dent that the eye does not focus a point sharply upon its retina, but rather as a small group of points, each one of which is seen somewhat expanded and distorted, or, in

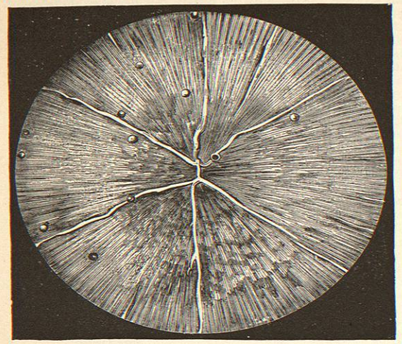

FIG. 375.

the case of a bright point, as a star-shaped figure made up of a number of points more or less completely fused together and rendered still more complex by innumerable fine radiating lines.

Thus, a brilliant fixed star or planet is seen as of very conspicuous magnitude, whereas the image of a fixed star in the most powerful telescope is but an intensely bright point of inappreciable diameter. A white dot, line, or letter on a black ground is similarly seen expanded, and is therefore visible at a greater distance than a black object of the same size upon a white ground; on the other hand, the form of the black object on a white ground may often be recognized at a greater distance than that of the bright object on a black ground. This phenomenon is called *irradiation*. The phenomena of normal irregular astigmatism are complicated also by aberration of curvature, in so far as the configuration of the cornea and of the crystalline lens varies from the theoretically perfect curvature requisite for the refraction of incident rays to a single focus, as well as by the slight inequality of refraction in different meridians, which is demonstrable in almost every eye, and which must, therefore, be considered as

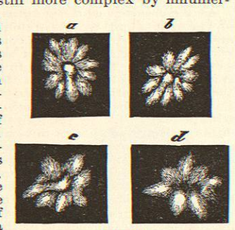

FIG. 376.

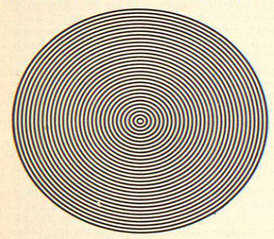

FIG. 377.

normal regular astigmatism. Hence the retinal image of a star is actually seen a little more expanded than it would be as a result of the scattering of the rays of light in traversing the crystalline lens, and, in the presence of regular astigmatism, the star is also seen elongated.

Abnormal irregular astigmatism attains perhaps its highest grade in conical cornea (*staphyloma pellucidum*), in which affection it may be a source of very great confusion of vision. Inflammatory processes in the cornea may lead to softening and consequent irregular distention of that tissue; and changes of curvature, both partial and total, may follow the cicatrization of corneal wounds, incisions made in operations, ulcers, etc. In all these cases the disturbance of vision may often be resolved in part into regular astigmatism, and sight may then be materially improved by wearing appropriate cylindrical glasses; in other cases the definition of objects is much improved by looking through a small hole or narrow slit punched in a blackened card or in a thin plate of metal; in rare instances it may be justifiable to attempt to change the position of the pupil by the operation of *iriddesis* (Critchett).

Abnormal irregular astigmatism, with multiple vision, may occur as a result of changes in the crystalline lens, incident to the incipient stages of cataract; myopic refraction also may be developed at the same time, probably through an increase in the curvature of the lens surfaces. *John Green.*

ASTRINGENTS.—Astringents—from *ad* (to) and *stringo* (I bind)—are agents which, acting locally, produce condensation and corrugation of tissues by precipitating their contained albumin and gelatin, and by diminishing the amount of fluids present in protoplasm. They also cause contraction of living muscular fibre, possibly by direct irritation. Secretion from mucous membranes and from denuded surfaces is lessened by astringents, which produce a constricting effect upon the capillary blood-vessels and also perhaps at the same time upon the glands and their ducts. All the astringents except alcohol produce some sort of chemical action which promotes destructive metamorphosis. Alcohol, on the other hand, retards these retrograde changes in the tissues. The subdivision into vegetable and mineral astringents is recognized and their action is either local or remote. Astringents are said to act locally, when they are applied directly to a part upon which it is desired to produce this particular kind of effect. On the other hand, they act remotely, when they are taken into the circulating blood and are thus brought in contact with certain internal (remote) organs. Among the astringents, which are used in this way are dilute sulphuric acid, gallic acid, lead acetate, etc. According to some authorities, this mode of action of astringents is precisely the same as that which takes place, when the remedy is applied locally in the ordinary, direct manner. Intestinal and urinary astringents are terms, which are applied to certain drugs, which exert a special astringent influence upon these organs of the body; the former contracting the walls of intestinal vessels and constringing the intestinal mucous membrane, while the latter manifest their influence mainly by diminishing the excretion of urine. Astringents are administered internally in the form of solutions, pills, or powders.

