and respiration. Especially important is the regulatory mechanism of the brain circulation. A rise in pressure in the carotid sinus results in a reflex lowering of the heart rate and blood pressure. The former is produced through the vagus, the latter through the vasomotor mechanism and through the effect on suprarenal secretion.

Wilks (73) considered the syncopal attack to be due to cerebral manifestations probably caused by a
lower motor neuron condition, but this has been almost completely disregarded by present day research workers.

Weiss, Capps, Ferris, and Munro (70) found that frequently the syndrome was caused by hormones and other chemical substances as well as the mechanical stimulation factor.

Weiss (68) found that from clinical experience the underlying morbid changes which produce the above reflex condition are numerous. Organic lesions such as inflammation, enlarged lymph nodes, malignant tumors of the neck, arteriosclerotic aneurysmal dilatation of the sinus, or sclerotic alterations in the coronary vessels and in the conductive system. Nervous hypersensitization such as that occurring following the administration of digitalis or in other emotional states. Ferris, Capps, and Weiss (17) added such abnormalities as tuberculosis, adenitis, meningovascular lues of the C.N.S., dietary deficiencies, chronic alcoholism, and the menopause. Stevenson (60) mentioned arteriosclerosis and hypertensive heart disease as possibilities in the causation of syncope.

It has been found that the syndrome is more common in males than in females, and far more frequent in the older age groups according to Robinson (48) who studied a long series of cases from this point of view.
SYMPTOMS.--The symptoms have to be divided into three separate groups because it was found by Weiss and Baker (69). There existed three entirely different manifestations of carotid sinus disease, namely:

1. Reflex vagal inhibition of the heart and a fall in the arterial pressure which depends on the cardiac slowing--The Vagal Type.

2. Direct reflex fall of the arterial pressure without any cardiac rate changes--The Depressor Type.

3. Onset of typical syncopal attacks, but without cardiac slowing or arterial pressure decrease--The Cerebral Type.

In people with a normal carotid sinus mechanism, any pressure over the sinus will produce very little effect except a slight slowing of the pulse and fall in the blood pressure. This may be a little more marked in arteriosclerotic and hypertensive disease.

In eliciting the history of patients who complain of syncopal attacks of this variety there is usually some pathological condition present in the neck or it may be brought on by tilting the head slightly backward or to the side. Even bending the neck or pressing the neck against a stiff collar has been known to produce such an attack. Ferris (15) suggested the factors of extreme
changes in position, psychic, and emotional changes, menstruation, and menopause. Active massage and pressure for 10-15 seconds just posterior to the angle of the jaw will produce syncopal attacks in those with a hypersensitive carotid sinus.

The subjective symptoms commonly indicated by the afflicted patients are generally consistent. Predominating among these symptoms are dizziness, light-headedness, tinnitus, blurring of vision, anxiety, perspiration, pallor, numbness, and tingling. Convulsions result if the syncopal attack endures for several seconds.

The objective signs and symptoms vary according to the type of carotid sinus syncope produced, namely: vagus, depressor, or cerebral. These will be differentiated as separate conditions shortly. The common signs observed during a syncopal attack are a loss of consciousness or mental confusion, facial pallor which resembles that of a dying person, slow or normal heart rate, decreased or normal blood pressure and an imperceptible pulse. Immediately following the attack the face becomes flushed. Profuse perspiration appears accompanied by hyperpnea and occasionally dyspnea. Pupillary dilatation is commonly seen immediately following the onset of the syndrome.

Vagal Type.—Characterized by dizziness, faintness,
and weakness resulting from cardiac asystole. This is
due to a sinoauricular or atrioventricular block. There
is a fall in blood pressure accompanying the cardiac
slowing.

**Depressor Type.**—A rather uncommon variety character-
ized by symptoms similar to the vagal type, but the cardiac
remains constant. The blood pressure drops remarkably
because of the generalized vaso-dilatation.

**Cerebral Type.**—There are no changes objectively in
the heart rate or blood pressure, although the subjective
symptoms are similar to preceding types.

In the first two groups, syncope is due to a cerebral
ischemia resulting from the bradycardia and hypotension.
In the last group the exact cause is unknown.

