CONTENTS

Foreword ............................................................... 1

Preadolescent and Adolescent Dystrophies of Bone Common to the Lower Extremities .................................................. 3
Kenneth L. Wheeler

Management of Epiphyseal Dystrophies of the Spine and Lower Extremities ................................................................. 10
James M. Eaton

Pulmonary Embolism: A Report of Two Cases ........................ 23
Otterbein Dressler

A Background for Rational Therapy ...................................... 28
Guy S. Deming

Published monthly by the
PHILADELPHIA COLLEGE OF OSTEOPATHY
48th and Spruce Streets, Philadelphia 39, Pa., U.S.A.
OSTEOPATHIC MEDICINE

The official publication of the Faculty of the Philadelphia College of Osteopathy

Editor: FREDERICK A. LONG
Associate Editor: OTTERTBEIN DRESSLER

Editorial Board
FREDERICK A. LONG, Chairman
OTTERTBEIN DRESSLER
PAUL T. LLOYD
EDGAR O. HOLDEN, Ex-officio

Advisory Board
WILLIAM BALDWIN, JR. D. S. B. PENNOCK
RUSSEL C. ERB JOSEPH PY
H. WALTER EVANS J. FRANCIS SMITH
RALPH L. FISCHER C. HADDON SODEN
J. ERNEST LEUZINGER H. WILLARD STERRETT
RUTH E. TINLEY

Business Manager: MR. LOUIS G. SCHACTERLE

OSTEOPATHIC MEDICINE is issued monthly, one volume of twelve issues being published each year. The subscription price is $4.00 per year, payable in advance. Single copies 50 cents.

Address all correspondence relating to business matters and subscriptions to Mr. Louis G. Schacterle. Address all other correspondence to Dr. Frederick A. Long. Both addresses 48th and Spruce Streets, Philadelphia 39, Pa.

Copyright 1943, by Philadelphia College of Osteopathy
Made in U. S. A.
FOREWORD

OSTEOPATHIC MEDICINE is being offered to fill the long felt need for a suitable medium in which to bring together the writings of the Faculty of the Philadelphia College of Osteopathy on technical and related subjects. While some papers given at various meetings and conventions have been included in the literature, many more have failed to find this wider scope of usefulness. These papers along with interesting case reports, autopsy reports, research reports, and contributions from the basic science departments have needed a vehicle through which they could be brought to the many who would like to share the opportunities we in a teaching institution enjoy. It is in recognition of a certain responsibility to those in the field who look to the colleges to keep them informed about the advancing front of medicine that the College is attempting through this medium to extend its work to the man in the field. Osteopathic Medicine will be the official publication of the College Faculty. In selecting papers for publication, the attempt will be made to pay particular attention to those of greater interest and usefulness to the general practitioner and general surgeon while not neglecting those of value to the specialist and to the investigator.

One volume consisting of twelve monthly issues will be published each year. Volume I, of which this is the first issue, will include the months of August through December, 1943. Subsequent volumes will correspond to calendar years. Paging in each volume will be consecutive beginning in the January issue and ending with the December issue of each year. The mailing date will be the first of the month. The size selected, we believe, lends itself to ease in handling and binding for library purposes.

Editorial control rests with the Editorial Board which will be assisted, when necessary, by the Advisory Board composed of
various department representatives. The decisions of the Editorial Board will be final.

It is the hope of the Board that *Osteopathic Medicine* will become a useful addition to osteopathic literature, and it will be the constant aim of the Board to merit for the publication the high regard of the profession which it is attempting to serve.

**The Editorial Board**

**OF THE**

**Philadelphia College of Osteopathy**

July 10, 1943
Adolescence is a vital period of bone growth. It is during this span of years that the centers of ossification attain maturity. Any pathologic state involving these epiphyseal structures in adolescence or childhood is obviously not in accord with normal development. Osteopathic principles declare the fact that functional integrity is directly dependent upon normal anatomy. It is likewise established that stability of any superstructure is directly dependent upon the architectural exactness of its foundation, and it is for this reason that we choose to discuss some osseous dystrophies common to the lower extremity.

It was in 1903 that Osgood radiographically established an apparently non-infectious, non-traumatic, rarefying pathology involving the anterior tubercle of the proximal tibia, now recognized as Osgood-Schlatter's disease. Analogous dystrophic lesions have since been described affecting the epiphyses of many of the long bones, some of the small bones of the hands and feet, and, as well, secondary centers of vertebral ossification. Of all the known osseous pathologies, no one group of diseases has caused as much confusion with regard to etiology, pathologic evaluation, and terminology. Most of these so-called epiphysites qualified as chronic, non-suppurative, non-tuberculous, non-traumatic, aseptic, etc., were unknown prior to the advent of roentgenology. Each lesion as recognized was designated by the original author's name. Simultaneous reports were made, and the names of both authors were employed by some while others chose an anatomically descriptive term as a means of identification. Due to the extensive skeletal distribution of these epiphyseal dystrophies, as many as thirty names of initial authors have been recorded. It was not until 1930 and again in 1935 that Harbin and Brailsford, respectively, were impressed by certain elements of similarity in these bone diseases and chose to class them as local manifestations of the same pathologic process, namely, an osteochondritis. At present writing, these many times clinically unrecognized dystrophies are perhaps even more properly termed osteochondroses.

**Legg-Calvé-Perthes' Disease**

Universally known as Legg-Calvé-Perthes' disease this osteochondrosis, involving the hip joint or more specifically the capitate epiphysis of the femur, was originally reported by Legg in 1909. Calvé of France, in 1910, described a series of quite identical cases under the title of pseudo-coxalgia; and in the latter part of this same year Perthes, of Germany, independently published a report of analogous hip joint changes and called them arthritis deformans juvenilis. Unable to demonstrate

*Read at the Annual Convention of the New England Osteopathic Association, Providence, R. I., May 15, 1943.*
inflammation within the synovial membrane of the hip joint, Perthes was obliged to rename the disease osteochondritis deformans juvenilis. Calvé subsequently chose to designate the dystrophy as coxa plana.

This osteochondrosis involving the weight bearing epiphysis of the proximal femur, usually occurs between four and twelve years of age with an approximately three to one preponderance in the male. Bilateral demonstration of the disease has been reported from as low as 12.5 to as high as 40 per cent of collected cases.

The early clinical manifestation of the disease has been described as a relatively painless, frequently intermittent limp with varying degrees of regional increased muscle tension. Some vague discomfort may prevail in the region of the affected hip; while in many cases, the discomfort is referred to the knee. A concomitant restriction or limitation of femoral abduction and internal rotation has been reported as of definite diagnostic significance. Some authors have noted a mild degree of irregular fever, though no other constitutional disturbances have been observed. Brailsford states that one is astounded by the trivial symptoms which are associated with most marked radiographic manifestations. This is further substantiated by the quite common incidence of old hip joint chondropathies observed in adults with no knowledge of childhood or adolescent hip disease.

Radiographic investigation of the hip joint during the early or symptomatic stage of the disease is often negative for significant changes. Periodic or serial film surveillance is obviously indicated; and it is Brailsford, in a recent publication, who states that within a period of two to three months from the onset of the first symptoms film evidence of the disease will be manifest as an increased radio-density of the femoral capital epiphysis. It is at this time that the capitae epiphysis is becoming plastic. In the Surgical Supplement of the J. A. O. A. of October 1940, Lloyd reported a heretofore apparently unrecognized early roentgen sign described as a small radiotranslucent fissure beginning at the articular margin of the epiphysis, usually at the point of maximum weight bearing, and extending in a longitudinal direction for a short distance into the capital structure.

The second or deforming stage of the disease has been noted to be relatively asymptomatic. However, radiographic investigation will demonstrate marked changes within the epiphysis characterized by small areas of osseous condensation distributed within an apparently rarefied or relatively radiotranslucent bony nucleus. Brailsford ascribes these changes to an avascular necrosis and emphasizes the now plastic state of the femoral head. The normal convex articular aspect of the epiphysis will at this period display varying degrees of flattening or pressure deformity. In the absence of proper treatment, progressive pathologic changes are manifest as epiphyseal flare with structural fragmentation. The adjacent metaphyseal zone often shows a prominence as to width, and the related diaphyseal margin of the femur may present an irregularity with small pseudo-cystic radiotranslucencies. Expansion and apparent shorten-
ing of the femoral neck are common to the advanced case. All of the changes mentioned above usually occur within an eighteen months’ period from the onset of the first symptoms.

