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THE JOURNAL OF THE ALLENTOWN HOSPITAL is published each February, May, August, and November by the Staff and Trustees of the Allentown Hospital. Original papers are invited from all members of the Staff and will be reviewed for publication by the Editorial Committee. Papers submitted must be typewritten, double-spaced, on one side of the page only. Illustrations must be original drawings or black and white glossy photographs. References should be listed alphabetically and should conform to the QUARTERLY CUMULATIVE INDEX MEDICUS: author's name and initials, journal, volume, initial pages and year. Papers should be submitted to the chairman of the committee.

ELECTROCARDIOGRAPHY

ROBERT JAMES TURNBACH, M.D.

IN a general hospital, many of the electrocardiograms are from patients on the surgical services. Some of these are part of a routine pre-operative general study of older patients, while many of them are from individuals known or suspected of having cardiac disease. The surgeon is not required to have a detailed knowledge of electrocardiography and most of the description of an electrocardiographic study will hardly need his perusal. However, each physician should be familiar with the significance of the terms used in the conclusion or final diagnosis at the end of the electrocardiographic interpretation.

The electrocardiogram is a record resulting from the electrical changes occuring in the activation and recovery of the heart muscle. Because of this, electrocardiographic changes may occur due to any factor altering the state of these processes in the muscle. Some of these conditions are interference with the blood supply in coronary artery disease, bacteria or their toxins, other toxic influences, acute rheumatic fever, variations of the blood electrolytes, endocrine abnormalities, and medications. Increase in the size of a chamber of the heart will frequently be diagnosed.

Although the record is produced by electrical activity of the heart muscle, the sequence and timing of the waves produced by this activity provide us with an accurate method of determining the state of the specialized conducting tissues. These tissues are responsible for the origin of the beat within the auricles and conduction of the stimulating impulse past the fibrous junction separating the auricular and ventricular muscle and its rapid spread to the inner surfaces of both ventricles. A very small area of damage in this specialized tissue may have profound effects both clinically and on the relationships of the waves of the electrocardiogram. A much larger lesion in a muscle not adjacent to these tissues may be unnoticed clinically or on the electrocardiogram. In most instances, however, damage to the conducting system is simultaneous with damage to heart muscle in other areas. Abnormal origin or abnormal conduction, or both, may be present.

Some of the more common conditions will be briefly mentioned:

Normal sinus rhythm, sinus tachycardia, and sinus bradycardia are terms used to describe the auricular rate when the impulses are arising in a normal manner. In sinus arrhythmia, the impulses are arising in a normal manner, but the rate is varying through the influence

of nervous impulses affecting the sinus node. Wandering pacemaker within the sinus node and wandering pacemaker to the atrio-ventricular node imply changing vagal effect and are not of clinical importance. The above mechanisms are physiologic.

Premature contractions arise in areas of increased irritability outside the normal pacemaker. Isolated premature contractions are extremely common without heart disease. However, in the presence of coronary artery disease premature ventricular contractions cannot be disregarded. Frequent premature auricular contractions may precede auricular fibrillation. Premature ventricular contractions from varying foci require close study of the patient's cardiac status for heart disease is usually present and serious ventricular arrhythmias may follow. Coupled premature ventricular contractions may be the effect of digitalis overdosage. Short runs of premature ventricular contractions may precede a prolonged attack of dangerous ventricular tachycardia and medication should be instituted to prevent it. Ectopic contractions occuring after a long pause are escape beats and are physiologic protective mechanisms.

In auricular fibrillation and auricular flutter, there is always some part of the auricles in a state of activity, usually with only a reduced number of the impulses reaching the ventricles. Organic heart disease or hyperthyroidism is almost always present and the presence of either arrhythmia requires serious consideration before deciding upon surgery. Paroxysmal auricular and paroxysmal nodal tachycardia are both called supra-ventricular tachycardias and frequently cannot be differentiated from each other. Differentiation is usually not important as both respond to the same treatment. In both cases, the impulses are arising at a rapid regular rate and the ventricles usually have the same rapid rate. They are very frequently present without heart disease. In some instances, hyperthyroidism may be present. The attack may stop spontaneously or in most instances, may be terminated quite easily by treatment. Some of the attacks may be more resistant. Following an attack, it is well to keep the patient under medication to prevent recurrence during surgery.

Paroxysmal ventricular tachycardia is due to digitalis intoxication or very serious heart disease, usually coronary disease, and it demands immediate measures for ventricular fibrillation and death frequently follows.

Atrio-ventricular heart block, whether complete or partial may be due to an acute process now present or it may be the result of damage done at some previous time. Any acute process causing atrio-ventricular

heart block would be a contra-indication to surgery. If no acute process is present, the necessity of the contemplated operation may dictate the course of action. Occasionally partial heart block, as shown by a prolonged P-R interval, may be found even in young individuals who never demonstrate any evidence of heart disease. In any instance, before deciding that this is true the patient should be studied for a period of ime.

Bundle branch block is frequently seen on electrocardiograms. As a whole, conditions which cause enlargement of the left ventricle may cause left bundle branch block, and conditions which cause enlargement of the right ventricle may cause right bundle branch block. However, other conditions may be responsible. Right bundle branch block may be due to acute cor pulmonale (usually due to pulmonary embolism). Myocardial infarction may cause right or left bundle branch block and in the presence of bundle branch block it may be difficult or impossible to diagnose myocardial infarction on the electrocardiogram. The individual with bundle branch block may have a moderate or marked amount of heart disease and every case will require individual evaluation. As a whole, bundle branch block is much less dangerous than partial or complete atrio-ventricular block. For with partial block there is always danger of complete block occurring with complete cessation of ventricular activity or ventricular fibrillation. Where the electrocardiogram already shows complete atrio-ventricular block it is always possible that there will be a return to normal conduction followed again by complete block with cessation of all ventricular activity.