Vegetable astringents depend for their action upon the contained tannic and gallic acids. Tannic acid is said to be digallic acid anhydride. It is a crystalloidal substance, a glucoside, having the formula $HC_{14}H_8O_9$. It combines with colloids, precipitates pepsin, and coagulates albumin and gelatin. In this respect it differs from gallic acid, which does not coagulate either albumin or gelatin, and which is therefore better adapted for internal use. In fact, before tannic acid can be absorbed it must be converted, in the system, into gallic and pyrogallic acids. Arranged alphabetically, the vegetable astringents are: alnus, castanea, catechu, diospyros, galla, geranium, granatum, hamamelis, hæmatoxylon, heuchera, kino, krameria, myrica, nymphea, quercus alba,

rhus glabra, rosa glabra, rubus statica, uva ursi, and all other substances which contain tannic acid. Among the mineral astringents may be mentioned: the dilute acids (acetic, carbolic, muriatic, nitric, sulphuric), alcohol, alum, bismuth subnitrate and other bismuth salts, cadmium sulphate, chalk, cocaine, cerium oxalate, copper sulphate, creosote, ferric chloride and ferric persalts, lead acetate and subacetate, zinc preparations, especially the oxide and the sulphate, and several other metallic salts.

Astringents are valuable styptics and hæmostatics, and they also harden and restore tone to relaxed tissues. They cause capillary vessels to contract, and they constrict glands and their ducts. They exert some control over inflammation and they diminish the secretion from mucous membranes and from denuded surfaces. They excite contractions in muscular fibre, and they cause spongy granulations to wither away. When applied to an ulcerated or denuded surface they bring about (through coagulation of the protoplasmic albumin) the formation of a pellicle which covers and protects this surface from the atmosphere and from external irritants. Thus, pain is lessened at the same time that healing is promoted by astringents. When they are administered internally, their action is either local, or, like tonics, which they somewhat resemble, they impart vigor and tone to various structures of the body. Thus, upon the nervous system they may exert a decided and oftentimes a beneficial influence. They diminish peristaltic action to some extent. With three exceptions all astringents irritate more or less. They are therefore contraindicated in acute inflammation. The three sedative astringents are lead acetate or (subacetate), cerium oxalate, and bismuth subnitrate.

Synergists: Tonics, especially the bitter tonics, also those agents which increase retrograde metamorphosis.

Antagonists and Incompatibles: Vegetable astringents are incompatible with the "ic" and "ous" salts of iron, also with the salts of antimony, copper, lead, silver, and zinc; with alkalies, alkaloids, and glucosides; and with pepsin, albumin, gelatin, emulsions, and the mineral acids.

Manner of Elimination from the System: Tannic acid is excreted by the bowels as gallic or pyrogallic acid. It is also eliminated by way of the kidneys.

Uses and Therapeutic Applications: To check excessive secretion from the skin, as in hyperidrosis or in night sweats; to check secretion from mucous membranes, as in the various catarrhs (nasal, buccal, bronchial, intestinal, urethral, vesical, vaginal, etc.); to lessen secretion from denuded and ulcerated surfaces. It must always be remembered that astringents are not to be used until the inflammation reaches that stage in which the secretion from the inflamed part is beginning to be excessive. Capillary oozing or hemorrhage from some remote organ, as from the kidney or bowel, may be controlled by the astringents; gallic acid being preferable to tannic in such cases. Diabetes insipidus and albuminuria are other pathological conditions in which the use of gallic acid is indicated. Where the part can be directly reached, as in epistaxis, hæmatemesis, hemorrhage from lower bowel, hemorrhoids, rectal fissure or ulcer, prolapsus ani, subacute or chronic conjunctivitis, otorrhœa, etc., tannic acid is preferable to gallic. In bed sores or where excoriation is taking place, as in dermatitis intertrigo, alcohol, bismuth, or tannic acid will be found useful as a means of hardening the skin. Finally, since tannic acid is chemically incompatible with the alkaloids and glucosides, it may serve as a useful chemical antidote in poisoning from these active principles. It accomplishes this good effect by throwing down a very slowly soluble, or entirely insoluble, therefore inert, tannate of the alkaloid or glucoside in question. *Leon L. Solomon.*

ATAVISM. See *Reversion*.