Smith (55) reports tremendous changes in the EKG
form in the vagal type. Striking differences are noted
in the cardiac conduction system with either sudden slowing,
varying degrees of heart block or cardiac standstill.
Respirations are deep and labored, but there is no cor-
relation between labored breathing, cardiac slowing, and
fainting attacks. Patricelli (43) noted ventricular ex-
trasystoles, nodal rhythms, and bizarre complexes in EKG
studies.

Ferris, Capps, and Weiss (18) classify the symptoms
and signs according to systems involved. Many subjective
and objective neurotic stigmata and neurogenic manifestations were found in these patients with carotid sinus sensitivity.

Central------
(fainting, dizziness, and weakness.
(convulsions.
(amnesia; catalepsy.
(sleeplike states.
(fatigue; weakness.
(pupillary changes.

Ocular--------
(strabismus.
(lacrimation.

Respiratory-----
(shyperpnea.
(apnea.
(yawning and sighing.

gaseous eructations.
(nausea; vomiting.
(increased peristalsis.

Gastro-intestinal-
(hypotension.
(peripheral constriction.
(peripheral dilatation
(sweating.

Vasomotor-------
(bradycardia.
(arrhythmia.
(palpitation.

Cardiac--------

Extremities-----
(convulsions.
(Babinski phenomena.

In no instances has there been observed that induced attacks harm the patient nor are there any reports of cases of instantaneous death resulting from a carotid sinus attack. Digitalis has a definite sensitizing effect on the carotid sinus reflex, particularly in the presence of coronary disease. Consequently, it is believed unwise to administer a volatile anesthetic following digitalization.
for fear of causing instantaneous death. Weiss (68)

Animal experiments have shown the following facts to exist, 1. increased pressure in the carotid sinus causes a slowing of the heart, vasodilatation and a fall in blood pressure, whereas, decreased pressure in the sinus gives rise to the opposite effects, 2. that all these reflexes are abolished by denervation of the carotid sinus or section of the intercarotid nerves, 3. that the reflexes have both the sympathetic and vagal efferent pathways, the heart rate being determined chiefly by vagal stimulation or inhibition, the vasomotor tone by sympathetic control. These facts have led to increased understanding of the syndrome and direction toward surgical therapy in humans.

Robinson (47) carried on a series of venous pressure experiments in eight patients with hyperactive carotid sinus reflexes. His work, carried out under excellent control, was significant in that the venous pressures failed to respond in any way preceding, during or after an attack of carotid syncope. This differs considerably from arterial pressure findings, as has been discussed previously. The symptoms are due to the arterial or capillary side of the circulatory tree placing it under definite vasomotor control.

PHYSIOPATHOLOGY.--The mechanism is due, in general, to a
hypersensitive carotid sinus from a mechanical, chemical, or hormonal cause. The carotid sinus is a powerful controlling influence upon the heart rate, blood pressure, and respiration, and especially in regulating brain circulation. A rise in pressure in the carotid sinus results in a reflex lowering of the heart rate and blood pressure. This belief was later modified by Weiss and Baker (6) when they described the cerebral type in which the blood pressure and heart rate were uninfluenced. Except for the one type just mentioned; however, the vagus produces a cardiac slowing, and the vasomotor system collapses to produce a vasodilatation and blood pressure drop. The general anatomical and physiological characteristics were discussed in the chapter on Anatomy and Physiology.

Proof of the effectiveness of the carotid sinus exists in the discovery that all these reflexes are abolished by denervation of the carotid sinus or section of the intercarotid nerves. Pressure on the carotid artery lower down in the neck does not elicit the reflex in clinical cases; therefore, it must be assumed that mechanical blocking of the carotid artery has no syncopal effect according to Baker (2). Finally Weiss and Baker (6) infiltrated the wall of the carotid sinus with novocaine and eliminated the carotid reflex indicating
the interrelationship between the reflex mechanism and the carotid sinus.

Smith and Moersch (57) attributed the striking changes in the cardiac mechanism to a temporary suppression of the sinus and the presence of a refractory period in which the heart does not form a new foci of impulses. The cerebral symptoms are on an ischemic basis which was built up rapidly. These authors proved this by compressing both carotid sinuses at once, and, thus, producing a more rapid syncope.

Again, Smith and Moersch (58) reported macroscopic and microscopic variations of the internal carotid artery and sinus. The sinus wall is essentially the same as the medial vessels except for an increased advential layer and a decreased media layer.