The healing stage of the disease is notably asymptomatic. Therefore, serial roentgen ray studies are necessary to establish the degree of epiphyseal regeneration which, according to Brailsford’s pathologic table, will occur over a period of one and one-half to four years from the onset of initial symptoms. A progressive improvement of radiodensity will be apparent throughout the capitate epiphysis with absorption of the aforementioned dense osseous fragments. The osteoporotic zone located in the adjacent diaphyseal portion of the femur is likewise gradually eliminated. Of major therapeutic importance is the fact that plasticity of the femoral head prevails throughout this healing stage. If the condition is bilateral, it has been established that the pathologic processes are completely independent of each other.

In contradistinction to tuberculosis, there is preservation of the hip joint articular fissure throughout the average four year period of unstable epiphyseal architecture. Perthes is of the opinion that the contour of the related acetabulum shows changes only in the advanced stages of the disease. Platt believes that the acetabulum is gradually moulded by adaptation of the fossa to the altered lines of force or pressure produced by the deformed femoral head. These permanent structural alterations, involving the proximal femur and acetabulum, obviously result in a modification of hip joint physiology and, as well, constitute one etiologic factor with regard to anatomic deficiency in leg length.

Superimposed hip joint pathology of an arthritic order, frequently occurring in early adulthood, will often climax the tragedy of no treatment or inadequate treatment during the florid and reparative stages of the disease. In 1939, Stammel, of the United States Army Medical Corp, reported that the juvenile osteochondrotic hip deformities can easily be overlooked in the applicant for enlistment only to evidence themselves as a disability when the young adults are subjected to the stresses and strains of military life. Legg’s disease with associated structural deformity unquestionably provokes arthritic changes with advancing age. However, it must be emphasized that early recognition and proper treatment of the malady will result in only minimal deformity with relatively normal hip joint function.

There is a paucity of autopsy or biopsy material in Legg’s disease though the pathology of this disease has been quite universally interpreted as an aseptic necrosis or, as termed by Axhausen and Brailsford, an avascular necrosis. Attempted cultures and animal inoculations without exception have proved negative.

The etiologic factor productive of this pathologic state or osteochondrosis is unknown. Harbin and many others contend that more than one etiologic agent prevails, and this view is substantiated by the now existent numerous causation theories. The occasional multiplicity of identical pathologies in one patient, and the familial occurrence of the disease
sometimes encountered, lend support to the hypothesis that some debilitating factor, possibly an endocrinopathy, constitutes the unknown, and that the frequently reported trauma, even though minimal, has been directed to already predisposed skeletal parts. Lloyd, in a recent publication, states that a traumatic history was elicited in most of the cases of Perthes' disease examined at the Philadelphia Osteopathic Hospital.

**Osgood-Schlatter's Disease**

The adolescent tibial tuberosity is not an uncommon site of an osteochondrosis, clinically described as a painful enlargement of the tibial tuberosity and now universally known as Osgood-Schlatter's disease. This pathologic state was authentically first recorded by Vogt of Germany in 1869. A detailed report and radiographic description of the disease was published by Osgood of Boston in 1903 and again by Schlatter of Germany in 1908. Anatomically, the proximal portion of the tibia develops from one center of ossification. This osseous nucleus exhibits an anterior tongue shaped process extending downward in front of or overlying the diaphysis. Complete ossification and fusion of the epiphysis does not occur until approximately eighteen to twenty-two years of age. Likewise, it may be emphasized that the anterior epiphyseal projection or tubercle is the site of insertion of the major portion of the patellar tendon.

Axhausen and many others are in agreement that the fundamental pathology of Osgood-Schlatter's disease, involving the ununited anterior tibial tubercle, is that which is common to all of the osteochondroses, namely, an aseptic necrosis. The traumatic etiologic theory is given unquestioned priority; however, an as yet unknown underlying debilitating or predisposing factor must be attributed equal importance. Of all the cases examined at the Osteopathic Hospital in Philadelphia, very few patients have reported a definite regional injury. Considering the age incidence of ten to sixteen years and predominance in the male, it is quite probable that athletic and other minimal direct or indirect traumas play a prominent role in the etiology of the disease.

Pain, which is variable in intensity, and an associated localized palpatory tenderness over the patellar tendon and proximal pretibial epiphyseal process constitute the common clinical manifestations of Osgood-Schlatter's disease. There is often observed a regional soft tissue swelling or fullness indicating an associated pretibial bursitis which augments the pain and resultant disability.

Radiographic investigation of the normal adolescent knee will demonstrate a well delineated anterior epiphyseal process separated from the tibia proper by a radiotranslucent metaphyseal zone. Anatomically, this process may vary in size or extent, though the discreet relief and radiodensity of the tubercle will be preserved.

Osgood-Schlatter's disease is characterized radiographically by rarefaction or demineralization of the epiphysis with loss of normal trabecular architecture, irregularity of contour, and structural fragmentation. Vary-
ing degrees of separation and elevation of the process or fragments of the same will be manifest, and modification of overlying regional soft tissues is often observed. Periodic film investigation is necessary to determine the arrest and ultimate resolution of the pathologic state. Prominence and deformity of the tibial tubercle may persist after complete epiphyseal ossification. A bilateral occurrence of the dystrophy has been recorded in approximately eleven per cent of collected cases.

Köhler's Disease

Köhler's disease is an osteochondrosis involving the tarsal navicular (scaphoid) bone, and was first described in 1908. This dystrophy, which is now identified pathologically as an avascular necrosis, usually occurs between three and ten years of age and is more common in boys. Bilateral involvement has been noted in approximately twenty-five per cent of collected cases.

A limp, pain upon weight bearing, and concomitant palpatory tenderness located over the navicular bone constitute the major clinical manifestations of the disease. Usually, there is noted some regional swelling of soft tissues over the dorsal aspect of the bone. It must be emphasized that clinical evidence of the lesion may be intermittent and also that symptoms may diminish with radiographically established progression of the pathology.

X-ray examination in the early stage of the disease will demonstrate a relative increase in radiodensity of the tarsal navicular bone. It is common to find the dystrophic changes well advanced at the time of initial radiographic investigation. The osseous nucleus will appear very dense, small or compressed, irregular, and often fragmented. All of the adjacent tarsal bones will display a decalcification or loss of normal density which Braiksford states is the phenomenon of oscillating interchange of calcium between necrotic and adjacent living bone.

Resolution of the pathology usually occurs within a period of approximately eighteen months to two years. The first indication of navicular regeneration is marginal, with the dense fragments and islands of structureless condensation gradually replaced by normal bone. At the termination of the healing stage there will be little or no deformity of the part, though a coarse cancellous architecture may prevail. Simultaneously, the adjacent tarsal bones normalize as to radiodensity. Continued functional trauma may delay the reparative process.

The etiology of this osteochondrosis is unknown. Anatomically, the pre-osseous cartilage of the navicular is the last of the primary tarsal growth centers to display radiographic evidence of ossification. Köhler is of the opinion that this fact and also the relative location, contour and notably poor blood supply of the navicular predispose the bone to this dystrophy.

Freiberg-Köhler's Disease

Köhler's disease II involving the capitate portion of the second metatarsal bone may also be classified as an osteochondrosis. This dystrophy
was originally described by Freiberg and Köhler in 1914; and upon a review of all literature, Köhler, in 1935, had recorded one-hundred and fifty cases. In the scientific supplement of the Osteopathic Digest, published June 1937, Lloyd reported four cases, and at present writing several additional cases have been observed at the Philadelphia Osteopathic Hospital.

This disease is usually encountered between ten and eighteen years of age with a four to one preponderance in the female and a decidedly more common involvement of the right foot. Many undoubtedly well founded etiologic hypotheses have been stated, though the fundamental cause of the disease is as yet unestablished. Zeitlin is of the opinion that anatomical or structural weakness of the musculature of the foot with an overloading of one of the metatarsals may constitute an important cause of the lesion. A developmentally short first metatarsal upon comparison with the second metatarsal bone has been emphasized with regard to metatarsal overloading, and the aforementioned insufficiency of the osteomuscular mechanism of the foot (Zeitlin) may account for the occasional involvement of the third metatarsal bone.

The basis or fundamental pathology of Köhler's disease conforms to an aseptic necrosis. Subsequent mollecular and hence gross architectural changes within the capitate epiphysis of the metatarsal in all probability constitute the criteria for the descriptive term "metatarsal infraction" employed by Freiberg.

Pain, located in the second or sometimes third metatarso-phalangeal joint, aggravated by weight bearing, and productive of a concomitant protective gait or limp constitute the subjective symptoms of this dystrophy. Regional palpatory tenderness and swelling of respective soft tissues over the dorsal aspect of the pathologic part are common objective manifestations. Hauser states that passive flexion of the involved toe will produce severe pain while others report that pain on motion is a less constant finding.