ATELECTASIS.—(Synonyms: Apneumatosis; collapse of lung.) The term *atelectasis* (ἀτελής, imperfect, and ἔκτασις, dilatation) is used to designate all non-inflammatory conditions by which either the whole or sharply defined portions of the lungs are undistended by air. Two forms are recognized: congenital and acquired atelectasis.

In some new-born babies, more or less extensive areas of the lungs are unexpanded by the forcible extrance of air into the alveoli. This condition, which is normal in fetal life, becomes pathological when it continues after birth, and is named *congenital atelectasis*.

In other cases, although the respiratory functions have been thoroughly established, collapse is induced as a consequence of some mechanical impediment to the movement of air through the bronchi; and a tract of lung of variable extent becomes again condensed and airless, as in the fetal state. This is called *acquired atelectasis* or *collapse of the lung*. There are two varieties: collapse from obstruction and collapse from compression.

Atelectasis is comparatively rare in adults, but is quite common in infancy and childhood, especially during the first few months of life. A considerable percentage of the mortality in infants is attributable to this cause. The liability to the occurrence of pulmonary collapse adds gravity to all diseases, but especially to those of the respiratory organs at this period of life.

ETIOLOGY.—Congenital atelectasis is not commonly due to vice or disease of the pulmonary organs, but is produced by any condition which prevents the prompt and efficient establishing of the function of respiration after birth. It may be the result of causes which have been in operation during the intra-uterine life of the child, or which have originated during or immediately succeeding birth. Physical weakness, premature birth, placental separation, compression of the cord, protracted labor, and kindred conditions are common causes of atelectasis. It is also not infrequently due to plugging of the bronchioles by liquor amnii and mucus, sucked in by efforts at respiration before the head has cleared the maternal passages. Intracranial effusions pressing upon the pneumogastric, the result of severe protracted or instrumental deliveries, may be placed among the rarer causes of this affection in the new-born.

Atelectasis from obstruction is always secondary to some disease or accident which interferes mechanically with the access of air to the lung cells. The lodgment of a foreign body in a bronchus may result in alveolar collapse. In the vast majority of instances this impediment is the presence of mucus in the bronchial tubes, the effect of an acute or chronic bronchial catarrh, and collapse is therefore a frequent complication of those diseases, like pertussis and measles, in which bronchitis is a part of the natural history.

Whenever one or more terminal bronchioles are occluded by viscid mucus and swelling of the mucosa, the collapse of that portion of the lung fed by the obstructed tube inevitably takes place as soon as the imprisoned air is expelled or absorbed. This purely mechanical explanation of collapse, first advanced by Gairdner of Edinburgh and adopted by nearly all writers on the diseases of children, has been challenged by Holt and others.

Weakness of the inspiratory muscles, and the consequent inability to overcome the obstacles in the tubes, is a powerful auxiliary factor in bringing about collapse, and hence any condition which decreases the physical vigor of the child strongly predisposes to this accident. It is, therefore, a common malady among those enfeebled by a bad inheritance, by chronic and wasting diseases, or by unsanitary surroundings. Rickets also plays an important rôle in the causation, associated as it is with softening of the ribs and narrowing of the thorax.

External pressure may render a lung, or any portion of it, airless (*atelectasis from compression*). Intrathoracic growths or exudations, spinal deformities, and upward displacement of the diaphragm by abdominal tumors or effusions may cause collapse of such portions of the lung as are subjected to pressure.

MORBID ANATOMY.—The collapse may involve considerable areas of the lung (*diffuse atelectasis*), or it may be

limited to small and scattered patches (*lobular atelectasis*). These varieties are found in both the congenital and the acquired forms of the disease, but in the former the lesion usually involves larger tracts of tissue, the half, or even the whole, of one lobe; it is most frequently observed in the posterior and inferior portions of the lungs, in the tongue-shaped projections, and in the apices; while in acquired atelectasis the patches are oftener limited to isolated lobules or groups of lobules, and are more widely disseminated through the parenchyma of both lungs.

