described: "After having seized the vessel by its extremity, I separate it from the surrounding tissue, and grasp it, at its deepest point in the wound, with another forceps, to hold it firmly while it is turned on its axis, three to eight times, by the first pair of forceps." He appears to have employed the method in several amputations. Its supposed advantage was the avoidance of a foreign body in the wound. He recognized the fact that animal ligatures would be equally good for this purpose, and also the disadvantage of torsion in diseased vessels, and that small vessels were not easily isolated. It was, perhaps, for these reasons that, although tried thoroughly by some of the leading surgeons in France and Germany, it fell into disuse until revived by Bryant, in England. The effect of torsion, according to the latter, is a twisting of the elastic fibres of the adventitia beyond the end of the vessel, and a retraction and incurvation of the middle and inner coats; the twist in the outer coat is permanent and cannot be unfolded by any legitimate force; the middle and inner coats are retracted in the direction opposed to the blood stream, approximated and overlapped. They sometimes assume a nipple-shaped projection, at other times a valvular form, being not unlike the semilunar valves of the heart, and closing as perfectly; in some cases, again, they appear to split; in all, the coagulation of the blood is favored. The safety from hemorrhage, according to Bryant, rests upon the twist of the external, the retraction of the internal coats, and the coagulation down to the first branch, while, in acupressure, the permanent safety depends upon the last alone, temporary protection being afforded by the needle.

Kocher found numerous and irregular lacerations of the inner coats over a considerable distance of the wall, and independent of one another, while in ligature the ruptures were circular and only close to the point of ligation. In unlimited torsion there is considerable narrowing of the lumen. Owing to these peculiarities, it has the advantage of favoring a rapid coagulation.

The effect of the limiting forceps in bruising the inner wall of the vessel, and thus favoring both coagulation and repair at the bruised spot, is noticed by Shakespeare, who recommends a similar bruising in ligature (the "modified ligature"). The process of healing is, according to the latter observer, the same in its essentials as that which occurs after ligature.

Acupressure.—The introduction of this method of hæmostasis is to be accredited to Sir James Simpson (unless an obscure passage in John de Vigo's writings be interpreted otherwise than as a description of the ordinary ligature). He saw in the ligature a foreign body in the wound which cut through the two coats at the time of its application, and ate through the outer coat by a process of ulceration, mortification, and gangrene. Variations in the shape or material of the ligature did not overcome these disadvantages. It was for this reason principally that amputation stumps healed with so much greater difficulty than wounds in the operation for vesico-vaginal fistula, although the latter were constantly bathed in leucorrhœal discharges and urine. The application of the ligature isolated a portion of the end of the vessel, which remained in the wound as a piece of dead flesh until it came away with the ligature. The needle, on the other hand, did no injury to the vessel and caused no irritation, its use being based upon "the great pathological law of the tolerance of living tissues for the contact of metallic bodies embedded in their substance." Bryant showed, however, that the ligatured portion did not slough, but became adherent and vascularized. Even though the vitality of such a fragment be completely destroyed, it need not become a slough unless another element, that of decomposition, be introduced, and may, therefore, be surrounded by healthy granulating tissue and become disintegrated and absorbed by a process already described.

Although English surgeons supposed that no injury was done to the vessel by the needle—Hewsen expressly states that no laceration of the internal coat takes place, and Shakespeare does not mention any alteration of the tunics—Kocher and other German writers have demonstrated longitudinal slits in the intima, but not so extensive as those occurring in torsion. The vessel is thrown into longitudinal folds, which become sufficiently firmly glued together to retain this shape long enough, after the removal of the needle, for the thrombus to form and become firmly attached to the walls. A specimen examined by Kocher at the end of twenty-two hours showed no thrombus, the walls being compressed and somewhat thickened, but a fine probe could be introduced between them. At thirty-six hours a well-formed, egg shaped thrombus is represented by Shakespeare. A drawing by Kocher shows a specimen fourteen days old, when the walls have already separated from each other, and the thrombus is short and wide, having a concave surface on the side toward the lumen, and a convex surface at the other end. The relation which the thrombus bears to the vessel is that of a cork to a bottle, beyond the neck of which it does not project. It is probable that the apex had been detached. Hewsen also mentions that the thrombus is confined to the compressed spot. The final cicatrix has a shape similar to that described as following ligature, and is developed by a similar process of repair. In acutorsion, the lumen of the vessel is narrowed for some distance from its extremity.