Radiographic examination of the foot at the time of inauguration of the symptoms will, according to Brailsford, demonstrate a slight increased radiodensity of the capitate epiphysis of the involved second or third metatarsal. Rarefaction of the capitulum will soon follow, and film studies obtained during the active stage of the disease will show irregular zones of condensation distributed within the rarefied epiphyseal structure. The articular margin of the metatarsal characteristically exhibits an irregular flattened or compressed contour indicating structural plasticity. The opposing proximal surface of the first phalanx will appear condensed and broadened, while the respective metatarso-phalangeal articular fissure shows an increased width. When the lesion is well established, the distal one-half of the metatarsal displays an increased circumference with loss of the normal cervical contour of the bone.

The osteal deformity of Freiberg-Köhler's disease is permanent, and arthrotic changes may subsequently be superimposed upon the physiologically unstable metatarso-phalangeal joint.
Epiphysiolyis

Epiphysiolyis, also known as “slid” epiphysis or adolescent coxa vara, has been reported as the most common disease involving the hip throughout the adolescent age period. This dystrophy of unknown etiology may be briefly described as an apparent dissolution of the metaphyseal fixation between the capitate epiphysis and cervical portion of the femur with resultant structural deformity. Brailsford is of the opinion that an undetermined intrinsic disease produces this metaphyseal disorganization, and that physiologic stresses and strains of weight bearing or a reported minimal trauma often cause the altered anatomical relationship of proximal femoral parts. A higher incidence of the lesion prevails in the male; and although not a diagnostic criterion, this disease is many times observed in association with the state of endocrine imbalance known as Fröhlich’s syndrome.

Both the clinical and radiographic criteria of epiphysiolyis may be divided into three chronological stages. The first, or “preslipped” stage of the disease, shows no gross deformity of the proximal femur. However, pathologic changes within the growth cartilage, and early metaphyseal dissolution will be radiographically manifest by an increased width of the radiotranslucent metaphyseal zone. The diaphyseal margin of the metaphysis characteristically exhibits an irregular contour and is poorly delineated, while the respective epiphyseal border possesses a discreet relief. Pain, of varying intensity located in the affected hip, an associated limp, and eversion of the foot constitute the clinical evidence of this first stage. The fact should be emphasized that recognition of these early signs of epiphysiolyis and the institution of proper treatment will prevent the inevitable deformity of the affected femur.

The second stage of this disease is often designated as the stage of fracture. There is usually recorded a history of trauma, even though minimal, with the subsequent sudden onset of symptoms compatible with femoral fracture. This acute stage may be characterized by a complete dissolution of the metaphysis. There is visualized an upward displacement of the femoral shaft with dependent internal rotation of the capitate epiphysis in the acetabular fossa. The head of the femur appears to have slipped off the neck of the bone.

Milch is of the opinion that preceding the fracture stage there occurs a gradual, progressive anteversion of the femoral neck which tends to exaggerate the eversion of the foot and leg. The degree of external rotation will hence exceed the limit which permits support of the femoral head, and the second stage of the disease will be initiated. He has chosen to designate the lesion as epiphysis coxa anteverta, and emphasizes the need for lateral as well as antero-posterior roentgenograms.

Stage three of the disease has been indicated as the healing or chronic stage. There occurs a premature fusion or union between the femoral epiphysis and diaphysis. Unless early and proper treatment has been instituted, coxa vara deformity with shortening of the leg will terminate the dystrophy.
MANAGEMENT OF EPIPHYSEAL DYSTROPHIES OF THE SPINE AND LOWER EXTREMITIES*

JAMES M. EATON

Associate Professor of Orthopedic Surgery

Disturbances of the epiphysis and adjoining bony structures resulting in an aseptic necrosis of the epiphysis with consequent functional impairment are naturally involvements of children and adolescents. However, the dystrophy may heal and not give rise to disabling symptoms until quite late in life, leading to considerable confusion in the minds of both patient and physician.

Whatever the cause of the epiphyseal derangement, whether it be traumatic, inflammatory, endocrine, embolic, or functional, one factor remains constant, and that is the plasticity of the involved bone. No epiphysis in the body is immune to these disturbances. No matter where it occurs, in all probability the same underlying pathologic process is present, though the particular location modifies its features in certain respects. The great variety of stresses and strains to which the epiphysis is subject causes the essential differences in clinical history and management.

An epiphysis may be described as that part of the bone concerned with growth in length, and which in addition takes part in the formation of joints, and acts as an attachment for important muscle groups. Hence, any involvement of these structures may seriously affect the symmetry and function of the limb as a whole. The epiphysis develops from a secondary center of ossification, and is at first separated from the main shaft of bone by an area of unossified cartilaginous tissue, the epiphyseal plate. Later, it joins the shaft or diaphysis to form the adult bone. The cartilage lying between the bone tissue of the epiphysis and the adjoining end of the diaphysis (metaphysis) is known as the epiphyseal cartilage, and this cartilage does not ossify nor does the epiphysis become joined to the body of the bone until growth has ceased.

The most frequent sites of involvement are (1) the proximal end of the femur, (2) the tongue-like projection of the proximal tibial epiphysis, (3) the tubercle of the calcaneum, and (4) the secondary epiphyses of the vertebrae.

There are many theories as to the etiology of this disturbance in normal bone growth, but they remain theories for the most part. This is due to the fact that surgery is usually not indicated, and hence, there have been few opportunities for a comprehensive study of the histopathology. The only general agreement that seems to exist is that the condition is neither tuberculous, rachitic, nor syphilitic.

Because of the prominence of a history of trauma, this has been considered by some as the sole determining factor. As the result of injury there may be an obliteration of a portion of the vascular supply of

the epiphysis, possibly embolic, resulting in an infarction of bone with resultant destruction. A compensatory hyperemia of the adjacent portion of the diaphysis is the natural response, and is the starting point of those hypertrophic changes which are evidenced as broadening. It is also felt that because of its more usual location in the weight bearing joints, or in the epiphysis which are subject to greater strain, trauma plays a most important part. It has been my observation that the trauma to which the patient's attention is directed as a cause of his complaint, is usually not of sufficient intensity to bring about the destruction of tissue that is usually found on roentgen examination. It may be that the trauma only serves to focus the attention of the patient on the area of involvement.

The condition frequently occurs after a general septicemia or some previous infective state, and hence, many feel that infection plays an important part in the etiology. Pathogenic micro-organisms have been isolated; and in some cases, although microbial growth has been observed histologically, the sections of bone removed were not typical of an old infective lesion. It is possible that infective micro-organisms circulating in the blood stream may localize in an epiphysis which has been subject to mild trauma. These organisms may form the basis of an embolus and produce an infarct. The trauma in these cases may be the stress and strain of weight bearing or of muscular overactivity.

With such growth disturbances one's attention gravitates to some endocrine imbalance affecting bony metabolism. Many of the patients suffering from these osteochondral disturbances present evidence of pituitary and thyroid obesity. A rather interesting observation is the high incidence of a metabolic disorder in the family histories of these cases, principally colloid goiter. Retarded bone development is a rather consistent finding in children with thyroid deficiencies.

In all the various osteochondroses there is a marked similarity in the symptomatology. The onset is gradual, and there may or may not be a history of trauma, which if present is commonly slight. As a rule the patient is in good health, and is practically never acutely ill, although there have been cases reported where the patient was suffering from a mild septicemia.

The local effects of the disease are similar to those of an early tuberculosis—slight pain, limp in a weight bearing joint, limitation of motion, and at times muscle spasm. The term "growing-pains" now held in disrepute may be re-adopted with some justification as it is highly probable that congestion of the rapidly growing epiphysis may give rise to such symptoms. All these symptoms are mild; many cases are symptomless, and are only discovered when deformity and disability develop. The condition may be unilateral or bilateral.

When the condition is found to be bilateral the interesting theory presents itself that the condition might be due to a disturbance in the trophic nerve structures, suggesting a central origin and not one due to a purely local disturbance of bony growth.

The x-ray appearance in the various forms of osteochondrosis are
very similar, and usually out of all proportion to the mildness of the clinical picture. These findings have been most ably presented by Dr. Wheeler and need no further elaboration by me.

**Legg-Calvé-Perthes' Disease**

In the lower extremity, probably the most serious epiphyseal disturbance is that affecting the capitate epiphysis of the femur. In this location the disease has been given a great variety of names, and is probably best known as Perthes' disease. It is also known as osteochondrosis deformans coxae juvenilis, coxa plana, quiet hip disease, flat head, Legg's disease, Calvé's disease, or pseudo-coxalgia. As far as is known this disease was first described by Legg of Baltimore in 1909, but for some unknown reason the name of Perthes is most frequently associated with it. This involvement is generally unilateral, though occasionally it is bilateral, and most commonly affects males between the ages of five and ten years.