Quoting Smith and Moersch (58), "It appears, therefore, that, in these cases of spontaneous and induced syncopal attacks, other factors are present besides those which the physiologist understands as the carotid sinus reflex." This is in all probability quite true because of failure of certain indicated therapeutic measures.

Weiss, Capps, Ferris and Munro (70) discussed the physiological anatomy of each of three various types of carotid sinus syncope. In the vagal type the efferent responses travel over the vagus nerve and set up a
partial or complete temporary heartblock which can be abolished in three minutes by the use of epinephrine. The depressor type carries afferent impulses by the vagus, whereas, the efferent fibers act on small blood vessels by way of the aortic depressor nerve. In the cerebral type afferent impulses arise from the carotid sinus, pass to the medulla and efferent impulses go to the vegetative centers in the region of the hypothalamus or to the supplying of blood vessels.

Gilchrist (22) associated fainting in the vagal and depressor groups to a cerebral ischemia which resembles a simple vasovagal attack markedly, and must be ruled out. Ferris, Capps, and Weiss (17) studied cerebral blood flow in the carotid sinus, and found the pial vessels to be the ones which constricted. However, the pial vessels carry very little blood which makes it impossible to alter the brain contents remarkably, and the tendency to faint does not parallel such changes. Hering (29) suggested that the carotid sinus exerts a major influence in regulating autonomic tonus, but this is questionable, because the B.M.R., Reynauld's Disease, dermatographism, and chronic fatigue were not altered by either stimulation or denervation of the carotid sinus. The obvious conclusions are that the carotid sinus is only one of a number of sensory areas in the body which influence the
autonomic nervous system control. When the carotid sinus is removed, other unknown centers take over the function. This is borne out by Freedberg and Sloan (19) who state that certain attempts to raise the blood pressure by denervation of the carotid sinus in man is a futile procedure. Patricelli (43) reports a case in which similar results were secured by doing a double vagotomy to no avail in attempting to raise a low blood pressure.

Thus, it is seen that although the principal mechanism of the vagus, intercarotid, sinus, and aortic depressor nerves are understood it is still a little difficult to assign to the carotid sinus the primary governing function of the blood pressure and vasomotor control of the blood vessels. Some other center or centers are probably in existence, as yet undiscovered, which assist the carotid sinus in its function.

DIAGNOSIS.--The evidence that hyperactivity of the carotid sinus may result in syncope and related manifestations is based on: 1. observations of spontaneous attacks in a number of cases; 2. on the fact that mechanical stimulation of the carotid sinus promptly induces syncope identical in nature to that of the spontaneous attacks; 3. on the history and character of precipitating attacks; 4. on the observation that occlusion of the carotid artery below the sinus fails to cause similar symptoms,
These attacks often simulate epilepsy, however, in epilepsy carotid sinus stimulation produces no effect.

Robinson (48) cites the criteria for diagnosis of the carotid sinus syndrome as follows; 1. similarity of induced and spontaneous seizures, 2. the nature and length of the aura; in sinal syncope the aura is short, immediately preceding the attack, whereas if it precedes the attack by hours or days the cause is from some other origin, 3. the patient is almost always in the vertical position before such an attack, 4. the duration of the attack is usually short from seconds to minutes because when the patient falls syncope ceases, and 5. the sudden clearing of the sensorium after such an attack which is a characteristic manifestation.

Weiss, Capps, Ferris, and Munro (70) suggested the therapeutic diagnosis in sinus syncope. In the vagal type, intravenous administration of atropine will abolish the attack within three minutes. In the depressor type atropine has no effect, but epinephrine will promptly abort the attacks. The cerebral type shows no effect from either atropine or epinephrine.

The general characteristics of carotid sinus syncope should make the diagnosis comparatively easy as compared to other varieties of syncopal attacks. In brief, the history, symptoms, and signs are all important in
indicating that simple cerebral ischemia resulting from arterial occlusion is not involved; 5. on the observation that novocain block of the sensitive sinus abolishes both spontaneous and induced attacks.

In differentiating the three varieties of carotid sinus syncope: 1. those showing cardiac standstill or slowing with or without blood pressure changes may be recognized as the vagal type; 2. those showing a marked fall in blood pressure without pronounced cardiac slowing are the depressor type; and 3. if there is no cardiac slowing or blood pressure changes, then it must be classified in the obscure cerebral type.