Wolff-Parkinson-White syndrome (short P-R, wide QRS) is a relatively infrequent diagnosis. These individuals are prone to attacks of paroxysmal auricular tachycardia. Formerly it was believed that this condition was benign. It is now evident that, at least in some cases, heart disease is present.

Although for years the value of the electrocardiogram was mostly in the diagnosis of arryhthmias, increased knowledge has led to a vastly increased use in the study of the muscle of the heart as a whole, or the muscle of a particular chamber, or even the tissue in localized areas.

Chronic cor pulmonale indicates the patient's heart has been under increased strain from his pulmonary condition and his right heart has become enlarged. An electrocardiographic diagnosis of chronic cor pulmonale or ventricular enlargment or ventricular strain requires further cardiac evaluation of the patient before operation. Although

these patients may have no history or physical signs of congestive failure, under the stress of operation and at times with intravenous therapy following operation sudden congestive failure may occur. A period of treatment before the operation with care during and after surgery may prevent catastrophe.

Acute cor pulmonale is usually due to pulmonary embolism. In a suspected case of pulmonary embolism, an electrocardiogram should be taken as soon as possible for the electrocardiographic signs may disappear rapidly. Early and frequently repeated electrocardiograms may be an aid in this diagnosis.

Recent myocardial infarction is of course a contra-indication to all surgery. If the possibility of recent myocardial infarction is suspected clinically the absence of positive findings on the electrocardiogram should not be consisdered as sufficient evidence that no infarction has occurred. Repeated electrocardiograms should then be obtained over a period of weeks for although the evidence may appear within less than an hour following the event, in some instances a prolonged period may lapse before definite changes occur on the electrocardiogram. This is especially true where other abnormalities are already present such as ventricular strain or bundle branch block.

The electrocardiographic changes due to a decreased flow of blood through the coronary arteries are variable and frequently not specific. As previously mentioned, the electrocardiogram is a record of electrical variations occuring during the activation and recovery of the heart muscle and, with decreased coronary flow, there may not be sufficient changes in the muscle to cause electrocardiographic abnormalities while the patient is at rest. Even with an abnormal electrocardiogram, the history may be far more important for it may suggest the probability of an on-coming infarction much more clearly then the tracing.

A diagnosis of old myocardial infarction necessitates ths study of the patient's whole cardiac status before surgery is attempted.

In acute or chronic pericarditis, the electrocardiographic pattern is due to the involvement of the cardiac muscle immediately beneath the pericardium. Fluid may be present in the pericardial sac without producing the changes of pericarditis. Here the fluid acts as an electrical shunt about the heart and reduces the amplitude of all the complexes.

Decreased or increased potassium is not infrequent in hospital electrocardiography. While decreased potassium may be due to treatment of diabetic coma or may occur in renal disease and other conditions, it is frequently associated with surgery of the gastro-intestinal tract and

may be related to prolonged intravenous feedings. Here the electrocardiogram cannot well be used for any exact quantative measure but it may be helpful in following treatment. A diagnosis of decreased calcium is much less frequent and is at times seen with disturbed parathyroid function or with renal disease.

Mention of digitalis effect does not mean that the patient has received toxic doses of this drug. However, in the presence of known digitalis therapy or digitalis effect on the RS-T segments, the presence of coupled premature ventricular contractions, any degree of atrio-ventricular heart block, or auricular arrest should be considered as due to digitalis intoxication. The presence of rapid heart action does not mean that digitalis intoxication is not present. Quinidine may be responsible for changes in the electrocardiogram. Unless it is known that the patient is receiving quinidine, an erroneous interpretation of the electrocardiogram may frequently result. Patients who are receiving large doses of quinidine should be followed carefully with electrocardiograms.

Chronic anemia, avitaminosis, hypothyroidism, accumulation of fluid in the body in general and especially in the chest, dehydration, bacteria and their toxins, other toxins, trichinosis, amyloidosis, leukemia, and allergic reactions all may produce changes in the tracing and are frequently difficult to evaluate without some additional knowledge of the patient. At times abnormalities have been present due to intrathoracic tumors. Shock, hemorrhage, and carbon monoxide poisoning often give electrocardiographic changes which are not pathognomonic. It will be apparent that in many instances the electrocardiogram will frequently show only non specific abnormal changes and further study will be indicated to determine the etiology.

A normal electrocardiogram may be present in organic disease of the heart or the blood vessels. Valvular deformities in themselves usually produce no abnormality in the electrocardiogram until there is sufficient muscle change to affect the tracing. This is also true of septal defects unless they involve the specialized conducting tissue. Arterial aneurysms produce no electrocardiographic disturbances. Arterio-venous communications will affect the electrocardiogram only through increase in the work of the heart.

In conclusion, it is well to remember than an abnormal electrocardiogram frequently will not indicate the severity of the heart disease and may not even be due to organic heart disease, and a normal electrocardiogram is not synonomous with a normal heart.

THE CHRONIC LEG ULCER

GEORGE BOYER, M.D.

A detailed description of such a well-known entity as the chronic leg or "stasis" ulcer is hardly required, since every physician has seen, if not treated, this common condition. The discouraging feature of this ulcer is its failure to heal with local treatment or its temporary healing only to break down again and again. Its chronic nature has caused many a physician to greet a patient with a leg ulcer with the same enthusiasm usually accorded the patient complaining of headache or backache. The purpose of this paper is to outline some of the old and newer treatments that may heal and keep healed these indolent lesions.

The term "stasis" ulcer implies association with faulty return of blood from the extremity. In practice this vascular defect occurs almost entirely under two conditions:

1. Phlebitis, followed by the post-phlebitic syndrome.
2. Varicose veins.

Why an ulcer develops as a result of these blood vessel abnormalities, and why it chooses the typical location above either malleolus, cannot easily be explained. An anatomic explanation relative to richness of blood supply of the ankle area is not valid, since the skin immediately over the malleoli has the poorest blood supply and should, therefore, be the most likely site for an ulcer to develop. However, the typical location of these ulcers is not directly over the malleoli, but rather in the area just above (fig.1). Injury as a cause of the ulcer is at best only a precipitating factor. Fungus infection (the ever-present "athlete's foot") is now generally regarded as an important factor in promoting the chronic nature of these ulcers; but it, too, offers no explanation as to the favorite anatomic site of development.