In acupressure, in the continuity, the proximal and peripheral ends of the thrombus are continuous, as are also the walls of the vessel, which at first are thickened by a connective-tissue growth; the subsequent changes differ in no way from those already described.

Ogston has tested mechanically the comparative strength of arteries secured by ligature, acupressure, and torsion, by subjecting them to the pressure of a column of mercury. It was found that a column one hundred and fourteen inches in height was insufficient to rupture the ligatured artery. Twisted vessels unfolded at an average height of thirteen inches (or a pressure of 6.5 pounds to the square inch). Estimating the internal blood pressure at from two to eight pounds to the square inch, he concludes that it would appear likely that vessels secured by torsion are very liable to secondary hemorrhage, especially when the heart, recovering from the immediate shock of an operation, begins to beat more forcibly. In acupressure, the column of mercury showed an average height of 23.5 inches. It would, therefore, seem a more reliable method, he says, than torsion, and less reliable than ligature. According to Shakespeare, the healing process is slower both in acupressure and torsion than in ligature.

A procedure somewhat similar to acupressure has been described by Pollock; it consists in substituting a silver wire for the ligature, the ends being brought out through the skin covering the lips of the wound and twisted together. They are allowed to remain, on an average, five and a half days, and the results of this procedure have proved satisfactory. *J. Collins Warren.*

ARTHRITIS DEFORMANS.—(Synonyms: Rheumatoid arthritis [Garrod]; chronic rheumatic arthritis [Adams]; nodosity of the joints [Haygarth]; osteoarthritis; French, *rhumatisme noueux*; German, *rheumatische Arthritis.*)

Definition.—A chronic, progressive disease of the joints, affecting chiefly the articular cartilages, bones, and synovial membranes, and producing loss of function and great deformity.

History.—Arthritis deformans is a disease of great antiquity. The chiragra of Horace and other early writers was in all probability deforming arthritis of the fingers. Celsus refers to it, and Aretæus[1] describes the appearance of nodules (πῶροι) on the joints which fairly correspond to those seen in this disease. All doubt that might yet remain as to the existence of the disease in the most ancient times has been removed, however, by the finding of bones bearing the characteristic lesions in the ruins of Pompeii,[2] in Egypt,[3] Pomerania, and elsewhere.

One of the earliest distinctive references to the disease in the older literature is that of Sydenham,[4] who clearly

described the clinical manifestations, but attributed them to rheumatism. Musgrave,[5] Haller,[6] de Sauvages, and others describe more or less definitely one or other form of the disease. Landré Beauvais,[7] in 1800, was the first to investigate its pathological lesions, the destruction of cartilage, and the new formation of bone; but, although he described these as belonging to a definite disease which he designated "Goutte asthénique primitive," it can hardly be claimed that he clearly differentiated them from the morbid changes that result from gout and other affections of the joints.

It remained for Heberden,[8] in 1804, to recognize characteristics which, he suggested, should distinguish the disease from both true gout and true rheumatism. He was at the same time the first to describe the peculiar nodes which sometimes form upon the terminal joints of the fingers and which are still known as Heberden's nodosities. In the following year Haygarth[9] published a monograph on "Nodosities of the Joints," based upon his clinical experience in thirty-three cases, all women, which early convinced him that there is a "painful and troublesome disease of the joints, of a peculiar nature, and clearly distinguishable from all others by symptoms manifestly different from the gout, and from both acute and chronic rheumatism."

The views expressed by Heberden and Haygarth were evidently but partially accepted at the time, for several authors who followed them failed to differentiate this form of arthritis from gout and rheumatism. Comparatively little was written on the subject, however, until the works of Brodie[10] and Adams[11] and the magazine article of Robert Smith were published.[12] These authors accepted the doctrine of the entity of the disease and did much to elucidate its pathological anatomy. To Adams belongs the credit also of having demonstrated that the so-called morbus coxæ senilis of men is pathologically the same affection as the deforming arthritis of the small joints more frequently seen in women. The recognition of the individual nosology of the disease was for a long time checked, in France at least, by the graduation theses of Charcot[13] and Trastour in 1853. They maintained that it was merely a form of chronic rheumatism, and the opinion was accepted by nearly all French writers until quite recently.

The morbid anatomy, first studied, as we have seen, by Landré Beauvais, was further elucidated in the writings of Cruveilhier,[14] Aston Key,[15] Lobstein,[16] Broca,[17] and others. Fuller[18] and Garrod[19] made valuable contributions to the literature of the affection, and so aided materially in establishing both its pathological and its clinical identity. Garrod went so far as to demonstrate that the presence of an excess of uric acid in the blood, a feature of gout, was not to be detected. Rokitansky, Förster, Virchow, and Volkmann should also be named in the list of those who investigated its morbid anatomy.