The history of previous spontaneous attacks induced by sudden pressure in the neck, turning of the head, and certain pathological conditions in the neck all should be suspected of stimulating a hyperactive carotid sinus. The combination and classification of the signs with the history should prove adequate in making the diagnosis. Manual stimulation of the sinus in these individuals will serve to bear out the tentative conclusion when a syncopal attack is produced. It must be noted that carotid sinus syncope practically invariably occurs in the upright position, but this is not as necessary as in the vasovagal, or postural hypotensive types of syncope.
at the correct conclusion.

**TREATMENT.**—This depends on the nature of the attacks and on the predisposing causes. During the attacks, the clothing should be loosened, particularly around the neck, and the patient placed in the horizontal position. Specific diseases such as lues, adenitis, digitalis intoxication, and dietary deficiency. Glandular therapy is of value in menopause factors. Adequate psychotherapy by changing the trend of thought and complete instructions concerning the movements of the neck are indicated.

Medical therapy depends on the type of attack. In the first group, the vagal group, atropine in doses of .5-1 mg. (1/120-1/60 grain) twice a day are beneficial in preventing recurrent attacks. The second group, or depressor group, is best aided by .5-1 mg. (1/120-1/60 grain) of epinephrine, or from .5 to 1 cc. (7-15 mm) of a 1:1,000 solution may be administered subcutaneously, or .5 cc (7 mm) of a 1:10,000 solution may be injected slowly intravenously. Ephedrine in amounts of from 15-30 mg. (1/4-1/2 grain) three times a day may also be used. In the third group, or cerebral group, no drugs have proved efficacious in preventing such attacks, therefore surgery has come to the fore.

Smith and Moersch (58) consider drug therapy of very little value, but this is not the general consensus of
opinion as held by most authors.

Robinson (46) reports two cases in which benzedrine sulfate was substituted in the place of ephedrine in the treatment of the vagal and depressor types. He found benzedrine to possess a more prolonged action and consequently of definitely greater value than ephedrine. The dosage given was 20 mg. three times a day. Since benzedrine is a sympathicomimetic drug producing vasoconstriction and increased blood pressure, there is no reason why this should not be successful.

Barbiturates are of little value in preventing or stopping syncopal attacks according to Patricelli (43) and Robinson (48).

Surgical therapy has become increasingly more popular during the last five years. This is especially effective in the cerebral variety of carotid syncope.

Weiss, Capps, Ferris, and Munro (70) give the technic of a surgical procedure of denervating the carotid sinus used most widely. The technic is as follows: a three inch incision is made along the anterior border of the sternocleidomastoid muscle with its center opposite the cricoid cartilage. The incision is carried through the skin, platysma, and superficial fascia to the muscle. The sternocleidomastoid is then retracted laterally exposing the carotid sheath. The internal jugular is re-
tracted medially and the common carotid artery is exposed by dividing the sheath. The internal, external, and bifurcation are then exposed and stripped, the process carried out from the periphery toward the bifurcation. The arteries should then be stripped for at least 2 cm. above the bifurcation. The intercarotid tissue should be freed laterally and beneath, and divided and isolated a short distance above its attachment to the bifurcation. The wall of the artery is very friable in this region, so care should be exercised. When the intercarotid nerve is sectioned, blood pressure and heart rate increase immediately. This is only a temporary condition for about 2-6 hours. Patients do not show any permanent alteration in blood pressure or heart rate.

The results of surgical denervation of the carotid sinus are excellent in most cases although great care must be exercised. Vasomotor disability and visual acuity improves strikingly for a temporary period only. Fatigue disappears. The heart rate and blood pressure increase temporarily, and then return to a low normal.

If a neuroses complicates the carotid sinus syndrome, surgery is of little value, because the neurotic element predominates the syncopal attacks.

Stevenson (60) brings forth the concept of Roentgen therapy in these cases. In general X-Ray in autonomic
nervous system diseases has proved rather disappointing. Stevenson cites five cases of carotid sinus syncope and the use of X-Ray. Following X-Ray therapy the patients remained free from attacks. The technical factors involved in these treatments are as follows: 165 Kv through a filter consisting of 5 mm. Cu and 1 mm. of Al; a 50 cm. target skin distance; a 10x10 cm. cone is centered over the neck area; a 500 R. single dose is given to one side on one day, and the same to the opposite side on the next day. The only complications noted have been a mild parotitis which quickly disappears. The general prophylactic value of X-Ray therapy is of questionable value as yet, because of the few cases investigated. Further studies should be made, however, because of reports of drug failures and surgical hazards in several cases, Smith and Moersch (57).