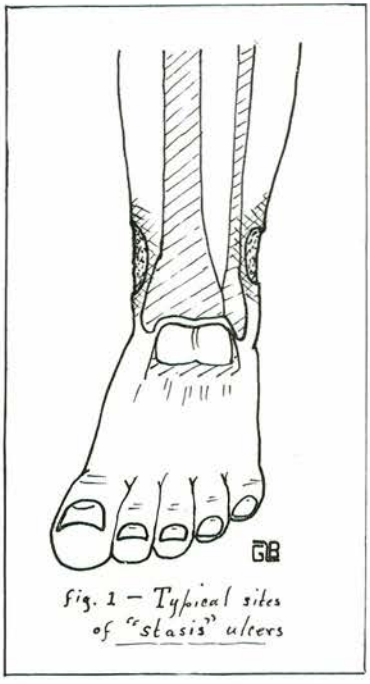


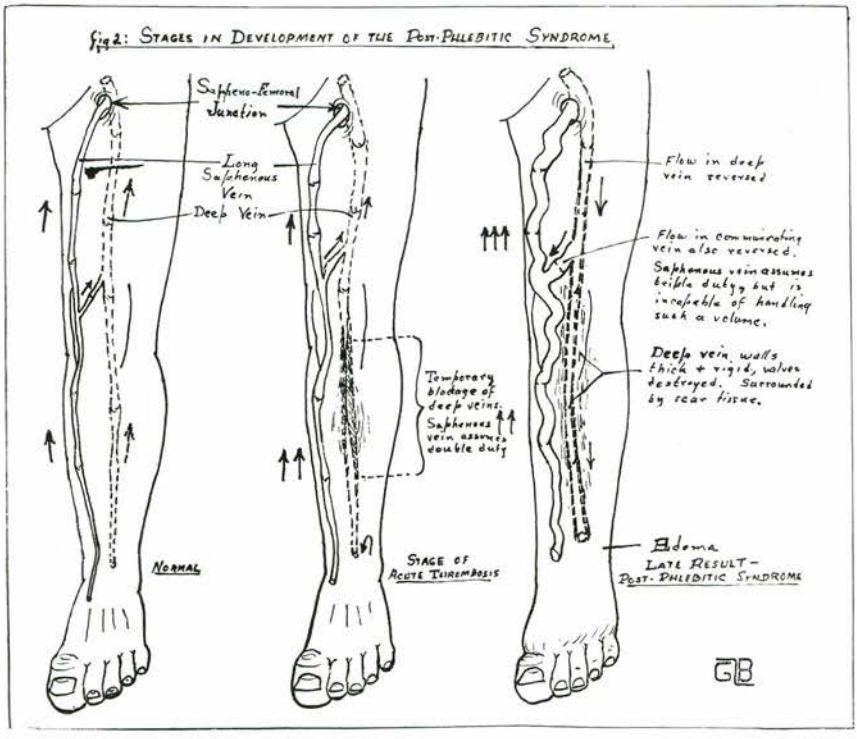
fig. 1 — Typical sites of "stasis" ulcers

When confronted with the problem of treatment, one should first determine if the given ulcer is of post-phlebitic or of varicose vein origin. A history of a "milk leg" associated with a pregnancy, or phlebitis following operation, or a prolonged

illness requiring bed rest, labels the ulcer as post-phlebitic. It should be stressed that some of these episodes may not have been recognized at the time, and therefore a carfeul inquiry as to the onset of edema of an extremity as well as questioning about post-operative complications is required. The gross appearance of the ulcer itself gives no clue as to its cause and the entire extremity must be examined for other signs as listed below:

ASSOCIATED PHYSICAL FINDINGS OF AN ULCER-BEARING EXTREMITY

Post-phlebitic	Varicose veins
1. Presence of a "milk leg".	1. Edema minimal or absent.
2. Edema of lesser degree, unilateral.	2. Superficial veins prominent by inspection or by palpation.
3. Superficial veins not prominent.	3. Tourniquet tests.



Blood serology and blood sugar determinations should be done in every case, although the incidence of "stasis" ulcer from syphilis or diabetes is very small. The tests devised for varicose vein evaluation are legion and often confusing. Likewise, the theory that varicose veins represent a compensation for deep vein obstruction (i. e. blood

returning from the foot has two roads to travel; if one be blocked, then the blood must necessarily follow the other route) is not entirely valid, since the varicose vein problem is not nearly so simple as that. Although immediately following a deep phlebitis the deep veins of the lower extremity may be occluded, this condition does not persist long, and is *not* the explanation of the resulting edematous limb. Sooner or later nature recanalizes the diseased vessel. All would then be well except that the resulting vessel has lost many of the characteristics of a good vein (viz. rigid walls lying in a bed of scar tissue have replaced the normally elastic walls lying in a pliable bed, and the series of valves has been destroyed). Thus the deep flow of venous blood is through a rigid tube, uninfluenced by surrounding muscular "milking" action, and with no valves to prevent backflow. Deep vein backflow, then with resultant pooling of blood distally, is the mechanism at work in the post-phlebitic syndrome (fig. 3). The compensatory development of varicose veins, which may occur in such a situation, does not indicate deep blockage, but rather that blood traffic over the saphenous system (a "secondary road") has become excessive.

With the currently widespread use of heparin, dicumarol, and more recently, tromexan, the question arises, what influence will the use of these anti-coagulants have on the post-phlebitic syndrome? If, by their action, extention of the disease process along the course of the vein and its tributaries is prevented; the sequallae should correspondingly be lessened.