Symptomatically there are usually three stages of the disease; the onset, the active stage, and the stage of recovery. At the onset limp is common, but not always associated with pain; in fact the absence of pain is commented upon by many observers. Frequently the pain, when present, is nocturnal in occurrence, as in early tuberculosis. Pyrexia has been observed, though these cases are rarely seen in this stage. Muscular spasm is usually present with slight flexion and some fixation of the hip joint. Characteristically, the greatest limitation of motion is in abduction. This stage usually lasts from two to three months. It would be ideal if these cases could be treated during this time because the bone becomes plastic with the increase in density of the femoral capitate epiphysis and the osteoporosis of the diaphysis.

During the active stage, which usually lasts from three to eighteen months, the limp tends to disappear, although it may return for short intervals and may occasionally continue indefinitely. The muscular spasm usually disappears quickly, but leaves a residual limitation of motion in abduction. The pain and tenderness disappear before the cessation of the muscular spasm, and the child may be limping markedly with a fixed and painless hip. The usual position is one of slight flexion with abduction, as contrasted with the abducted hip of early tuberculous disease. During the active stage the affected hip joint shows limitation of abstraction medial rotation, and flexion. The limitation of motion is due first to the spasm of the abductor muscles, later to an actual shortening of these muscles, and still later the deformed femoral head provides mechanical obstruction to full movement. The trochanter on the affected side is more prominent, and if grasped with the thumb and fingers, its antero-posterior diameter will be felt to be definitely thickened compared with the well side. The muscles of the affected side are usually under-developed, more from lack of use than to any trophic disturbance. There is little, if any shortening, in spite of the deformity of the femoral head.

The stage of recovery is characterized by gradually diminishing sub-
jective and objective symptoms until the function of the hip is restored so completely that there is little or no difference from the normal. However, two signs persist throughout life, namely, trochanteric thickening and limitation of the range of abduction.

The prognosis of Legg-Calvé-Perthes’ disease is good, as it is usually self limiting, with a strong tendency to spontaneous recovery. Approximately fifty per cent of the cases have some limp with restriction of motion, particularly in abduction. There is usually a predisposition to arthritis deformans at a later age.

The final result depends upon the type of disease: The mushroom type does not show marked atrophy or fragmentation of the epiphyseal centers. In some cases the epiphysis migrates laterally toward the greater trochanter, while in others this displacement is slight. Abduction, and sometimes rotation is limited when the epiphysis shows marked migration, otherwise motion may be normal in adult life. There is little if any shortening in this type. The fragmented type shows marked variation in x-ray density in the epiphyseal center and the neck, fragmentation of the epiphyseal bone center, and shortening and rounding off of the upper end of the neck. The epiphysis in some cases appears to be obliterated. Limitation of motion and shortening in this type are considerable.

Management: It has been demonstrated rather conclusively that the bone remains plastic for a period of from one to four years, and it is this long period of bone plasticity in osteochondrosis to which the clinician should give attention. Disregard of this is responsible for the deformity caused by stresses and strains on the inadequately immobilized joint surfaces. The clinical signs and symptoms of this disease usually disappear long before the plasticity of the bone. Treatment must, therefore, be regulated by the roentgenographic appearance and not by the absence of clinical signs or symptoms. The plasticity of the bone can be judged by the roentgenographic appearances only; therefore, rest of the affected bone should be enforced as long as the roentgenogram suggests plasticity.

In the acute stage the patient should be confined to bed with traction applied for three to four weeks. Following this time a plaster-of-Paris hip spica should be applied including the foot on the affected side, with the extremity in approximately 20 degrees of abduction and 5-10 degrees of internal rotation. This immobilization should be continued for a period of at least three months, then the cast should be removed and x-ray studies made. If there has been fragmentation or marked pathology in the epiphysis it would be well to continue immobilization in a plaster hip spica for another three months. If the involvement is mild, then the patient should be permitted to be ambulatory, but under no consideration should weight bearing be allowed on the affected hip until sufficient time has elapsed to allow for complete regeneration of the bone.

At the present time we employ a well-leg lift of one and one-half to two inches on the heel and sole of the shoe of the uninvolved side, and the patient is supplied with a suitable pair of crutches. They are instructed that under no consideration should they bear any weight on the af-
fected limb in standing or kneeling. At all times they should be able to swing the affected side free of the ground. It has been my experience that these patients are most cooperative, and apparently follow orders very well in this respect. A Thomas walking caliper brace or a Bradford abduction-traction brace may be also applied to insure no weight bearing on the affected limb.

Periodic roentgenograms should be made to check on the progress of the disease and the effectiveness of treatment, and these examinations should be continued at intervals for several years—as long as the bone remains plastic and consolidation has not taken place. Recurrences are not common, however. When the disease has been arrested, postural or erect film studies should be made to determine accurately the shortening on the affected side, and appropriate “lifts” should be applied to the shoe on that side to compensate for the deficiency in extremity length. Unless this is done, static deformities of the vertebral column will result, the degree of scoliosis and altered vertebral mechanics depending on the amount of shortening.

It would be well in the presence of obesity that suitable weight reduction measures be employed to relieve the weight bearing surfaces of the joints of this added stress and strain. If there is a definite endocrine involvement this should be treated to balance the endocrine system accordingly.

Operative treatment is not indicated, particularly as the disease tends to a spontaneous recovery with comparatively little deformity in the average case. In young patients there is no ankylosis of the hip joint as the articular cartilage is not violated. If, at a later time, disability of the hip results from impingement of the femoral neck on the acetabulum, one should consider acetabuloplasty or arthroplasty of the hip, particularly in those cases suffering from a painful and disabling arthritis deformans.

Displacement of the Upper Femoral Epiphysis

This condition is also known as epiphyseolysis capitis femoris, or adolescent epiphyseal coxa vara, or more correctly, as Milch has suggested, epiphyseal coxa ante-verta. Slipping of the upper femoral epiphysis differs from the displacements which may follow injury to other epiphyses at the ends of the long bones in that the line of separation is at the junction of the metaphysis with the epiphysial cartilage. The epiphysis slides backwards and slightly downward, but unlike other traumatic epiphyseal separations it does not carry with it a fragment from the margin of the diaphysis over which it is displaced. In about one-third of the cases, there is almost simultaneous involvement of both hips, often without a history of injury to either hip, suggesting that the lesion is not a simple traumatic one. The great majority of these displacements arise without any recognized injury or strain.

The usual history is one of a trivial twist or strain that is followed for weeks or even months, by increasing disability of the affected hip joints. These patients are usually of the male sex at the age of puberty,
and are usually obese. There is limitation of all hip movements and slight shortening of the limb, with the deformity of 40 to 90 degrees of external rotation. As Watson-Jones has said, "these signs are unmistakable, but they never should have arisen. The diagnosis has been made too late." The epiphysis is already fully displaced and becoming fixed in its deformed position, and a good result cannot be anticipated whatever treatment may be pursued at this time. A provisional clinical diagnosis can always be made before there is fixed external rotation deformity, and only then is there a reasonable prospect of successful treatment.

If a child between the ages of ten and sixteen develops an intermittent limp, or complains of occasional stiffness in the thigh and knee, epiphyseal coxa vara must be suspected. Pain in the knee is even more frequent than pain in the hip. The symptoms are typically intermittent, and in the intervals the patient may run, jump, and pursue normal recreational activities. The important clinical sign is limitation of internal rotation movement. The range of motion must be estimated accurately in degrees employing a protractor, the normal range being between 50 degrees and 60 degrees. It is impossible to fully flex the hip in the normal manner so that the knee touches the front of the chest. In these cases, full flexion of the hip results with the knee lying at the side of the chest wall near the axillary line, and the limb externally rotated. On the appearance of any of these clinical signs, complete radiographic examination is imperative.

Trauma, as a primary etiological factor, has been discredited because some of these cases have arisen while the patient has been confined to bed while the other hip has been under observation and treatment. The exact nature of the predisposing factors is unknown, but many patients with this involvement show the characteristic changes of dystrophia adiposogenitalis. Inasmuch as the anterior lobe of the pituitary gland exercises a control over endochondral ossification, possibly pituitary disfunction causes metaphyseal decalcification.

As has been mentioned previously, the displacement of the epiphysis is downward and backward and may progress in this latter direction until its margin locks in the trochanteric fossa at the back of the neck of the femur.

Early x-ray recognition is difficult from antero-posterior films. A lateral roentgenogram must be made; and without this view, suspected epiphyseal slipping cannot be excluded.