PROGNOSIS.--The efficacy of the medical, surgical, and X-Ray treatments is quite adequate in most cases; therefore, one must conclude the prognosis is excellent. No deaths have been reported as directly due to the carotid sinus syndrome, but it may become a basis for many neurotic manifestations as well as dangerous to an individual's well being if allowed to continue.
INRINSIC SYNCOPE
ADAMS-STOKES SYNDROME

DEFINITION.--A syncopal attack resulting from sudden slowing of the heart because of an organic lesion of the conductive system produces a heart-block.

HISTORY.--This syndrome was first described in 1827 by Adams (1) in his original report describing the condition of "an officer in the revenue aged 68 years, of a full habit of body, had for a long time been incapable of any exertion, as he was subject to oppression of his breathing and continued cough." In May, 1819, Mr. Adams saw the patient who was just recovering from the effects of an apoplectic attack which had suddenly seized him three days before. Adams noted the irregularity of his breathing and remarkable slowness of the pulse, "which generally ranged at the rate of 30 in a minute." The gentleman had not had less than twenty apoplectic attacks. Before each of them, the patient felt heavy and lethargic with a loss of memory. He would then fall into a state of complete insensibility, during which time, his pulse would become slower than usual and the breathing loudly stertorous. On November 4, 1819, the patient died and was posted by Adams. In explanation of the attacks Adams stated, "Indeed upon considering the latter condition of things, where the heart is slow in transmitting
the blood it receives we find, I imagine, even in this a means of accounting for the lethargy, loss of memory and vertigo, which attend these cases. For the venous blood, which under these circumstances is supposed to accumulate in the brain, is evidently ill-suited to the functions of this organ."

Stokes (61) in 1846 described "seven cases of permanently slow pulse." In five, "organic disease of the aorta or valves, or both" was discovered on autopsy.

Since the first reports by these two men there have come reports that earlier observations had been made of this syndrome, but the primary credit appears to be due to Adams and Stokes. Thus the name "Adams-Stokes Syndrome."

ETIOLOGY.--The primary cause is an A-V block with prolonged asystole. The attacks may be permanent or temporary in nature resulting from either of the following causes, namely: 1. a sudden shifting in the pacemaker within the ventricle, or; 2. a sudden temporary inhibition of the ventricular pacemaker. In some instances, the auricular impulses are prevented from reaching the ventricles and the latter may not contract for several seconds. Complete and permanent dissociation of the heart with resulting tachycardia and fibrillation are often encountered. Such cases represent that the essential feature of Adams-Stokes syndrome is not the level of the heart rate,
but the functional capacity of the ventricles.

Davis and Sprague (11) state ventricular fibrillation is the common mechanism of syncope in the Adams-Stokes syndrome. Lewis (36) agrees this is due to a fibrillation and is often fatal. Some injury or disease of the Purkinje system allows circus movements in the ventricles. During these attacks, cerebral anoxemia occurs and syncope results.

Walker (64) considers the bradycardia and cardiac asystole due to a disturbance in the depressor mechanism proper. A discussion of this mechanism is given in the chapter on Anatomy and Physiology.

Schwartz and Jezer (51) report cases of complete auriculoventricular dissociation who were subject to recurrent attacks of fibrillation.

Common etiological factors producing the A-V dissociation, are rheumatic heart disease, coronary sclerosis, endocarditis, myocarditis, diphtheria, scarlet fever, syphilis and any other pathological manifestation interfering with the A-V conduction mechanism.

SYMPTOMS.--Clinically the syndrome is manifested by various signs and symptoms dependent on the degree and rate of slowing of the heart. If asystole is not marked the only symptoms are light-headedness, dizziness, and a sensation of faintness. If the slowing is sudden and severe, the
patient may collapse without warning. In such cases there is a generalized pallor and appearance of collapse. Convulsions occur more frequently in this type than in any other type of syncope and are often very severe. If the slowing is gradual and lasts for a prolonged period, the patient may be constantly in a semi-stuporous or psychotic state. The heart rate becomes progressively slower, reaching such rates as 25-35 beats per minute.