The development of venography (x-ray visualization of the lower extremity vein pattern by injection of diodrast - fig.3) is believed by many peripheral vascular disease authorities to make the complicated battery of tourniquet tests unnecessary. The

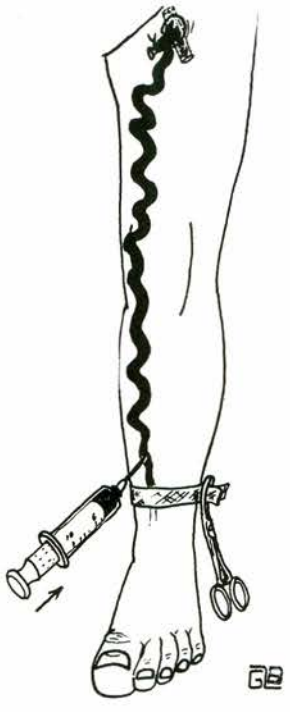


Fig. 3: VENOGRAM (Patient in Trendelenberg position)

In varicose veins, to show incompetent, "communicators", or dilated branches at Sapheno - Femoral junction missed at prior operation, 10cc of Diodrast injected **above** a tourniquet (illustrated).

In post-phlebitic syndrome to show competency of deep veins, 10cc of Diedrast in-jected **below** tourniquet (not illustrated).

Long leg X-ray films are taken in 3 to 5 seconds.

principle of this test is based on the assumption that dye injected distally will follow the venous path of least resistance, and is identical with the

backflow pattern which exists in varicose veins. By means of venography one can demonstrate whether or not the deep circulation is intact and locate the presence of incompetent communicating branches between the deep and superficial veins.

TREATMENT

After determining the probable cause of a given ulcer, the actual treatment may be started:

1. Post-phlebitic ulcer.
   a. Treatment of fungus infection (done in every case, even if classical interdigital lesions are not seen.)
      (1) $KMnO_4$ soaks in acute stage: ½ hour daily, feet and legs, 1:8000 solution.
      (2) Gentian violet (aqueous) 2% in acute stage: Paint feet and legs daily to weekly as infection subsides.
      (3) "Castaderm" (The Lannett Company, Inc.): Weekly or twice weekly, applied as in (2) above, for chronic infections.
   b. External support (To counteract diffusion of stasis blood through vessels producing edema).
      (1) Elastic bandages or stockings.
      (2) The time-proved Unna boot.
   c. Sympathectomy.
   d. Excision of ulcer.
   e. Skin graft.

2. Ulcer associated with varicose veins.
   a. Treatment of fungus infection as above.
   b. Saphenous vein ligation (high), additional ligations as indicated by venography.
   c. Unna boot, elastic support.
   d. Excision of ulcer.
   e. Skin graft.

It is rather startling to observe that one of the conditions which lumbar sympathectomy greatly benefits is the post-phlebitic syndrome —a venous rather than an arterial defect. It is easier to understand how sympathectomy may aid the healing of a stasis ulcer in an individual who shows excessive sweating of his extremities, and this symptom is, indeed, a definite indication for the operation.

The Unna boot is still one of the best single things to use for healing an ulcer even though recurrence is frequent following removal of the boot. The boot is worn from one to three weeks before being re-applied.

The foot and leg are then washed (or better, scrubbed) with one of the new antiseptic soaps (e. g. "Gamophen") removing all previously applied paint. Examination and evaluation of the skin then dictates continuation, variation, or cessation of treatment, depending upon the results. Circular support and a "smoothing" effect on the ulcer and its surrounding skin is obtained by the use of elastic bandages and stockings and by direct adhesive strapping over the ulcer.

Lumbar sympathectomy by enhancing blood flow in general and eliminating co-existing vascular spasm, promotes the development of collateral circulation and allows a more efficient venous return with subsidence of edema. Few ulcers will remain healed by the above conservative treatment alone even if treatment be continuous. Should treatment be interrupted, recurrence of the ulcer is almost inevitable. This chronic character of the leg ulcer, together with the necessity for continued, uninterrupted treatment, must be explained to the patient, stressing particularly the need for constant support (elastic bandage, stocking, or Unna boot). Most patients will elect to try conservative treatment rather than to have an early lumbar sympathectomy. A trial of conservative treatment for perhaps two to six months is within reason, before again suggesting operation, unless the patient voluntarily requests it.

Following sympathectomy, some post-phlebitic ulcers with little surrounding scar tissue may heal and stay healed. Most, however, require wide excision followed by grafting. The excision includes not only the ulcer, but all of the surrounding pigmented and indurated skin and subcutaneous tissue down to red muscle or bone. A split-thickness skin graft, either in one sheet or in "postage stamp" sections is applied to the excised ulcer bed when healthy red granulations have developed. The triple procedure (sympathectomy—excision—grafting) requires an average hospitalization of three weeks.

In treating the varicose ulcer, saphenous vein ligation prepares the extremity and the keynote of success is a painstaking thorough search for all branches entering the saphenous vein near its junction with the femoral vein. By failing to ligate one small branch of the saphenous vein the procedure may be a failure when that tributary enlarges and takes over the function of the saphenous vein itself. Occasionally the short saphenous vein may require ligation in the popliteal space. Any demonstrable incompetent communicating branches also require ligation. When this latter condition appears extensive, a sub-fascial dissection of the leg has been used to insure complete elimination of all faulty communicators. Varicose ulcers, if old, may require excision and grafting in the same manner as described for post-phlebitic ulcers.

CEREBRAL PALSY

FORREST G. MOYER, M.D., F.A.A.P.

THE term infantile cerebral palsy, like that of Little's disease, loosely defines a series of clinical syndromes and does not describe a disease. The cerebral palsies represent a group of conditions which affect the control of the voluntary muscle systems and have their origin in lesions of the motor centers of the brain. They are characterized by paralysis, weakness, incoordination, or other alterations of motor function.