Management: There are two principles of treatment: (1) The displacement must not be reduced forcibly by manipulation or operation, (2) Active treatment must not cease until the epiphyseal line has been fused. It has been observed that the maneuver of forcible reduction by manipulation is dangerous as it may cause necrosis of the epiphysis. The epiphysis is largely dependent for its blood supply on the vessels of the ligamentum teres, and manipulation may so damage the blood vessels as
to cause thrombosis and avascularity. Reduction after open exposure of the epiphysis and femoral neck is even more dangerous, because incision and retraction of the capsule of the hip in itself produces avascular necrosis of the femoral head or its epiphysis.

Gradual traction is the only safe method of reduction, skin extension or skeletal traction from the tibial tubercle being used. No attempt is made to force correction by using wide abduction, rather, traction is applied to the limb in the position in which it lies. If the epiphysis is not already fixed in its deformed position, it can be reduced within ten days by heavy traction, which is then followed by light traction to prevent redisplacement.

Treatment must not cease until the epiphysis line is fused, and the patient must not leave the hospital until the epiphyseal line has fused, either spontaneously or by operation.

Two procedures are available. Light traction may be continued on a frame or on a sliding bed for six to twelve months until fusion of the epiphyseal line is proved by antero-posterior and lateral films. The epiphysis may be fused by operation; the epiphyseal plate may be drilled in many directions, a tibial bone graft may be driven across it from the trochanteric region, or fixation secured by a flanged nail or screw exactly as for fractures of the femoral neck. The joint itself must not be opened for reasons mentioned previously. If metallic nails or screws are employed, they are usually removed when the epiphyseal line is fused.

The opposite hip must be kept under close observations as bilateral epiphyseolysis must always be suspected, and the patient should be instructed to report at once if he notices stiffness, pain, or limping on that side.

If necrosis of the epiphysis has already developed, treatment is based on the general principles mentioned in the management of avascular necrosis of the femoral head. In the child the prognosis is good, and a painless mobile hip may be secured by prolonged traction and freedom from weight bearing. Avascular necrosis in the adolescent should be treated by conservative measures which are successful in the child, and approximately one-third normal movement of the hip may be regained. But usually these hips give rise to disability later in life. In the adult the joint seldom survives, and an arthrodesis is usually necessary. However, if sufficient neck remains it is possible to assure the patient a stable weight-bearing extremity with a reconstruction operation by the method of Albee, Colonna, Whitman, and others.

If the upper femoral epiphysis is seriously displaced and its position is unchanged by heavy traction, the epiphysis has obviously begun to unite in its displaced position, and manipulative or operative reduction must not be contemplated until radiographic evidence shows firm epiphyseal fusion, and then the deformity is corrected by a subtrochanteric osteotomy. Correction of the deformity reduces the strain on the hip joint and delays the development of secondary arthritis.

In both Perthes' disease and epiphyseal displacement—in fact in any
epiphysiolysis affecting the lower extremity—one must constantly check the extremity length of the patient with serial roentgenograms made in the erect weight-bearing position which may produce a pelvic tilt, inclination of the sacral base plane, and altered vertebral mechanics in the superincumbent spine. If such deficiencies are corrected by means of suitable lifts applied to the heel and sole of the shoe of the affected limb, irreversible disabilities of the lumber spine and pelvis will be prevented and the patient saved for a much more useful life than is possible if these disabilities occur. Roentgenograms should be made at intervals of six months to a year until all growth in the lower extremity has ceased, and suitable correction made and re-checked with these studies throughout the years. When instituting this correction of a deficiency in extremity length, one should re-check within a period of four to six weeks on the effectiveness of therapy because many times the estimation of correction necessary may be insufficient or too great for the individual case. Men-suration of the limb may be employed, but there is too great a possibility of error for one to rely upon this means of measurement of extremity length.

**Osteochondrosis of the Vertebral Epiphyseal Plates**

A great many synonymous terms have been used to describe the condition in which the secondary centers of ossification of the vertebral bodies become affected. Probably it is known best as Scheuermann’s disease, but is also known as osteochondritis deformans dorsalis, vertebral epiphysitis, juvenile kyphosis, kyphosis osteochondropathia, vertebral osteochondritis, kyphosis dorsalis adolescentum, or kyphosis dorsalis juvenilis. The disease is usually self-limiting. Frequently, involvement of other epiphyseal structures may be observed in the same patient. Scheuermann’s disease is usually manifest between the tenth and twenty-fifth years, the peak incidence being from the fifteenth to the sixteenth years. Both sexes are usually affected about equally.

The onset is usually marked by extensive fatigue, backache, and pains in the limbs. Characteristically, the patient complains of tiredness in the back, the erect position aggravates the pain, while the recumbent relieves it. The musculature of the back is usually not affected until an advanced stage of the disease has been reached. There may be tenderness over the spinous processes and crests of the ilia. A deformity of the anteroposterior type develops, and the patient usually seeks medical aid for this kyphosis. The dorsal spine becomes rounded, and this gradually increases in severity.

The disease evidently has a limited cycle as evidenced by the x-ray changes; within a period of two to three years, the deformity becomes stationary, rigid, and the subjective symptoms completely disappear. The zone of the 10th to 11th dorsal vertebrae is that most completely affected.

There are three stages of the disease according to x-ray appearance. In the period of irritation, the vertebral epiphyseal plate appears rarefied, the outlines are frayed and moth eaten, the intervertebral spaces appear
irregular and mottled, and the adjacent vertebral borders are indistinct. During the period of destruction it is difficult to differentiate the intervertebral spaces from the bodies, as in the lateral view everything appears as a continuous fused mass. In the period of repair the vertebral outlines become more distinct, but are still irregular. The epiphysis now becomes differentiated and appears as a dense shadow although not returning to normal size. At the conclusion of the growing age this stage of ossification is also completed, and the progress of the deformity is checked. Typical Schmorl's nodes may be noted from the prolapse and herniation of the nucleus pulposus of the discs.

**Management:** In cases of advancing deformity accompanied by pain, the patient should be put to bed on a hard mattress and supported beneath the mattress with boards to prevent sagging of the bed, which would increase the deformity.

When the pain has been relieved, corrective supports should be employed and the patient allowed to become ambulatory. It may be possible in some cases to correct the deformity by the use of hyperextension while employing rest in recumbency, though in many instances this may not be possible. However, I feel that some attempt should be made to correct the kyphosis while the epiphyseal plates are still plastic, even though it may be necessary to employ anesthesia to relieve the pain of such procedures. Gradual hyperextension may be employed by inserting sand bags or other suitable elevation beneath the mattress of the bed.

After correction has been obtained, corrective supports should be given the back by means of adequate braces or plaster-of-Paris casts, and the patient allowed to become ambulatory. The braces are worn from one to two years, and then exchanged for a corset, with shoulder straps which assist in the maintenance of the proper posture. This support must be worn until ossification is completed, usually the twenty-fifth year. The musculature of the back which has become inefficient during the active period of the disease must be given attention. Massage, postural exercises, and especially deep breathing exercises within the limits of tolerance are most effective.

**Osteochondritis Vertebrae**

This condition differs from Scheuermann's disease in that the pathologic process affects the primary centers of ossification of the vertebral body. This condition was first described by Calvé in 1925. It is not very common, but the resultant deformity is very severe.

This disease occurs much earlier than Scheuermann's disease. It is characterized by slight pain in the back, fatigue, muscle spasms and tenderness, and occasionally by the phenomenon of night-cries.

The x-ray findings are rather distinctive. The vertebral bodies are irregular in outline, blurred, and fragmented. The intervertebral discs above and below the diseased vertebra remain intact, and in fact appear thicker than normal with evidence of new tissue causing the intervertebral space to appear wider. Deformity of the vertebral body occurs, developed
under weight-bearing stress and resulting in a wedge formation of the body.

This disease evidently has a limited cycle, as in vertebral epiphysitis, fragmentation and destruction ceasing in two to three years. Following this the bone returns to a state of greater density, repair and restoration sets in, the vertebral outline again becomes sharp, and the entire body of the vertebra appears more sclerotic.

**Management:** During the acute stage the patient should be recumbent, in a cast, in order to prevent deformity developing even while in bed. When the acute stage has passed, and the pain has ceased, the patient may be ambulatory with a plaster-of-Paris jacket which later is replaced by suitable braces to prevent further deformity from weight-bearing. Support to the spine must be worn until the period of ossification has been completed, and practically all these cases have some residual deformity of the spine in the form of kyphosis.

**Osteochondrosis of the Tarsal Scaphoid**

This condition, affecting the tarsal navicular, also known as Köhler's disease, is analogous to the other osteochondroses. It usually occurs in young children between the ages of three and ten, and affects boys more often than girls. A definite history of trauma is obtained in a minority of cases. The condition is analogous to the dystrophies known as Kienbock's disease affecting the carpal lunate, and Preiser's disease affecting the carpal navicular. The navicular is the last bone of the foot to ossify; and, as it forms the keystone of the inner longitudinal arch, it is subjected to considerable strain while in the cartilaginous state.