Heard, Marshall, and Adams (28) cite a typical case of Adams-Stokes syndrome. A white male, age 63, civil engineer was admitted to the hospital for treatment of heart failure accompanied by fainting attacks with or without convulsive seizures. Approximately three months before admission the patient noticed dyspnea on climbing hills, mental confusion, rapidly increasing effort fatigue and periodic attacks of unconsciousness. The family physician found the heart rate to be 18 beats per minute with a regular rhythm. Frequent syncopal attacks occurred during the next few days with periods of ventricular asystole. A heart rate above 20 was never observed. On hospital admission the rate was 30, blood pressure 150/44, Cheyne-Stokes respiration, edematous ankles and pulmonic rales observed during examination. During the course in the hospital BaCl₂ administration caused the heart rate
to jump to 60-72 beats per minute, but relapsed in 17 days to a rate of 20. Death occurred in an attack of convulsive syncope.

Silberberg (54) describes well the heart sounds generally heard in these cases. The first sounds is accentuated and the auricular sounds may be heard in the long pauses. Syncope usually occurs when the 2:1 block changes over to a complete block, that is, in the refractory period.

Generally, then the outstanding signs and symptoms are the cardiac slowing or asystole accompanied by pallor, dizziness, and probably unconsciousness.

PHYSIOPATHOLOGY.--The intrinsic nerve mechanism of the heart lies in the sinoauricular node, the atrioventricular node, the Bundle of His and Purkinje system. These tissues are not histologically true nervous tissue, but, instead are specialized bundles of muscle tissue capable of exciting a rhythmical cardiac beat, and transmitting the impulses through the heart muscle making the heart independent of nerve elements if the need ever arose.

In cases of Adams-Stokes syncope the conduction system is interfered with by an organic lesion with the result of a typical syncopal attack on a cerebral anemia basis. Either marked slowing or complete heart block occurs suddenly. When the ventricle stands still over
10 seconds cerebral symptoms dominate the picture, over 15 convulsive seizures and over 20 seconds death often results. In cases where the rate shifts into a fibrillation, ventricular in origin, Davis and Sprague (11) consider this to be the cause of syncope and death in the Adams-Stokes attack. Ventricular dissociation and fibrillation or standstill are the danger signals of a bradycardia or heartblock.

**DIAGNOSIS.**--The diagnosis is chiefly made on obtaining a history of previous attacks of syncope with associated symptoms of pallor, lightheadedness, dizziness and a sensation of faintness. A sudden slowing of the heart rate is essential in the diagnosis of an Adams-Stokes attack.

The differential diagnosis should be moderately easily arrived at in this particular syndrome because only the Adams-Stokes syndrome of reflex origin (vaso-vagal attacks) and the vagal type of carotid sinus syndrome give a slowing of the heart rate to a marked degree.

The distinction is relative rather than absolute; however, for one sees patients with definite evidence of myocardial disease and with partial or complete auraliculoventricular block between the seizures.

In summarizing, the syncopal attacks of acute
cardiac disease, as often seen in these cases, resemble that of an acute peripheral circulatory collapse of neurogenic origin. However, in addition to the faintness, loss of consciousness, and signs of circulatory collapse, there are two distinguishing features—a slowing of the heart and fulness of the veins. Many patients with aortic valve lesions and associated myocardial damage present the Adams-Stokes attacks.

**TREATMENT.**—Although Adams and Stokes discovered the syndrome only recent investigation has produced any satisfactory results as far as treatment is concerned. Levine (34) remarked that the use of digitalis would be of benefit in congestive heart failure with these attacks. The circulation is improved with this medication.

In the prevention of the Adams-Stokes attacks, general measures should be used to increase the heart rate and to make the ventricular contractions more forceful. Levine (34) upon clinical investigation with several co-workers suggests adrenaline as an excellent drug for initiating a ventricular beat, but it must be given by intracardiac injection. He has found that it is of little value in preventing attacks recurring; however, Cohn and Levine (10) found BaCl₂ to be of marked value because it increases ventricular irritability and if
A-V block is present then some other part of the ventricle will set itself up as the new pacemaker. They suggest a dosage of 30 mg. four times a day orally and .5 cc. adrenaline chloride one-half hour later intramuscularly. Excellent results were reported on several cases by these authors with the use of BaCl₂. It was found that adrenaline alone wears off quite rapidly although the patient is free from fainting attacks for a period of time. Horine (31) verified Cohn and Levine's findings with the use of BaCl₂ by reporting several cases which responded satisfactorily to this drug. Weiss (68) on the other hand was very much disappointed with the results derived from the use of this drug.