Cerebral palsy is a more frequent cause of crippling than is poliomyelitis. Recent figures indicate that its incidence is about 1 in 200 births. Its prevalence in the population is approximately 450 per 100,000. The number of cases in the United States has been estimated to be between 150,000 and 200,000. There is a steady increment of cases and this holds true for the rural and city areas. The incidence in Lehigh County is not known, but 106 children have been seen at the Cerebral Palsy Clinic at the Allentown General Hospital. This clinic has been in operation since February, 1950 and carries a case load of 73 patients at the present time.

It has been estimated that there are seven children per 100,000 of population born each year with cerebral palsy. Since the mortality rate during infancy is about one seventh of the total number, there are actually about six new cases of cerebral palsy each year per 100,000 population. Two of the six will be feebleminded. Four will have normal mentalities. One of these will be severe and homebound. One will be so mild as to require no treatment. Two will be of moderate degree and capable of considerable improvement.

Two main problems arise in the care of cerebral palsies. The first is the severe case in the mentally normal child who is homebound and must either be placed in an institution for physical improvement and education or must be taught at home. The second is the care of the moderately severe patients who are capable of going to a public school or a school for the handicapped and who need regular treatment for years. Mental deficiency takes precedence over crippling and such children should be placed in institutions for the mentally defective whether they are crippled or not. Present facilities for the care of cerebral palsy are too limited to care for the child who is mentally retarded.

For many years the treatment of the cerebral palsied child has been neglected and all such cases were thought to be spastic and feebleminded. Now careful classification and differentiation allows a great deal to be done. More and more centers are being established, many through the efforts of lay groups composed largely of parents of palsied

children. The lack of facilities is partly due to the fact that cerebral palsy is a multiple defect, often associated with deafness, blindness, speech defects, convulsive disorders, personality and intelligence changes as well as motor defects. It is, therefore, too comprehensive a condition to be treated by a single physician but can best be handled by a group of coordinated specialists. The Lehigh County Crippled Children's Society is an example of such an organization born at the behest of parents, supervised by public spirited citizens, and supported by public contributions. There has been a tremendous increase in research, care and rehabilitation for these cases.

There are five main types of cerebral palsy: spasticity, athetosis, rigidity, tremor and ataxia. Each type represents a specific treatment problem. The spastic child has had damage or maldevelopment of the motor cortex, internal capsule or some portion of the pyramidal tract system. Muscles are affected in groups and the resulting paralysis may be spastic, flaccid or both. The athetoid's difficulty is due to a defect in the basal ganglia resulting in impairment of the proper selection of impulses. Thus a fantastic number of impulses may reach the skeletal muscles, causing all sorts of bizarre and involuntary motions. This motion, the cardinal sign identifying an athetoid, is unpredictable, irregular and unrhythmic. Rigidity is thought to be due to a diffuse brain involvement and is characterized by lead pipe resistance in one or more muscle groups. Ataxia is due to cerebellar dysfunction and is characterized by incoordination and lack of balance. Tremor is due to involvement of the red nucleus and is manifested by rhythmic flexor-extensor motions. Spastic and athetoid cases make up 90% of all cases of cerebral palsy.

There are some factors which predispose to the occurrence of cerebral palsy. It is more common in premature infants. It is more common among first-born children and those with heavy birthweights where prolonged labor is more frequent. It occurs more frequently in children born to older women who have heavier babies as a rule, and in boys, who weigh more than girls on the average. Cerebral palsy is more frequent among the white than the colored.

The two most important causes of brain injury are anoxia and cerebral hemorrhage. Anoxia, whether pre or postnatal, tends to involve the basal nuclear regions and/or other extraphramidal areas of the brain so that the clinical picture of athetosis, tremor, rigidity or other extra-pyramidal symptoms may result. Cerebral hemorrhages on the other hand are generally submeningeal or intracerebral in the region of the internal capsule. Thus they primarily involve the pyramidal tracts and result in a clinical picture characterized by spasticity and pyramidal tract signs. In cerebral palsy there is predominantly or entirely either signs of pyramidal tract or of extrapyramidal tract involvement.

Mixed lesions are not so common. This is due in part to the fact that when there is extensive involvement of both systems, the damage is usually incompatible with life.

The factors which may precipitate damage to the brain may be divided into (1) those which occur in the prenatal period from the time of conception to the onset of labor, (2) those which operate in the natal period from the onset of labor to the time of viability of the new born infant, and finally (3) those which operate in the postnatal period from the time that the child is viable throughout the rest of its life.

The prenatal causes of cerebral palsy may be hereditary or congenitally acquired in utero. Among the hereditary causes are such diseases as spastic paraplegia, familial athetosis, congenital tremors, tuberous sclerosis, neurofibromatosis, Sturge-Weber syndrome, Tay-Sachs' disease, hepato-lenticular degeneration and many others. Anoxia, cerebral hemorrhage, metabolic disturbances, infection and the Rh factor may affect the fetal brain and thus give rise to the congenitally acquired cerebral palsy.

Anything that interferes with placental circulation is a potential cause of anoxia. Compression of the umbilical cord between the head and bony pelvis or by kinks could prevent an adequate blood flow to the brain. Threatened abortions or bleeding during pregnancy, premature separation or infarcts of the placenta are frequently encountered among mothers giving birth to cerebral palsied children. Decreased maternal blood pressure may be a factor.

Fetal cerebral hemorrhage may be the result of fetal anoxia, direct trauma to the fetus or uteris, or to the maternal toxemia of pregnancy. Anoxia may cause direct damage to the vessels or cause cerebral softening with secondary hemorrhage. Direct trauma is rare. Toxemia of pregnancy may cause spasm of the fetal vessels with death of brain tissue and secondary hemorrhage.

Any maternal infection may involve the fetal brain. Since the rate of development of the fetal brain appears to be greatest between the second and fourth months of fetal life, maternal infection during the first trimester of pregnancy is associated with a higher incidence of prenatal encephalitis. The fetal brain is very susceptible to viral infection, and therefore German measles in particular and to a lesser extent herpes zoster, mumps, chickenpox, measles and other viral infections may cause cerebral palsy. Syphilis and toxoplasmosis may also cause brain damage.