J. K. Mitchell,[20] in 1831 and 1833, suggested the nervous origin of rheumatic diseases. He was followed many years later by Senator, Ord, and other authorities, until this has become the most generally accepted theory with reference to deformative arthritis. The name arthritis deformans was introduced by Virchow.

Etiology.—The idea that arthritis deformans is in any way related to either rheumatism or gout is no longer tenable. The disease may occur, however, in persons whose family histories show the taint of gout or rheumatism or in those more or less subject to either of these affections.

It may occur at any period of life, but the frequency of its onset is increasingly greater from thirty-five to fifty-five, rapidly declining after the latter period. It is much more frequent in women than in men, commencing in most cases during or after the menopause and somewhat oftener in those who have been sterile. Occurring earlier in life, it sometimes follows rapid child-bearing. Uterine disease was a prominent feature in the etiology of 26 of the 33 cases reported by Ord.[21]

Heredity plays at best a doubtful part in the etiology. As A. E. Garrod[22] intimates, statistics regarding the family history are of little value except in those rare instances in which several generations or their recorded histories have come under the observation of one physician, so great is the confusion in the minds of the laity with reference to gout, rheumatism, and arthritis deformans. His statistics of five hundred cases from private practice, he concludes, "lend countenance to the idea which was formerly very prevalent that there exists an arthritic diathesis a predisposition to arthritic disease, upon which gout, rheumatism, or arthritis deformans is apt to be developed." The daughters of gouty fathers are supposed by some authors to be especially predisposed.

Exposure to cold and wet is believed to have less influence in the induction of arthritis deformans than it has in rheumatism. It nevertheless appears to have had an influence in the development of some cases and undoubtedly may increase the suffering; but temporary exposure is probably of much less consequence than is residence in damp quarters. Bad hygienic surroundings, insufficient or improper food, and exposure are given as important factors in producing the disease in children.

Mental and nervous depression, worry and care are recognized as exciting causes and are capable of producing exacerbations. The exhaustion of nerve centres by sexual indulgence and the leading of a dissolute life have been named by Weber[23] as potent factors in some cases. Gonorrhœa and other diseases of the generative organs have been repeatedly mentioned in this connection, although their occurrence in the history of many cases cannot be regarded as of any real significance. The disease is somewhat more frequent in the poor, especially in those who suffer from malnutrition and anæmia. Hadden[24] attributes it in part to too exclusive use of amylaceous and saccharine food.

Tuberculous history or infection has been frequently referred to, but it must be admitted that tuberculosis usually develops in early life and carries off its victim before he reaches the age of greatest liability to this affection. It is also difficult to estimate the etiological importance of a disease so prevalent. Influenza and other acute infections seem at times to exert an unmistakable influence on the induction of the disease.

Some authors look upon the most chronic form of monarthritis deformans occurring in the hip (morbus coxæ senilis), especially in the very aged, as merely a result of senile change in the joint.

There are two principal theories in regard to the etiology: First, that the disease is of nervous origin, and, second, that it is a chronic infection. The former of these theories, suggested by Mitchell, is well supported by clinical facts, chief among which are: (1) the symmetrical distribution of the joint lesions; (2) the similarity of these lesions to those occurring in locomotor ataxia, syringomyelia, and other affections of the spinal cord; (3) the frequent occurrence, in the course of the disease, of nutritive changes (dystrophies) of the skin, nails, muscles, and bones; and (4) the evident importance of shock, worry, grief, and mental exhaustion in the etiology of some cases.

Unfortunately sufficient post-mortem investigations have not been made to determine the nature of the changes which are supposed to exist in the nervous system. Falli[25] reports two autopsies on typical cases in which he found atrophy of the anterior horns of the spinal cord, and also, in one of the cases, degenerative changes. Neuritis has been demonstrated in several cases, but, as Osler[26] remarks, it is doubtful whether the change is primary or secondary. Ord compares the disproportionate atrophy of muscles to that of progressive muscular atrophy, and infers from their similarity that the disease may be due to lesions in the trophic centres of the cord or to peripheral irritation. Blake[27] attributes the nerve changes to the absorption of various toxic substances from within or from without, and, in the same way, Bouchard[28] attributes Heberden's nodosities to that form of auto-intoxication which is associated with dilatation of the stomach.