The clinical signs are usually those of a low-grade inflammatory lesion; however, the radiographic study of the lesion may be accidental. Slight pain and swelling in the region of the tarsal navicular are present, with the pain aggravated by weight-bearing causing the patient to limp, and to walk and bear weight on the lateral border of the foot. The affected region is painful on movement and tender on pressure. There may or may not be slight reddish discoloration in addition to the swelling.

**Management:** Until revascularization is complete the bone must be protected from weight-bearing by rest in bed for a period of several weeks, and then by rest and immobilization in a plaster-of-Paris walking cast for a period of from four to six weeks with the foot inverted and the arch well supported. Following the removal of the cast, x-ray study should be made to check on the effectiveness of treatment; and if healing has taken place, continued support of the arch by means of leather or rubber pad inserts will help take the strain off the longitudinal arch until such time as the symptoms disappear. It might also be helpful to wedge the inside of the sole and heel to divert the weight-bearing to the outer side of the foot until the symptoms disappear.

When the symptoms have subsided, exercises to strengthen the muscles of the foot and legs, massage, and physiotherapy may be instituted.

Occasionally, irregular extrusion on the dorsum of the navicular is
seen, which later may require removal because of the pain and of the inability to wear proper shoes.

Recovery is usually complete in a period of eighteen to twenty-four months.

**Osteochondrosis of the Tibial Tubercle**

This condition, commonly known as Osgood-Schlatter’s disease, is a separation-fracture or an osteochondrosis of the tibial tubercle. This condition is found more frequently in boys; the usual age is from twelve to fifteen years; and trauma is nearly always a distinct feature of the history.

The tibia is developed from four centers of ossification, one for the shaft, one for the lower end of the bone, and two for the upper end of the bone. Any or all may be affected.

The tuberosity of the tibia arises as a tongue-like protrusion from the lower end of the upper tibial epiphysis, but it may have two centers of ossification, one extending down from the upper epiphyses, and one reaching up from the shaft.

Partial separation of the tuberosity from trauma, such as violent contraction of the quadriceps muscles, occurs mostly in individuals indulging in strenuous sports. There is usually immediate pain over the affected site, aggravated by any attempt to straighten the knee. The tuberosity is tender and swollen, and roentgenograms show the detachment of the tongue-like epiphysis.

The anatomical development of the tibial tubercle is quite variable in form, and in the absence of a clinical picture as mentioned above, diagnosis is questionable.

While in most cases injury may be an exciting factor, it probably does not play an important part. The onset of pain and tenderness is insidious. The patient may complain of some aching in the front of the knee after any exercise, which is increased by full voluntary extension of the joints since the affected epiphysis is then pulled on by the quadriceps muscles. There is also pain on passive flexion. The epiphysis is tender, and in many cases there is some localized edema.

The x-ray appearance is not always characteristic and is not easy to diagnose with confidence. In this, as in other osteochondrotic processes, it is important to make serial studies. When the condition is bilateral, it has been noted that the side showing the greater radiographic signs does not necessarily manifest more marked clinical signs.

This condition must be differentiated from osteomyelitis, sarcoma of the head of the tibia, bone cysts, and infra-patellar bursitis. The first three offer little difficulty, but infra-patellar bursitis may be difficult to distinguish unless fluctuation is present. Aspiration of the bursal fluid indicates the source of the trouble.

**Management:** Immobilization of the knee in almost full extension by plaster-of-Paris casts for eight to twelve weeks should be established. After six weeks, a walking iron may be applied to enable the patient to
be ambulatory with the aid of crutches. Protection must be continued until relief of the pain and tenderness is experienced and radiographic evidence of re-attachment of epiphysis appears. A brace, applied with a lock joint at the knee to prevent undue flexion when weight-bearing, is worn until the symptoms subside. Physiotherapy may be applied to stimulate the circulation about the parts.

If there is considerable tenderness and pain, rest in bed is indicated until they have subsided, even while immobilized in the cast. Flexion of the knee joint should not be allowed until the symptoms have subsided, and violent exercises should be prohibited for a period of from six to nine months.

Recurrences are common, and the cure is never complete until the epiphysis joins with the tibia. Complete restoration of the tuberosity to normal is usual.

Osteochondrosis of the Metatarsal Head

This condition is also known as Köhler’s disease No. 2, Freiburg’s infraction, or subchondral osteolytic deformity of the metatarsal head. The epiphysis of the second metatarsal head is much more often affected than the heads of the other metatarsals. In this condition there is great broadening of the metatarsal head, stiffness, swelling, and pain in the metatarso-phalangeal articulation.

While the usual age for the condition is from twelve to fifteen years, it may arise at any age and is not confined to the period before the epiphysis is fused. This fact gives rise to doubt that the condition is primarily a true epiphyseal lesion. Freiburg contends that the condition is traumatic, and that the primary condition is a traumatic fracture or infraction of the articular surface. However, as the second metatarsal is most frequently affected and the condition is associated with developmental abnormalities which overload the middle group of the metatarsals, it is natural to assume that repeated trauma is referred to the head of the metatarsal and epiphysis causing them to undergo necrotic change as the result of circulatory disturbance. The condition occurs more frequently in girls than in boys; and, as a rule, trauma plays an important part in the history.

In the acute stage, when the articulating surface is collapsing, there is extreme pain in the foot, movements are painful, and there may be considerable edema of the forepart of the foot, especially marked on the dorsum. Later, when collapse is complete, the pain may subside, leaving a deformed joint which is slightly limited in motion. There is usually some palpable enlargement of the metatarsal head, and occasionally some reddish discoloration over the swelling.

The x-ray changes are characteristic. Early, there is a slight sub-capital osteoporosis, and the toe is usually “clawed” or elongated. There is broadening of the metatarsal head, which is irregular and flat in contour, with indentations or collapse of the articular surface. Increase of the joint space is seen. Thickening of the shaft of the metatarsal is in
evidence. One feature not commonly seen in osteochondroses elsewhere is that the detached portions of the articular surface may lie free in the joints, as a true dissecans type.

Management: In the acute state, bed rest and immobilization by plaster-of-Paris cast is usually employed, serial roentgenograms being taken at intervals of eight to twelve weeks to determine the progress of healing. These patients may be fitted with a well-leg lift and crutches to avoid any weight-bearing on the affected foot.

In the latter stages, if stiffness and pain persist when the cycle of pathology is complete, relief can be obtained by excision of the affected metatarsal head. Usually, complete relief will be afforded by this procedure, and restoration of function in the joint approaches that of normal, although there is some shortening of the toe which does not materially affect the function of the foot.
PULMONARY EMBOLISM: A REPORT OF TWO CASES
Otterbein Dressler
Professor of Pathology

Pulmonary embolism is one of the most devastating hazards of major surgery and obstetrics. The suddenness with which it strikes, without warning, coupled with the brief life expectancy makes it one of the most dramatic incidents of hospital practice. For all his skill it would seem that at best all the surgeon can do is stand helplessly by and watch his most brilliant success end as a most dismal failure. Like many another circumstance in disease, here is a process that repeats itself over and over again; yet we seem no closer to the solution of the problem than were our predecessors. It is well, therefore, that we just as frequently review the ground in the hope that we might, sooner or later, find that ray of light which might guide us to a satisfactory solution to this terrifying problem.

Autopsy No. A-43-264
Died: 2-27-43, 9 p. m.
Autopsied: 2-28-43, 9 a. m.

Clinical Data

Case I
An adult, married, housewife, said to be 48 years of age, was admitted to the hospital on February 2, 1943 at 3:45 p. m., complaining of a vaginal discharge and some backache. The vaginal discharge was said to have continued for the past 35 years. The patient stated that she had menstruated continuously from October, 1942 to the first week in January, 1943, but since that time there had been no bleeding. The patient further stated that she noted some irritability and some hot flashes, suggestive of the menopausal syndrome. She was gravid II, para II.

The usual laboratory examinations failed to reveal any abnormal data. An internist examined the patient and was of the opinion that she was an average surgical and anaesthetic risk.

On the morning of February 20, under spinal anaesthesia a panhysterectomy was performed with the removal of fibroids, and the appendix was taken en passant. The surgical pathological report will be found under number SP-43-9074.