Maternal diabetes may cause damage to the fetal brain. This is thought to be due to maternal hypoglycemia. Maternal liver damage may also cause hypoglycemic states. Maternal nutrition and vitamin

deficiencies may be factors. They do not cause damage often because the baby is a parasite and will not show the effects of the mother's poor nutrition unless it is extreme.

The Rh factor accounts for eight per cent of all cases of cerebral palsy seen. An Rh negative mother who has been sensitized by a transfusion of Rh positive blood or by an Rh positive fetus will produce anti-Rh positive agglutinins. Subsequent pregnancies will be affected by this sensitization. Fortunately not all Rh negative women produce such antibodies. When they are produced, they circulate in the fetal circulation and begin to destroy the fetal blood. The infant is born with anemia, severe jaundice, and exhaustion of his blood forming organs. Many immature blood cells or erythroblasts are present and give the name erythroblastosis fetalis to this disease. Such children may have severe brain damage. Some die, some recover completely, and a certain proportion are left with a typical form of athetosis often associated with deafness and paralysis of eye muscles. The treatment for erythroblastosis fetalis is exchange transfusion. Dr. Diamond of Boston feels that such a transfusion, given early, may prevent the occurence of kernicterus which results in cerebral palsy. This view is not shared by everyone, but exchange transfusion remains the most effective treatment we have to offer. Any infant who develops jaundice in the first 24 hours after birth should be considered to have erythroblastosis until proven otherwise. Indications for exchange transfusion are not within the scope of this paper.

Anoxia and vascular damage make up the factors which operate from the onset of labor to the time the baby is viaible. They are the most common causes of cerebral palsy.

Anoxia is the most important natal factor. The moment that the placenta separates from the uterus or the umbilical cord is clamped off, the child is dependent upon its own lungs. Mechanical blockage of the respiratory tract is one of the common causes of asphyxia. The injudicious use of analgesics and anesthesia in the mother during labor is an important cause of asphyxia. The respiratory center of the infant is much more sensitive to morphine than is that of an adult. Such babies may be blue because of poor respirations and circulation. Obstetrical anesthesia which causes maternal asphyxia may also asphyxiate the fetus. Spinal and caudal analgesia may cause a drop in maternal blood pressure sufficient to reduce seriously the oxygen supply to the infant. Anoxia is more common in breech deliveries, especially when there is a delay in the delivery of the head. Kinking or pinching of the cord during delivery may interfere with fetal blood supply and thus cause anoxia. Abruptio placenta and placenta previa are associated with a high incidence of blue babies, asphyxia, and cerebral palsy. Severe anemia in the child or mother may cause anoxia.

Most of the traumatic causes of brain injury at birth may be considered to be physiologic. The sutures and fontanelles of the infant skull allow the skull to conform to the shape of the birth canal. When the child is big or the pelvis small, these natural safeguards may be insufficient to protect the brain from injury, especially if the labor is hard and prolonged. Too strenuous application of forceps may cause trauma. Dr. Perlstein feels that trauma caused by obstetricians as distinguished from natural obstetrical trauma is probably responsible for less than 5 per cent of all cases of cerebral palsy. Sudden pressure changes can cause hemorrhages and other injuries to the brain. Such release of pressure may occur in precipitate deliveries or caesarean sections where there is insufficient time to allow for natural decompression. Premature babies have thinner blood vessel walls and hence are more prone to develop cerebral hemorrhage. Syphilis and blood dyscrasias may cause bleeding.

The postnatal causes of cerebral palsy are the least common of the factors producing such damage. Penetrating wounds and skull fractures may cause cerebral palsy. Meningitis, encephalitis, and brain abscesses are responsible for some cases. Lead poisoning often involves the brain, causing encephalopathy which usually results in spasticity. Children are subject to vascular accidents. The most common cause is the rupture of a congenital aneurysm of the cerebral vessels. Vascular embolism may result from endocarditis. Strangulation, drowning or carbon monoxide poisoning may cause anoxia.

There is a statistically significant correlation between certain etiologic factors and specific clinical syndromes. Dr. Perlstein has listed some of these in the following table.

Prematurity	Spastic paraplegia
Breech delivery	Athetoid or spastic paraplegia
Toxemia of pregnancy	Spastic hemiplegia or quadriplegia
Birth trauma	Spastic hemiplegia or quadriplegia
Anoxia	Athetosis
Rh factor and kernicterus	Athetosis with deafness and paralysis of upward gaze
Maternal rubella	Spasticity with deafness or auditory aphasia, cataracts and congenital heart disease
Precipitate or caesarean delivery	Spastic quadriplegia, ataxia, or rigidity
Placenta previa or abruptio	Athetosis

Although many infants who sustain brain damage exhibit unmistakable signs, some give only suggestive evidence. In general the following abnormalties, occurring in various combinations, suggest cerebral

injury: (1) undue apathy or restlessness; (2) otherwise unexplained pallor or cyanosis; (3) respirations more irregular than is customary for the newborn infant; (4) feeble whining cry often interspersed with a shrill cry; (5) poor nursing, disinterest in feedings, and impaired swallowing; (6) vomiting; (7) rapid pulse if shock is present, or slow pulse if the intracranial pressure is elevated; (8) unexplained variations in temperature; (9) disappearance of the Moro reflex which may reappear days later; (10) ocular signs including strabismus, nystagmus, dilation of the pupil; (11) bulging of the fontanelle (12) twitchings, tremors, convulsions, spasticity, opisthotonos. Spinal taps, pneumoencephalograms, and electroencephalograms are further aids in diagnosis. Diagnosis can be made in most cases before the age of six months and in almost all cases by one year. A knowledge of the normal development pattern is essential. Early diagnosis is important, since it enables one to initiate appropriate therapy and give a more accurate prognosis. Prognosis is influenced by the degree of severity of the palsy. The occurence of convulsions makes for a poor prognosis. The presence of mental deficiency makes reeducation and therapy difficult and often impossible.