Schwartz and Jezer (51) reported two cases of complete A-V dissociation who were regularly with fibrillation to intravenous use of quinidine. This drug's activity was previously reported by Dock (12) who found a diminished tendency to fibrillation by decreasing the irritability of the ventricles. The use of quinidine is a very controversial subject at present, Borg and Johnson (7).

Several other drugs have been investigated as to their effect in these cases. Atropine has been used rather extensively in large therapeutic doses of 2-4 mg. (1/30-1/15 grains) to raise the ventricular rate, but such doses have an undesirable side effect. It has been found rather
useless in its effects on Adams-Stokes attacks. Thyroid .1 gm. (1½ grains) has been administered to many patients with varying results. The rationale of thyroid is to increase the B.M.R., indirectly affecting the heart rate and increasing the output, thus, reducing the cerebral anemia. It, however, is one of the numerous controversial drugs from the standpoint of efficacy.

One of the most beneficial drugs used, namely: ephedrine, has become more and more popular of recent years. The dosage is 20-30 mg. (1/3-1/2 grain) given orally three or four times a day. Ephedrine increases the excitability of the myocardium; hence, in case of a shift of the pacemaker, the ventricle instantly takes up a regular idioventricular rhythm. In rare instances, ephedrine may abolish the block.

At the present time, the tendency seems to be toward the daily use of either epinephrine or ephedrine until such time that some other drug appears more advantageous.

SYNCOPE OF ABNORMAL CARDIAC RHYTHM

This is a syndrome of syncopal attacks based on sudden abnormalities of rhythm. The chief types are:

1. Paroxysmal tachycardia.
2. Auricular flutter.
3. Auricular fibrillation.
4. Ventricular fibrillation, flutter and standstill.

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It would be impossible to take up a complete discussion of the various cardiac arrhythmias. The only reason they are mentioned in this paper is due to the fact that occasional attacks of syncope are produced from such arrhythmias. Let it be sufficient to say that because of some intrinsic disturbance in the conductive mechanism of the heart an abnormal rhythm is established, oftentimes, resulting in dizziness and syncope. They must be considered in a differential diagnosis of syncopal attacks of cardiac origin.

**Paroxysmal Tachycardia.**

This is a clinical term indicating a sudden heart rate increase from normal to around 160-200 beats per minute and lasting for seconds, minutes, or hours before suddenly ceasing. The patient is usually conscious of the new cardiac rhythm, feels a fluttering sensation in the chest, is conscious of the racing heart, and may feel exhausted and prostrated afterwards. Some cases may show no ill effects at all, whereas, in others the onset is accompanied by syncope and falling down. The syncope is produced by the small amount of blood discharged by the heart during the seizure. The physical examination should show the heart rapidity as the chief diagnostic point. The sudden fall in arterial pressure produces a cerebral anemia accompanied by weakness, vertigo, and
Willius (74) describes a typical case of paroxysmal
tachycardia occurring in children. All typical changes
were in evidence. Treatment by means of quinidine sulfate
6-7 grains in twenty-four hours was attempted, but success
was not apparent. Willius and Amberg (76) in conjunction
reported another case of paroxysmal tachycardia in a child.
Quinidine sulfate was again tried and was found to be
only temporarily successful. The importance of these
cases in children is emphasized by Willius and Amberg
when they state, "paroxysmal tachycardia is unusual in
children," especially those producing syncopal attacks.
In all available literature only twenty-five cases have
been previously reported and all of these were not verified.

Horine (31) mentions paroxysmal tachycardia as an
occasional etiological factor of syncope. Firm pressure
over the carotid sinus is often efficacious in producing
a cessation of such an attack, but quinidine sulfate is much
more reliable. Excessive digitalization in congestive heart
failure cases infrequently causes a patient to experience
syncopal attacks.

Herrman (30) suggests several methods of treating
tachycardia attacks. Carotid sinus pressure is, according
to Herrman, fifty per cent effective. Drugs such as opiates
are sometimes of value. Acetyl methyl choline (Mecholyl
-80-
Merck) is efficacious in a few cases. It has a tendency to make the carotid sinus reflex more sensitive. Weiss (68) adds ocular pressure to this list in the matter of preventing tachycardial attacks.