The theory of microbic origin has attracted much atten-

tion, but as yet it lacks substantiation. The following facts lend support to it: 1. Several investigators, among them Schüller,[29] Bannatyne,[30] and von Dungern and Schneider,[31] have found micro-organisms in the fluid or tissues of the joint. 2. The disease sometimes begins with an acute onset corresponding in clinical aspect to that of an acute infection. 3. It frequently follows more or less closely upon an infection, as gonorrhœa, puerperal sepsis, influenza, or an acute exanthem. 4. Enlargement of the spleen and lymph glands has been noted in some of the cases reported as occurring in children. Schüller does not, however, look upon the bacteria in the joints as being more than indirectly the cause of the disease, possibly in some manner preparing the joint for the deposit of lime salts.

Morbid Anatomy.—The pathological process involves primarily the articular cartilages, the synovial membranes, and the bones. The disease usually advances symmetrically, so that the lesions in one articulation correspond closely to those in the corresponding joint of the opposite extremity. Later in its course changes occur also in the capsular and other ligaments, in the periosteum, and in the muscles; and to complete the picture we must include the lesions in the nervous system to which reference has been made.

In the articular cartilages the process begins in the centre, the area farthest removed from blood supply and most subjected to friction. It is described by Cornil and Ranvier[32] as a multiplication of cells throughout the entire thickness of the cartilage and the formation around them of capsules resembling cartilage cells, but incapable of deep staining with iodine. Similar secondary capsules develop within these. The cartilage then becomes fibrillated upon the surface by the rupture of the most superficial capsules into the joint cavity and by the formation of parallel tubules from the rupture of the deeper-lying capsules one into another. The degenerated cartilage is finally removed by friction, aided, no doubt, by mucoid degeneration (Rindfleisch) and absorption, exposing the underlying bone over a gradually increasing area. Around this a process of new formation takes place not unlike that so often seen in caries. Cell proliferation continues, forming nodular masses which ossify and constitute the chief element in the production of deformity and the limitation of motion. Sometimes, instead of the nodular growth, a more or less complete bony ring is formed, producing the condition known as "lipping." As a result of either process the end of the bone often appears to be much enlarged. In the spinal column the overhanging edges frequently blend and firmly weld the vertebræ together (spondylitis deformans).

The exposed surfaces of the bone undergo hardening (osteosclerosis), and through friction acquire an ivory-like polish. The minute orifices of Haversian canals may be seen in the polished surfaces. While this eburnation is taking place the underlying spongy portion of the bone is undergoing a rarefying ostitis resulting in an osteoporosis. As a result of the latter process, when it reaches the surface, the articular face is often grooved and deformed, even in parts not yet denuded of cartilage. The heads of the humerus and femur have been almost entirely removed in this manner. Warren[23] aptly says that as a result of these various changes, "the head of the bone appears as if it had at one time been composed of a substance capable of softening from heat, and in that condition had been held carelessly while it was allowed to cool." Billroth compares it to "a fluid which had been poured out and stiffened while flowing." These comparisons are especially applicable to the knee and elbow joints. The shafts of the long bones in some cases show enlargement and increased density. The neck of the femur may become bent at a more acute angle to the shaft. Bony ankylosis occurs only in the vertebral column, and there is no tendency to the formation of fibrous adhesions as in other joint affections.

The synovial membranes become highly vascular; their blood-vessels push upward into the cartilages and doubtless assist in their destruction. Some authors look upon this hyperæmia of the synovial membranes as the first step in the morbid process. In the polyarticular type of the disease there is often a pannus-like growth of the membrane over the cartilage (Warren). The membranes are thickened, and their fringes elongated. Their cells proliferate and cartilaginous nodules are formed. These often become freely movable, although still attached by a fibrous pedicle. They are sometimes detached and lie loose in the joint cavity. Adams found no fewer than forty-five of these "foreign bodies" in one elbow joint; as many as four hundred have since been noted in a single articulation. They are usually small and round, but sometimes attain the size and shape of chestnuts. They rarely ossify and probably sometimes unite, having the appearance of being glued together. Fatty tissue also is frequently found in the folds and fringes of the synovial membranes. The synovial fluid, often increased in the beginning of the disease, is generally diminished in the later stages, and the joint becomes abnormally "dry." Mucin has been found in excess in the synovia; sodium urate is never present. Schüller has repeatedly found lime salts, especially the crystalline oxalate, in the cartilages and synovial membranes. Suppuration occurs only as a result of operative interference or exploratory puncture.