The patient responded well and was on the way to recovery. On the evening of the seventh post-operative day she was reported by her visitors to be in good spirits and apparently doing very well. Immediately after the visitors left, the following final progress notes were entered by the intern: “Called about 8:45 p. m. to see the patient who had suddenly begun to feel ill. Her hands were cold and clammy; the pulse was thin and thready; the pupils dilated gradually. The patient thrashed about somewhat as if in pain. Difficulty in catching the breath was evident. In rapid succession oxygen and spirits of ammonia were inhaled
and the latter given orally. An intravenous of 1000 cc. of 10 per cent glucose and saline was administered. One cc. of coromine was given intramuscularly, followed by 2 cc. of coromine intramuscularly. Cyano progressed, and the patient expired at 9 p. m.”

**Autopsy Protocol**

The body was that of a well nourished, adult female, said to be 48 years old. She was of the short, heavy type and presented a midline incision from the umbilicus to the pubes, said to be seven days post-operative for hysterectomy.

Fifty cc. of clear amber fluid was found in the pericardium. The pulmonary artery was opened and a large thrombus curled up at its bifurcation removed. A similar large thrombus was found in the right atrium blocking the tricuspid valve. The heart measured 13 x 10.5 x 6 cm. and weighed 240 grams. The myocardium was soft and thin.

There was no fluid found in the pleural cavities. The lungs weighed, right and left respectively, 350 grams and 330 grams. There were no noteworthy changes in these organs.

Some adhesions were demonstrated in the region of the recently removed appendix, and the cecum was becoming adherent to the sigmoid. The sigmoid was greatly distended and crowded the pelvic cavity. A suture was noted between the sigmoid and the parietal peritoneum of the brim of the pelvis anteriorly. There were no noteworthy changes in the digestive tube.

Bile flowed freely from the gallbladder upon pressure. The liver weighed 2030 grams and was very friable.

The spleen measured 13 x 9 x 3 cm. and weighed 150 grams. The splenic pulp was comparatively soft.

The urinary bladder contained 200 cc. of urine. The kidneys measured, respectively right and left, 11 x 6 x 3 cm. and 10.5 x 5 x 3 cm. and weighed, respectively right and left, 160 and 130 grams. Fetal lobulation was demonstrated. The ureters were not distended. The suprarenal glands presented no noteworthy changes.

The mammary glands presented no noteworthy changes but sections were taken for histological study.

An anomaly of the vena cava was demonstrated. The inferior vena cava bifurcated above the region of the kidneys into two lateral vessels corresponding to common iliac veins. The left renal vein branched off from this left common iliac vein. As these approached the brim of the pelvis, the left common iliac artery crossed the left vein; and at this point a thrombus was found in the vein and also in its branches. We believe this was the source of the large thrombus found in the heart and in the pulmonary artery.

**Anatomic Diagnosis:** Pulmonary Embolism

*Anomaly of the Inferior Vena Cava and Common Iliac Veins*
Autopsy No. A-43-267
Died: 3-12-43, 9:35 a. m.
Autopsied: 3-12-43, 4 p. m.

Clinical Data

An adult, married, very well developed male, said to be 62 years old was admitted to the hospital on March 1, 1943 at 10 a. m. complaining of increased frequency of urination. He stated that this condition had been present for the past year and was gradually becoming worse. He further stated that he had some burning on urination but no pain, and that there was also trouble in starting and, particularly, in stopping the flow of urine. He further stated that there was some dribbling after urination.

The physical examination of the patient revealed a blood pressure of 130/86 and cardiac sounds that were described as being of poor quality. Bronchial breathing was described over the lungs with some fine rales.

Laboratory examinations showed a low specific gravity of urine, 1.003, with an output of total solids of only 7.98 gms. per L. There was no knowledge of the total 24 hour quantity. The blood picture was average and the serology by compliment fixation and precipitation was negative for syphilis.

On March 3, 1943 prostatectomy was performed under spinal anaesthesia with the use of pontocaine. A drop in blood pressure was described and on being returned to the room, the foot of the bed was elevated until the pressure returned to his normal. A Pilcher bag had to be re-filled because of bleeding on the second post-operative day.

On the morning of the ninth post-operative day the patient's condition was reported as "good" at 9:25 a. m. A few minutes later he complained of pain in one leg, and at approximately 9:30 became cyanotic, gasped for breath and became unconscious. Emergency methods were instituted, but the patient died at 9:35 a. m.

Autopsy Protocol

The body was that of a very well nourished male, said to be 62 years old. A superpubic incision of recent date was noted but showed good healing. There was some swelling of the right leg and thigh, more marked in the right groin. There was a suggestion of cyanosis about the face.

The pericardial sac contained less than 50 cc. of clear amber fluid. The right atrium was much distended. The pulmonary artery was blocked by a thrombus, which gave the physical appearances of having been derived from a large vein. This thrombus extended into the branches of the pulmonary artery and also continued in the right ventricle with some strands in the right atrium. The heart measured 14 x 12 x 7 cm.
and weighed 380 grams. The myocardium was well preserved. There was some atheromatous infiltration into the mitral curtain.

The thorax was of the emphysematous type and the lungs were emphysematous except for a well defined infarction involving the right lower lobe. This infarction we would judge to have been present for some days.

The esophagus was dilated with some gastric contents and the stomach was somewhat similarly dilated. There were no noteworthy lesions of the intestinal tract except for some distention about the cecum.

The gallbladder emptied with some difficulty upon pressure.

The liver weighed 2100 grams and showed some small thrombi in the portal veins.

The pancreas weighed 210 grams and showed a small lesion, evidently of old fat necrosis.

The spleen measured 14 x 6 x 3 cm. and weighed 240 grams. The splenic pulp was somewhat soft, and an accessory spleen was noted in the mesentary.

The great omentum was attached to the undersurface of the suprapubic incision. The deeper portions of the suprapubic incision emitted a urinous odor suggesting leakage into the loose fat and muscle layers.

The urinary bladder showed the defect of recent prostatectomy. The ureters were not dilated. The kidneys measured respectively, right and left, 12 x 5.5 x 3 cm. and 14 x 6.5 x 5.5 cm. They weighed respectively, left and right, 260 and 160 grams. A central solitary cyst 2 cm. in diameter was found in the left kidney. The suprarenal glands presented no noteworthy changes.

The great vessels of the pelvis and the thighs were explored but no further evidence of thrombosis could be demonstrated.

Anatomic Diagnosis: Pulmonary Embolism.

Discussion

Two more or less typical cases of pulmonary embolism are presented with autopsy findings. Cases such as these are so common and our knowledge so scant that we are apt to regret the misfortune of the patient and the doctor and place the record in the file.

In both instances the emboli responsible for death were dislodged thrombi from great vessels in the abdomen or lower extremities. It is interesting to note that thrombosis and clotting of blood are still thought to be synonymous by students and surgeons alike. The recent attempts to prevent these catastrophies of embolism, such as the intravenous use of anti-coagulants, etc., have been chiefly directed to the prevention of the clotting of blood. The facts are that thrombosis is not the same as blood clotting; the mechanism is somewhat different.

The classical etiological factors in thrombosis are (1) disease of the vessel walls (inflammation, sutures, injury), (2) alteration in the current of blood (whirls, stasis, etc.), (3) and changes in the blood itself (chem-
ical changes and viscosity). The last factor is the least important, yet this is the factor attacked by chemical methods. By all odds the first two factors are the important ones.

In the first case a short, heavy female was operated upon and then placed back into bed. In this type of individual, if the thighs are approximated, pressure is made upon the great vessels and coagulation facilitated by factor two above. These patients might well be placed in bed with the thighs separated in an effort to prevent thrombosis. In this case, however, the problem is complicated by an anomaly of the vessels of the abdomen which may have been an important factor.

In the second case, a patient in circulatory collapse was placed in the customary position with feet elevated and head depressed. It is postulated that under these circumstances in such cases the plantar vessels collapse and, therefore, facilitate intra-vascular coagulation. That small emboli became impacted in the pulmonary vessels before the major catastrophe in this case is evidenced by the older infarction noted above in the autopsy report.
A BACKGROUND FOR RATIONAL THERAPY
Guy S. Deming
Associate In Research

It is desirable at the outset to establish certain concepts which shall be basic to subsequent discussion.

The human organism is the physiological unit under discussion. This whole has at all times to meet the vicissitudes of life as a unit. How successfully it does so depends upon a multiplicity of factors within and without the organism itself. Health and disease are manifestations of relative success in meeting the demands of living, and life, in the words of Herbert Spencer, is "the continuous adjustment of internal relations to external relations." The concepts of health, of disease, and of therapy developed in this discussion are based on the ancient recognition of the healing force of nature given a modern interpretation by such men as Bernard, and Cannon.