The collapsed portions are depressed below the general surface of the lung, feel tough and dense, like soft leather, and are of a dark blue or steel color. They are airless, do not crepitate upon pressure, and sink readily when thrown into water. When they are incised, the section is smooth, non-granular, and, if scraped, exudes a small quantity of bloody serum. After death, if the lesion is recent, the atelectatic portions can be readily inflated through the bronchus, and instantly assume the color and qualities of normal lung; but after some time has elapsed, they undergo changes which destroy their dilatability, and eventually end in the total disappearance of the vesicular structure. The pleura is normal in uncomplicated cases.

When a considerable tract of lung is disabled, important changes ensue in the unaffected tissues and also in the organs of circulation. Pulmonary emphysema is a common sequel. The impediment to the movement of the blood through the lungs results in stasis in the pulmonary artery and the entire venous system, and leads to hemorrhagic infarctions and œdema of the unaffected lung tissue. The same condition also tends to prevent, in congenital cases, the closure of the fetal channels of circulation, especially the foramen ovale.

Symptoms and Course.—The symptoms of atelectasis are chiefly those of "inefficient breathing and incomplete decarbonization of the blood." They exhibit varying degrees of severity in proportion to the rapidity of development and the amount of lung tissue involved. When the collapse is limited to scattered lobules, the symptoms are by no means marked or distinctive. But, on the other hand, if it be so extensive as suddenly to arrest the function of a large part of both lungs, death may take place almost instantly. This occasionally occurs in whooping-cough or capillary bronchitis, affecting feeble, young children.

The symptoms of congenital atelectasis are usually present from birth. In a large majority of instances the infant is born more or less deeply asphyxiated, respiration is established with difficulty and is notably inefficient, but not always, for occasionally the child, although less vigorous than usual, exhibits no serious lung symptoms for some days or weeks after birth.

The literature of the subject furnishes numerous examples of children who have lived for several weeks with a considerable portion of the lung—even an entire lobe—atelectatic, and so altered in structure as to be incapable of inflation after death.

A noted case is reported by Dr. Ryan (London *Lancet*, vol. i., 1868.) A child, aged five weeks and in good condition, died suddenly. At the coroner's inquest, both lungs were found shrunken, inelastic, non-crepitant on pressure, and presenting in every particular the usual appearances of fetal lung. They sank in water, and when they were cut into many pieces no portion of them floated. The microscope showed an absence of cellular structure. Holt comments on the frequency with which the discovery is made that a child, using less than one-half of its lung tissue, has lived for months without showing marked signs of cyanosis.

The breathing is fast and shallow. The child lies quietly without attempting muscular movements, and his whole demeanor indicates lack of vigor. Most of the time is passed in sleep. The cry is not loud and strong, but is a piteous moan or mere whimper, and at times almost inaudible. The child nurses feebly or not at all. The surface, especially the face and finger tips, become cyanotic and the extremities cold. The temperature is normal or subnormal, and the pulse feeble and rapid. The fontanelle is depressed.

In the unfavorable cases, these symptoms become more pronounced, and muscular twitchings foreshadow the coma or convulsions which so often immediately precede the fatal termination. It is not at all uncommon for still-born children who have been resuscitated with difficulty, perhaps by the prolonged use of artificial respiration, to die suddenly after a feeble existence of a few hours or, at most, a day or two. In many of these cases, even when the breathing has been apparently thoroughly established and the cries fairly strong, the post-mortem examinations have shown that only very limited portions of the lungs had been inflated. The autopsy usually reveals a patulous foramen ovale and sometimes thromboses of the cerebral sinuses.

Acquired atelectasis is always a secondary affection, and the symptomatology is largely influenced by the antecedent disease. As previously stated, it almost invariably occurs as a complication of primary bronchitis, or of one of those specific diseases of which bronchial catarrh is an essential element. When collapse of a considerable area of lung occurs in the course of a pulmonary catarrh, the symptoms at once assume a graver physiognomy. The breathing is more hurried, very shallow, and altered in rhythm; the respirations sometimes number from 70 to 80 in the minute. The child grows more restless, the lips become cyanosed, the extremities cold, and the whole appearance indicates profound depression. The temperature falls below normal. The nares dilate widely with each inspiration. The suprasternal depression, and the deep sulcus around the base of the chest which forms with every inspiration, attest the physical difficulty of getting sufficient air into the lungs. When these symptoms are present, unless the obstruction in the bronchial tubes is promptly removed, permitting the free access of air to the closed vesicles, the child sinks into a state of stupor, and dies asphyxiated or in convulsions. Such severe symptoms are, however, exceptional. In most cases, the collapse involves only scattered lobules, and is indicated by symptoms similar to those just enumerated, but less violent.