AURICULAR FLUTTER.

This is a clinical manifestation indicating a rapid auricular rate reaching 200-300 beats per minute. As a result the ventricular contractions at the onset are so weak the patient loses consciousness. An attack may be sudden or brief and with no cardiac changes. This syndrome is much more common in later life, that is, in the period of degenerative heart disease. The syncopal attacks are due to a cerebral anemia because of weakened ventricular contractions. Usually these patients complain of vertigo and weakness with sudden syncopal attacks following emotional influences or physical exertion. Atropine in 1/50 grain doses subcutaneously have proved of some value in the treatment of these conditions.

AURICULAR FIBRILLATION.

This is a condition consisting of a complete absence of normal auricular systole, the auricles in diastole undergoing a series of grossly irregular quiverings. The auricular activity rate varies between 350-600 beats per minute. The ventricles respond irregularly, but quite rapidly. Because of the irregularity syncope occasionally
develops in these cases of organic heart disease. The treatment is digitalization which serves as an adequate control for the syndrome. The prognosis depends on the underlying heart disease.

VENTRICULAR FLUTTER, FIBRILLATION AND STANDSTILL.

This condition causes a sudden instant syncope with an abrupt abolition of the pulse and heart sounds, sudden fall in blood pressure, dilatation of the pupils, pallor, and cyanosis. Unless the attacks can be interrupted immediately death results quickly. Physiologically the ventricle loses its synergic contractions, and becomes a mass of quivering ventricle. This lack of pumping action of the heart leads to cerebral and cardiac anoxia quickly with death. Both digitalis and quinidine have been held responsible for the ventricular condition, but there must be some pre-existing damage to the Purkinje system. A complete atrioventricular dissociation exists. Treatment consists of reestablishing the normal response and refractory period of the ventricle. If the syndrome is on a basis other than organic in nature, quinidine is excellent for causing a cessation of such attacks, but, if an organic lesion exists intracardiac injection of adrenaline may be administered. Epinephrine's effect in such conditions is controversial; however, because, some authors report no effect. It is a good drug to prevent
recurrent attacks nevertheless. $\text{BaCl}_2$ and ephedrine have become more and more popular in the treatment of the arrhythmias during the past few years. The discussion of treatment in the chapter on the Adams-Stokes syndrome gives a more complete description of the actions and uses of these two drugs.

In certain cases of ventricular arrhythmia with sudden standstill, a sudden precordial blow is indicated to initiate an impulse.

In concluding the chapter on the Syncopal Attacks, due to Cardiac Arrhythmias, it is necessary to point out again that it was my purpose only to mention briefly the possible manifestations producing syncope in this group. The capacity of realizing that syncope may be due to an arrhythmia is the salient point to be remembered here.
CONCLUSION

A discussion of the various types of neurogenic and cardiac syncope has just been completed with the idea of presenting, in a rather compact form, the various syndromes manifested. It is not the purpose here to include a discussion of all types of syncope, primarily because of the magnitude of the subject.

A simple classification has been presented which should prove advantageous in quickly differentiating the various types of syncopal attacks on a cardiac or neurogenic basis.

In reviewing the literature on neurogenic and cardiac syncope, I find it to be a rather limited subject. Great quantities of material have been written, for example, about the carotid sinus syndrome, hypotension and the like, but very few have gone to the trouble of forming a synopsis type of differential diagnosis. One is obliged to search and search deeply for important facts from a mass of literary rubbish for such a purpose.

In this thesis, the following topics have been discussed: The vasovagal, vago-vagal, postural hypotension, Gower's, Nothnagel's carotid sinus, Adams-Stokes, and cardiac arrhythmia syndromes. In each case the etiology, symptomatology, physiopathology, diagnosis, and treatment are taken up in considerable detail. In each case
the important characteristic diagnosis, although, some syndromes are of questionable accuracy at the present time.

Because of the sudden call for immediate treatment and diagnosis in most of these cases, a physician may be led into the trap of diagnosing one of these situations as an example of petit mal or grand mal epileptic seizures. This has proved to be the circumstance far too often and because of this it is my belief a deeper study should be made into cardiac and neurogenic syncope than is undertaken by most physicians so as to enable them to meet the situation adequately.
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