The treatment of cerebral palsy is a complex problem which requires the combined skills of many people. For many years the orthopedists and surgeons were the only people interested. Since we are dealing with children, pediatric guidance is necessary for the proper nutritional and general developmental improvement of the cerebral palsied child. It is important to remember that the patient is first, a child; secondly, a handicapped child, and only thirdly, a cerebral palsied child. Both the parents and the child need help to adjust to this problem. The accepted formula for the treatment of these children is: physical therapy to help them walk correctly, occupational therapy to teach them to use their hands, speech therapy so that they can talk, and education to give them something to talk about. It is not enough to accomplish these things. The children must be given help and guidance to cope with their handicap and develop their personalities so that they may become acceptable and useful members of society.

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A CASE OF ACHALASIA OF THE ESOPHAGUS (CARDIOSPASM) CAUSED BY HYPERTROPHY OF PERI-ESOPHAGEAL DIAPHRAGMATIC MUSCLE SLIPS.

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ACHALASIA of the esophagus is a clinical syndrome characterized by dysphagia and regurgitation of food during or immediately after meals, for which no definite organic stricture, neoplasm, or other esophageal lesion can be demonstrated. As the disease progresses the esophagus gradually becomes enlarged and funnel-like (megaesophagus) and, in the later stages of the condition, may become S-shaped and tortuous.

The etiology of this condition is not definitely known. One theory ascribes it to the absence of the cells of Auberbach's plexus at the lower end of the esophagus. The lack of these cells interferes with the normal propulsive movements at the cardia. Another theory describes reflex spasm of the cardiac sphincter because of pathology in the upper gastro-intestinal or biliary tracts. Since the disease occurs predominantly in over-emotional and severely neurotic individuals, a psychosomatic cause for cardiospasm has been postulated. It is the purpose of this paper to describe a case of achalasia in which a definite etiological factor could be found at operation.

The cardiac sphincter is a physiological one. The muscular ring encircling the lower end of the esophagus is commonly known as the cardiac sphincter, although in man the thickness of the muscle in this situation is scarcely greater than the rest of the tube. The opening and closing of the cardia is brought about by a delicate balance of the autonomic nervous system, the parasympathetic elements being largely, but not entirely, inhibitory to the contraction of the sphincter, and the sympathetic elements, exitatory.[1]

A careful review of the literature since 1930 has disclosed only one reference in which spasm of the diaphragm is mentioned as a possible etiological factor.[2] No references were found describing hypertrophy of peri-esophageal muscle slips arising from the diaphragm as a constricting agent which would tend to cause the syndrome. Normally the lowest portion of the thoracic esophagus is surrounded anteriorly and laterally be slips of striated muscle arising from the diaphragm just cephalad to the esophageal hiatus. These muscle slips are relatively thin, although in several cadavers dissected by the author they were broad in extent, sometimes covering as much as two inches of the lowest thoracic esophagus. In the case to be described, these fibers were organized into three definite, thick bundles of muscle which could very easily have exerted enough extrinsic pressure upon the cardia to have caused the achalasia.

CASE REPORT

M.M., a 51 year old white female was admitted with the chief complaints of progressive substernal pain, nausea and vomiting immediately after the ingestion of food. During the three year period of her symptoms her weight decreased from 230 to 161 pounds. The condition became so severe that at admission no food at all could be swallowed. She had no other symptomatic complaints. Her past history did not disclose any serious illness.

Physical examination disclosed no abnormalities. Her blood pressure was 160/90. Esophagrams disclosed a markedly enlarged, funnel shaped esophagus characteristic of achalasia (Fig. 1). Blood chemistry studies and the blood count were normal. Urine analysis was normal. The Wassermann was 4 plus with 3 plus Kahn and Mazzini.

Esophagoscopy revealed a markedly dilated esophagus packed with fetid food particles. An alcoholic solution of a protein digestant (Caroid Powder) was administered in order to clean out the esophagus. Esophagoscopy was repeated and disclosed a slight degree of esophagitis. The instrument could not be passed into the stomach.

It was felt that because of the megaesophagus and the three-year history conservative management by bouginage would be both useless and dangerous. The patient was then prepared for surgery by esophageal suction and parenteral feedings. Exploratory thoracotomy was performed eight days after admission.

OPERATIVE PROCEDURE AND FINDINGS

The left hemithorax was entered through the bed of the 8th rib. The left pulmonary ligament was cut and the lung was retracted upward. The phrenic nerve was crushed, thus paralyzing the diaphragm. A longitudinal incision was made in the mediastinal pleura just anterior to the descending aorta. The esophagus was dissected from its bed and was found to be markedly enlarged. Just above the esophageal hiatus were seen three distinct bundles of muscle which appeared to represent abnormal hypertrophy of the diaphragmatic slips of muscle usually found at this location. Each of these bundles was as thick as a man's index finger and almost completely surrounded the circumference of the esophagus. Each bundle lay above the other in a cephalad direction and appeared to compress the lumen of the distal inch and a half of the thoracic esophagus. Thus a definite organic etiology for the patient's condition could be demonstrated. Each of these bundles was clamped and cut, thus relieving the obstruction. The diaphragm was incised radially from the hiatus and the peritoneal cavity was opened. An enlarged, extremely soft and friable spleen immediately protruded into the wound and was inadvertantly ruptured upon gentle palpation. Splenectomy was performed. The cardia of the stomach was pulled

into the chest and a longitudinal incision was made through it extending into the anterior wall of the esophagus. This wall was markedly hypertrophied and was at least one-quarter inch in thickness. The longitudinal incision then was sewed transversely after the method of Heineke and Mikulicz, thus considerably enlarging the diameter of the cardial-esophageal lumen. The diaphragm was closed snugly about the cardia, a water-seal drain was placed in the 9th interspace and the chest wall was closed in layers.