The physical signs of atelectasis vary with the extent of the lesion. If several contiguous lobules, or the greater part of a lobe is affected, so as to cause consolidation of a considerable area, the physical signs are pronounced; but when, as happens in a fair proportion of the cases, the collapsed patches are disseminated through both lungs and vary in size from a pea to a filbert, each consisting of one or more lobules separated by a network of normal cells, the physical signs are necessarily negative. However, the very absence of signs in the presence of decided lung symptoms will assist in the diagnosis. For example, if in the progress of a mild bronchitis, without corresponding increase in fever, grave symptoms suddenly arise,—the dyspnœa, lividity, and general distress being greatly aggravated,—and physical interrogation of the chest reveals no solidification of the lungs, the occurrence of lobular collapse offers the only satisfactory explanation of the sudden change.

When present, the physical signs are those of consolidated lung. The sonority of the chest is diminished over the affected spots, but the dulness has a marked tympanitic quality owing to the proximity of normal lung, and especially, as commonly occurs, if emphysematous patches surround the collapsed lobules. The normal breathing sounds are absent, and may be replaced by bronchial respiration and bronchophony. Vocal resonance is increased, and in acquired atelectasis abundant mucous râles are audible over the entire chest. A very important and characteristic feature of atelectasis is the suddenness with which the physical signs are changed. Occasionally, during an examination, dulness and bronchial breathing will be replaced by normal resonance and vesicular murmur; or within a brief period, abnormal sounds may appear and disappear in different portions of the lungs. This can happen in no other pulmonary disease, and depends upon the closing of the bronchi by

plugs of mucus and their speedy removal by forced expiration in coughing, crying, etc.

Diagnosis.—The recognition of congenital atelectasis, if extensive enough to give rise to symptoms, is comparatively easy. But the post-natal form is always associated with other morbid conditions which render the diagnosis difficult and sometimes impossible.

Capillary bronchitis, catarrhal pneumonia, and lobar pneumonia are the only diseases for which collapse is liable to be mistaken. Catarrhal pneumonia is rarely developed except in portions of the lung already collapsed, and hence cannot be differentiated by physical signs alone. Diffuse atelectasis differs from lobar pneumonia in the absence of fever, the percussion note is more tympanitic, bronchial respiration is less marked, and the crepitant râle is absent. The suddenness with which the physical signs are manifested and reach their full development in collapse is an important diagnostic point. If in the course of a bronchial catarrh symptoms of considerable severity suddenly supervene, such as rapid and shallow breathing, duskiness of the face, faint cough and feeble cry, with little or no increase in fever, the nature of the attack can scarcely be doubted. If along with these symptoms the physical evidence of solidified lung is present, the chain of evidence is complete.

The thermometer renders valuable aid in differentiating between the above diseases. Capillary bronchitis is normally attended with only moderate febrile movements, the mercury fluctuating between 101° F. and 103° F. A sudden exacerbation of fever in bronchitis, in which the thermometer registers 104° F. or higher, strongly suggests the onset of catarrhal pneumonia, on the other hand, a sudden fall of the mercury, without corresponding improvement in the symptoms, points strongly to collapse.

Prognosis.—In congenital atelectasis, if restorative measures are adopted early and the lesion is not extensive, the prognosis is good. But if the child be premature or feeble, or if the fetal circulatory openings are unclosed, the outlook is bad in the extreme. The prognosis in acquired atelectasis is always grave; and if the condition occurs in whooping-cough, it is apt to be fatal. Convulsions are of bad omen. Lobular collapse is the initial lesion in many cases of catarrhal pneumonia, of which caseous degeneration and phthisis are not infrequent sequels. Emphysema, more or less extensive, is nearly always left behind, if any considerable tract of lung has been involved in the collapse.