The bursæ in the vicinity of the affected joints are often distended by effusion, sometimes forming cysts. These were first described by Morrant Baker.[34] They form a bulging which imparts a doughy sensation on palpation at the sides of the articulation or elsewhere. The fluid is at first confined to the joint, but later, according to Baker, with the increase of tension, it escapes through channels of normal communication into the bursæ; or it may form a hernia of the synovial membrane. If the tension within the sac becomes too great, it ultimately escapes into a cavity bounded by the muscles and other tissues.

The muscles appear wasted and of a brownish color. Spender[35] says that the phenomena of muscular atrophy are myelopathic and capable of definition as a strict spinal paralysis. They at least correspond to the type which is met with as a result of nervous lesions, some bundles showing greater degenerative changes than others. Some of them in fact are entirely replaced by connective tissue. "The muscles perish, fibre by fibre."

The ligaments, periosteum, and tendons not infrequently undergo marked thickening in proximity to the diseased joints.

Symptoms.—Following the classification of Charcot, the symptomatology is to be considered under three heads corresponding to a like number of types of the disease. Clinically these varieties have comparatively little in common. They are the multiple or progressive form, the monarticular form, and Heberden's nodosities.

1. The multiple, progressive type may be again subdivided into an acute and a chronic form, to which some add another (subacute) form, differing only in degree.

The acute form occurs more frequently than do any of the others in subjects under forty. It is readily mistaken for subacute rheumatism, but it is rarely so severe as to suggest the acute type of that disease. There is ordinarily enlargement of the joints from the beginning, yet the suffering is not so severe as to confine the patient to bed. When the onset is very acute, however, the pain may be beyond all comparison to the swelling. The skin is not usually reddened. The disease is symmetrical in its manifestations or soon becomes so; the small joints, as those of the fingers and toes, are oftenest affected. It does not migrate but continues to affect the articulations first involved, while others, often larger joints, become similarly affected. Its progress is generally in a centripetal direction. Elevation of temperature rarely exceeds 102° F. Headache, malaise, and anorexia are now and then complained of, and the patient nearly always appears anæmic. There is no tendency toward involvement of the heart. After a time the acute symptoms subside only to recur again at uncertain intervals and

with variable force, often apparently as a result of exposure, injury, or an indiscretion in diet.

The chronic form of the multiple arthritis deformans is much more frequent than the acute and it is more insidious in its onset. It frequently begins in a single joint of a finger or toe, the corresponding articulation of the opposite member soon becoming affected. The middle finger is not seldom the first to be attacked. The disease then extends in a centripetal direction, ultimately involving nearly every joint in the body, including the temporo-maxillary, the vertebral, and occasionally the sterno-clavicular. The carpo-phalangeal articulations of the thumbs often escape.

The patient first notices a stiffness of the joints, especially in the morning. They are painful and are tender on pressure. As in the acute type, the progress is not constant, intervals of apparent rest sometimes extending over weeks, months, and even years. Each exacerbation renders the deformity a little more pronounced and the joint movement a little more restricted. Pain, seldom much noticed during the intervals, is not always severe in the acute stages, but it may be distressing. It is usually less severe in advanced cases. In addition to the joint pains the patient sometimes suffers from others of a neuralgic character, particularly in the ball of the thumb or the inner side of the wrist, and from bone pains. Neuralgic pains are supposed to be most severe and most persistent when due to trophic degeneration of nerve roots. Fever occurs only in the more acute exacerbations. The pulse is accelerated; its tension is variable, but it is as a rule lower than in gout or rheumatism. Numbness and tingling of the hands and feet are in some instances early symptoms, even preceding other manifestations, as noted by Homolle[36] and Howard.[37] Free sweating is less frequent than in rheumatism, but Anders[38] has observed it in advanced cases, and Spender regards localized perspiration, especially of the fingers, an early symptom. As a result of trophic changes the skin becomes pale, dry, smooth, and glossy, sometimes irregularly pigmented (freckled) over the affected joints. Spender notes cutaneous pigmentation on various other parts of the body. Onychia may occur: Anders has seen three cases. Bedsores occur only very rarely.