Nearly one hundred years ago Claude Bernard called attention to the fact that organisms live in two environments. The organism as a whole is enveloped by the same external environment which surrounds inanimate objects. But every living cell of the body is bathed by and can live only because of the presence of circulating body fluids. This "totality of the circulating fluids of the organism" Bernard called the internal environment. He pointed out that it is the blood and interstitial lymph which provides appropriate and favorable surroundings for the living cells of the organism; that they are highly important factors in establishing and maintaining steady states in the body; and that they are under the control of agencies which keep them remarkably constant. He said further that this constancy of internal environment is absolutely essential to a "free and independent life", and that "all the vital mechanisms, however varied they may be, have only one object, that of preserving constant the conditions of life in the internal environment." Cannon in his book "The Wisdom of the Body" termed this constancy homeostasis. This is what he says: "The constant conditions which are maintained in the body might be termed equilibria. That word, however, has come to have fairly exact meaning as applied to relatively simple physico-chemical states, in closed systems, where known forces are balanced. The co-ordinated physiological processes which maintain most of the steady states in the organism are so complex and so peculiar to living beings—involving as they may, the brain and nerves, the heart, lungs, kidneys and spleen, all working co-operatively—that I have suggested a special designation for these states, homeostasis. The word does not imply something set and immobile, a stagnation. It means a condition—a condition which may vary, but which is relatively constant." Two brief but pregnant statements can be distilled from this description: Homeostasis is the relatively steady state of the internal environment of living organisms necessary for life. It is maintained by the co-ordinated physiological processes peculiar to living beings.
It must be clearly understood that homeostasis means relative constancy. There are oscillations about a mean, and in health these oscillations remain within limits which constitute the range of variation compatible with continuing function at optimal levels of the living elements of the body. If these limits are exceeded, the result may be irreversible changes in groups of cells which may threaten life. This is illustrated by variation in the pH of the blood which is normally approximately 7.4, just on the alkaline side of neutrality. If the pH falls to 6.95 coma and death may result. An increase to 7.7 may bring on tetanic convulsions.

Cannon has distinguished two general types of homeostatic regulation. One involves the temporary storage of materials, their release for use, and the riddance of excess by overflow. The other involves regulating the rate of continuous processes. In both types of regulation, the balance is known to be extraordinarily delicate and subject to change under the influence of factors constantly operating within the organism and arising in the environment. That the conditions of internal economy essential to life are kept within a safe range indicates the existence of mechanisms for the continuous adjustment of the organism to its environment.

There are two general classes of reactions involved in preserving homeostasis: Those which respond after an external agent has begun to operate, and those which anticipate the need for response. In the first category are such rapid reflex reactions as coughing, sneezing, and vomiting to rid the body of noxious agents, and the slower reactions of organ repair, the gradual increase of erythrocytes at high altitudes, the formation of antitoxins and antibodies. The second, or anticipatory class of reactions is observed when emotional stimuli prepare the body for unusual effort. At such times, marked changes occur in respiration, heart rate, blood pressure and distribution, digestive activity and all the other modifications which sum up to a suppression of the presently unnecessary and a reinforcement of the immediately advantageous body processes in a situation which may involve a life and death struggle. These changes constitute a departure from the ordinary homeostatic levels but are purposeful. They mobilize the body’s present resources for augmented muscular exertion. If struggle or flight supervenes, mechanisms respond which tend to stabilize the organism at a higher level of output. The changes wrought in the internal environment are utilized as intended, they support the higher level of energy production, and are neutralized by it. Subsequently, restorative processes come into operation, and the organism is returned to its former metabolic level. If, however, the potential energy evoked is not utilized according to the pattern established by evolution, it still must be dissipated; and if this situation occurs repeatedly, it can create grave disturbances in body function. Instances of such perversion of natural responses are documented by research in psychosomatic medicine.

The mechanisms which operate to maintain homeostasis are con-
cerned with ensuring an effective fluid matrix for all the living elements. They include hunger and thirst to assure adequate supplies of food and water; a combination of chemical and nervous stimuli which assures adequate oxygen supply; and nervous, chemical and hormonal agencies acting alone or in combination which maintain homeostasis, for example, of salt, sugar, proteins, fat, and calcium in the blood, of the pH of the blood, of body temperature, and of the rate of oxidation.

Every homeostatic mechanism of the vertebrate organism represents in one way or another the physiological functioning of the two main integrating systems of the body—the nervous system and the blood circulatory system. Each is a co-ordinator of bodily activity. Each is indispensable to a “free and independent life”; the nervous system because it is the special conduction apparatus between receptors and effectors, the blood circulatory system because it transports oxygen, food, and hormones to every cell, and carries waste products away from the cells for excretion. Between the two integrating systems—blood stream and nerves—is a reciprocal relationship of the greatest importance. The nervous system—brain, spinal cord, and peripheral nerves—like every other organ in the body is dependent on its blood supply. But the blood supply is dependent upon a normally functioning nervous system. The blood flow to the various organs, including the nervous system itself, is largely regulated by vasomotor centers of the brain and spinal cord. This intimate, reciprocal relationship between these two great coordinators of total vertebrate physiology is indispensable to success in meeting the vicissitudes of life.

The justification for the above statement derives from the evidence that in the presence of environmental changes which affect the organism as a whole the homeostatic mechanisms maintain that relatively constant state of the internal environment without which the organism cannot continue effective. Because they do this, Cannon considers the mechanisms protective in function—protective because they institute reactions which enable the organism to adapt its total function to meet the demands imposed by its environment and so maintain life at a favorable level. For these reactions to be useful they must be promptly initiated—they must not require conscious thought—at the level of uninhibited physical activity they must concentrate the available resources of the body to support greater voluntary muscular effort. Finally and inclusively they must relate the internal to the external environment, implement the responses to the demands.

The mechanism which satisfies all these criteria is the neuro-humoral system. It mediates visceral responses and adjusts the internal environment to make possible a successful adaptation of the whole organism to the constellation of forces which play upon or within it. The neuro-humoral system is indispensable to full exercise of all the biologic drives of the organism because the nervous system is the mechanism which functions in both great fields of activity and relates the internal to the external environment.
In one field the nervous system acts outwardly by a quickly responsive exteroceptive apparatus planned for action with striated muscles as the effector organs. This is the somatic nervous system which, in Gellhorn's words, "regulates by means of locomotor and postural reflexes and actions, the relation of the organism to its environment." As a functional unit this apparatus is made up of the central nervous system and the peripheral nerves which reach the outer surface of the body, the special sense organs, the joints, and the striated muscles. It includes afferent nerves for sensation and motor nerves for action. This arrangement equips the organism for prompt, appropriate response to its immediate environment which it may conceivably alter by labor, or escape from by locomotion.

In the other field the nervous system acts inwardly by a more slowly responsive interoceptive apparatus planned for action with smooth muscles and glands as the effector organs. This is the part of the nervous system which regulates visceral activity by means of vasomotor, visceromotor, and secretomotor reflexes and actions to the advantage of the organism as a whole. As a functional unit this apparatus is made up of the central nervous system and the peripheral nerves which reach throughout the visceral zone. The peripheral representations include afferent nerves, and the motor nerves which comprise the autonomic nervous system. When activity of the exteroceptive system disturbs the fluid matrix of the body it is the function of the autonomic nervous system to assist in maintaining homeostasis.

The co-ordination of all nervous system activity is effected by the central nervous system which is common to both the arbitrarily designated exteroceptive and interoceptive systems. It is the central nervous system which plays the essential role of distribution and integration. Within the central nervous system afferent impulses originating anywhere in the body regardless of type or location of receptor can be distributed by association neurones to higher centers, to efferent somatic neurones, or to autonomic neurones. Within the central nervous system afferent impulses are integrated and motor actions are modified or conditioned by the sum of the afferent impulses converging on the efferent neurone cell bodies.

The regulation of the internal environment carried out by the autonomic nervous system is accomplished by the combined action of its two divisions—sympathetic and parasympathetic. As experimental physiology increases the knowledge of these two homeostatic regulators it becomes clearer that they should be considered not as antagonists in the old sense but as synergists responding simultaneously which by shift in predominance of their opposite effects on individual organs maintain dynamic equilibrium between demand upon and response of each organ necessary for effective functional integration of the whole organism.

Health of the organism as a functionally integrated whole means success in making responses necessary for life at optimal levels—success
in meeting the demands of living—success in making “the continuous adjustment of internal relations to external relations.” And disease means the loss to some degree of the power of functional integration—the loss of appropriate responses to adverse circumstances—the loss of appropriate adjustment of internal to external environments. Since in disease the response may be either inadequate or excessive the object of therapy is to assist the recovery of the most suitable response attainable—to stabilize the internal and external environments at suitable levels. The physician has two main avenues of approach: Restore function to the optimal level, alter environment. In practice he will do both in varying ratio as his judgment indicates in each individual case.