Treatment.—The treatment consists in the adoption of measures and remedies to strengthen the respiratory process, to clear the air passages of all obstructions, and to sustain the strength. Artificial respiration by any of the recognized methods is of great importance in congenital cases. In crying and coughing, deep, full inspirations are instinctively taken, and hence these acts should be frequently provoked. Nothing conduces more strongly to perpetuate atelectasis than to indulge a feeble infant in a vegetative existence. Infants should not be permitted to sleep too long at one time, or to remain any great length of time in the same position. The body heat, often subnormal, should be carefully maintained by swathing the infant in cotton or flannel, and in extreme cases it may be kept for days or weeks in an incubator. In acquired or post-natal atelectasis remedies addressed to the bronchial catarrh, pleurisy, or other associated diseases are indicated and will be discussed in other columns of the Handbook. It is only proper to remark here that those remedies should be chosen which, like the preparations of ammonium, increase the flow of serum and lessen the viscidity of the tough secretion which occludes the bronchioles. Opiates should be sparingly used. If not contraindicated by debility, emetics serve the twofold purpose of expelling viscid phlegm from the bronchial tubes and producing powerful inspirations. Those emetics only are admissible which act promptly and with little depression, as sulphate of copper and ipecac. Alcoholic stimulants are always indicated. Hot immersion baths, made more stimulating by the addition of mustard, and mildly irritating embrocations to the chest are useful. Nutritious diet and tonics, by which the respiratory muscles gain permanent volume and vigor, constitute our chief reliance, as soon as the immediate danger is tided over.

W. J. Conklin.

ATHEROMA. See *Blood-Vessels, Diseases of.*

ATHETOSIS (ἀθετος, without fixed position).—A cerebral affection characterized mainly by continuous, slow, deliberate motion of the fingers and toes, and by inability to retain them in any position in which they may be placed.

This new differentiation being now recognized by eminent pathologists both in English-speaking and continental countries, there is a deluge of reported cases of athetosis, many of which, however, are not strictly in accord with the definition; but the details in regard to the affection are well described, notwithstanding the objections of those who saw in athetosis nothing more than a complex symptom, or a variety of post-hemiplegic chorea.

Symptoms.—The morbid movements of the fingers and toes, symptomatic of athetosis, are involuntary, grotesque, and complex in character, being of a more complicated form than those of simple flexion and extension. The contractions, which do not cease even during sleep, come on slowly with apparent deliberation and with great force. The fingers and toes assume various distorted positions, and carry out movements that would be nearly impossible in the normal state. There is a peculiar distorted position of the thumb and of the index finger, with sprawling abduction of the other fingers, which the hand constantly tends to assume in typical cases, and when once seen can never be mistaken. That which appears to be pathognomonic of athetosis is the localization and the peculiarity of the incessant complex involuntary movements of the smaller and more rapidly acting muscles of the limbs. They seem to prefer the peripheric ends of the extremities, such as the fingers and toes, and rarely the face. A gliding protrusion of the head is occasionally a characteristic of the disease, and in exceptional cases the morbid kinesis has extended to every voluntary muscle of the body. The patient is able to control these movements for a limited time by position and the exercise of an extreme effort of the will; but the disorder is increased by attempts at restraint, by exhaustion, cold, and emotion.

The essential feature of the disease seems to be an incoordination between the flexors and extensors of the muscles of the fingers and toes, in consequence of a lesion in the centre controlling the muscular movements of these members. It has been noted that the phenomena have partly the character of associated movements, for while the fingers moved, the arm was hard and rigid, and during the motion of the toes the muscles of the calf were in a state of tonic contraction. The muscles of the affected extremity are hypertrophied; but it often happens that the hand and foot affected may be atrophied. There is also vaso-motor disturbance in the affected extremity, which may be red, livid, moist, and colder than the corresponding extremity, and pain may also occur in the affected limbs. In some cases, the electric contractility of the muscles is enfeebled or it is increased; in others, it is normal. Relaxation of the ligaments and joints of the affected extremities has also been noted as a characteristic. The ankle clonus is frequently present.

The advent of athetosis is always sudden, and in most cases occurs in young children whose hereditary antecedents are bad, or in those who have suffered from an attack of convulsion and unconsciousness, or, what is more common, hemiplegia, a distinct attack of which in many cases precedes the appearance of the clonic spasm. It is often associated with epilepsy, idiocy, chronic hydrocephalus, and imbecility. Hammond says, however, that of the eight cases occurring in his experience, hemiplegia was not an antecedent condition in four. More recent authority is that athetosis is found in twenty per cent. of all cases of hemiplegia and infantile cerebral palsy. It has