Muscular atrophy is a prominent feature of all advanced cases; it is more profound than that which results merely from disuse. It affects both sets of muscles, but the extensors to a higher degree than the flexors. The joints are usually flexed unless this is prevented by the encroachment of the osteophytic formations. Gowers[39] calls attention to increased myotatic irritability with normal or lowered electrical irritability as a common symptom. He therefore attributes the atrophy to changes in the terminations of the pyramidal fibres in the gray matter. Sudden spasmodic or tonic, painful contractions of the muscles occur in some cases and for the time greatly aggravate the joint pains. Such contractions are exceedingly distressing to the patient when they occur in the muscles of the jaw in connection with arthritis of the temporo-maxillary articulation, often seriously interfering with the taking of food. The reflexes over the affected joint may be increased or diminished, or they may remain normal so far as they can be tested.

Crepitus of a peculiar character, cracking, creaking, or grating, is one of the earliest symptoms. In advanced cases, a sound is sometimes produced by movement of the affected joints which may be heard at some distance.

Chronic laryngitis is met with in some cases and is regarded by some writers as due to disease of the cartilages of the larynx similar to that in the joints.

Deafness has been known to result from involvement of the small bones of the ear.

Asthma occurring in connection with the disease, as noted by several authors, can hardly be regarded as more than an accidental complication.

2. The monarticular type. There is much ambiguity in the use of the word monarticular in this connection, since it is applied to a type of the disease which does not always confine itself to a single joint. It is in many cases only the predominance of the manifestations on the part of one of the larger articulations that justifies the use of the term. The points of difference between this and the multiple type are very striking. It occurs more frequently in men than in women, notably in those over fifty years of age. It attacks by preference the larger articulations, especially the knee, shoulder, elbow, and hip. It confines itself as a rule to the articulation first affected, but the opposite joint sometimes becomes involved to a less degree. The vertebral column is often attacked (spondylitis deformans), few or many articulations, rarely a single one, being involved, and this occurs alone or along with involvement of other joints, especially the shoulder or hip. The entire column becomes rigid in extreme cases. When the shoulder or hip is affected, the shortening of the limb keeps pace with the absorption of articular cartilage and bone. In hip-joint cases the shortening may be increased by the bending of the femoral neck, still more by the dislocation of the head of the bone, favored by the extensive absorption of the articulating surfaces and aided by muscular contraction. The affected joints not infrequently have the appearance of being subluxated or dislocated, owing to the great deformity, when in reality there is little or no displacement. When fluid accumulates in the joint, in the bursæ, or in adventitious cysts, the deformity is greatly augmented. Movement of the affected joint is attended with the characteristic creaking crepitus. Pain is a prominent symptom, occurring more or less periodically and aggravated by motion. Associated with hip-joint involvement, it not infrequently simulates sciatica in its extension down the thigh and leg.

The buttock and thigh are shrunken from muscular atrophy, and, from the same cause, the appearance of the shoulder resembles that of progressive muscular atrophy. The loss of motion is almost complete; bony anchylosis does not take place, however, except between the vertebræ. The knee jerk is exaggerated on the affected side. The patient is unable to cross the diseased leg over the sound one without assisting the movement with the hands (Garrod).

A. E. Garrod refers to a peculiar type of monarthritis deformans which is limited to the carpo-phalangeal articulations of the thumbs, joints which are most frequently immune to the polyarticular form of the disease. It is generally attributable to undue exercise of these joints incident to the occupation of the individual, and may be readily recognized by the enlargement and the characteristic crepitus.

Heberden's nodes are sometimes associated with this type of the disease.

3. Heberden's nodosities are small, immovable exostoses, "little hard knobs," which develop on either side of the distal joints of the fingers, arising from the small tubercles at either side of the dorsal surface of the second phalanx. In the joint proper the characteristic destruction of cartilage and eburnation occur. The disease is more frequent in women than in men: it comes on, as a rule, between the thirtieth and the fortieth years of age. The nodosities usually constitute its only manifestation in the case, unless it be associated with the monarticular form. They at times develop in gouty subjects, but are entirely independent of that disease and distinct from tophi. Sodium urate is not found in the joint fluid. The joints are often swollen, slightly reddened, and sensitive to pressure in the early stages of the disease, but later they occasion little inconvenience aside from stiffness and deformity. Heberden described the nodes as of the size of a pea; they may, however, exceed that size. In some cases the enlargement is fusiform, a doughy mass forming around the joints. The disease follows the same intermittent course that characterizes the multiple type, acute attacks being induced by injury, or apparently by errors in diet. The finger tips may be deflected toward the radial side as in the ordinary type of the disease. The thumbs generally escape. Charcot calls at-