The patient's post-operative course was uneventful. She was ambulatory the first day. On the fourth day water by mouth was permitted. The fifth day, full liquid feedings were given. The sutures were removed on the seventh day. On the ninth day solids were given and were well tolerated. She was discharged on the eleventh day.

Her follow-up disclosed a very satisfactory result. Three weeks after surgery she could eat steak, potatoes or any other food without any discomfort whatsoever. Her previous symptoms had completely disappeared and meals became a source of real delight to her.

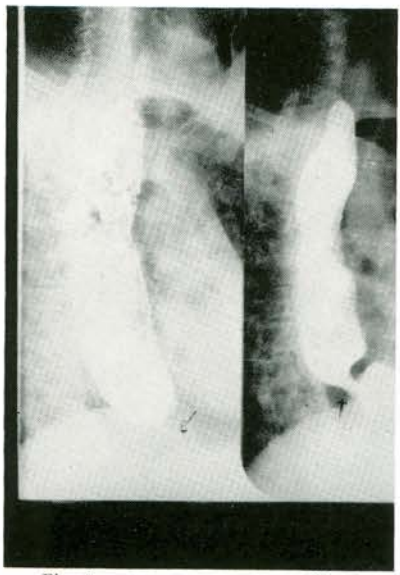

Fig. 1

Fig. 2

An esophagram (Fig. 2) taken six weeks post – operatively showed a noticable diminution in the diameter of the esophagus and a very adequate cardial lumen through which barium passed very rapidly. Her main concern at that time was the treatment of her latent syphilis. Over a period of four months she gained 25 pounds.

SUMMARY

The causative factors of achalasia of the esophagus are briefly reviewed. A case of cardiospasm is presented in which marked hypertrophy of diaphragmatic muscle fibers about the cardia caused obstruction of the lumen. The successful treatment of this condition by severence of these muscle bundles and cardioplasty after the method of Heineke and Mikulicz is described.

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CASE PRESENTATION

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and
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A 76 year old white female was admitted to the Allentown Hospital with the chief complaint of loss of appetite and burning in "pit of stomach" approximately three months' duration.

History of Present Illness: Three months before admission the patient noticed some epigastric distress, "sort of a pressure". At first it was associated with meals, directly after eating. This became more constant and there was a marked loss of appetite. The distress was relieved sometimes by a half glass of warm water then a half glass of water with a little salt. She would then have gaseous eructations and feel better.

The patient stated that she was not afraid to eat, but just didn't feel like eating anything. The distress gradually became associated with pain which radiated through to the back and was more severe just before admission. She never experienced any vomiting and there was nothing to suggest a gross hemorrhage. There was a loss of weight of approximately 10 pounds in the past three months. She had never been very heavy.

Systemic Review: Essentially negative except for the above mentioned loss of weight.

Family History: Non-contributory.

Past Medical History: No serious illnesses, no previous operations.

Fig. 1

Physical Examination: B.P. 110/70, P. 75, R. 18; 76 year old, poorly nourished white female in no particular acute distress. Weight, 100 pounds. Physical essentially negative except for some epigastric tenderness. No masses palpable. Liver not enlarged.

Laboratory Studies: Hemoglobin 64%; RBC 3,260,000; WBC 10,600 with normal differential. Urea 13 mgm. %; Plasma chlorides 540; Serum protein 6 grams.

X-ray Examination: Examination by Barium meal showed a large ulcer crater of the stomach on the lesser curvature and slightly posterior. This crater is located just above the incisura angularis and is penetrating in type, projecting out from the lumen of the stomach. It measures 2.5 cm. in diameter and is shown in Fig. 1.

No other abnormality was demonstrated in the esophagus or duodenum and there was no gastric retention at two hours.

At operation the large ulcer crater was found on the lesser curvature of the stomach posteriorly. It was fixed to the pancreas and transverse mesocolon just to the left of the middle colic artery. A 75% gastric resection was performed. A portion of the crater was not dissected from the transverse mesocolon and lower end of the pancreas. However, a frozen section was obtained and this proved to be negative for malignancy. The post-operative progress was uneventful and unusually smooth for a person of this age.

Discussion: A gastric ulcer in a patient of this age is not rare, but the likelihood of it being malignant is often entertained. All diagnostic aids should be employed.

Some authors believe that the size of the ulcer is a criteria of malignancy, pointing out the increasing incidence of carcinoma the larger the ulcer and arbitrarily setting a limit of 4 cm., above which malignant ones are most likely.

A radiological differentiation is sometimes attempted but not always diagnostic. As a rule the penetrating type may be considered benign, that is those projecting from the normal line of the lumen of the stomach. Conversely, an intraluminal ulceration is more in favor of an ulceration in a new growth. However, malignant degeneration in a previously benign ulcer should also be considered and there is nothing particularly diagnostic of this type. Exploration, frozen section if suspicious and then the specified resection is the only way of being sure.

The location of the ulcer is also of importance. Many well-known radiologists have only seen one or two benign ulcers of the greater curvature in their vast experiences, but then to base benignancy on the presence of an ulcer on the lesser curvature would be in error. Seventy to eighty per cent of malignancies of the stomach occur in the prepyloric or antral region of the stomach.

In gastric carcinoma the ulcer syndrome is usually lacking. In a

series of 1000 cases of gastric carcinoma reported only 7% exhibited the usual ulcer history. Adding 12% who complained of indigestion which would be compatible with, but not typical of peptic ulcer, this would make 19%. Evidently the remaining cases were silent or presented as complications either due to obstruction, extension, hemorrhage or picked up on a routine or survey x-ray examination.

Conclusions: A case is presented of a rather large gastric ulcer in a 76 year old female which proved to be benign.

Reference: Cancer, Vol. 3, 515-552, 1950, Relationship of Gastric Ulcer to Gastric Cancer.

CUMULATIVE INDEX
VOLUME II

(Number 2 of Volume 2 was a non-professional resume of the Allentown Hospital and its services and the pages were not numbered)