Staff Meeting Bulletin
Hospitals of the . . .
University of Minnesota

Gout

Volume VIII | Thursday, December 3, 1936 | Number 9

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
UNIVERSITY OF MINNESOTA

Volume VIII | Thursday, December 3, 1936 | Number 9

INDEX

Published for the General Staff Meeting each week during the school year, October to May, inclusive.

Financed by the Citizens Aid Society

William A. O'Brien, M.D.

I. LAST WEEK

Date: November 19, 1936

Place: Recreation Room
Nurses' Hall

Time: 12:15 to 1:25 P.M.

Program: Movie: Master Will Shakespeare

Abstract: Pericarditis

Case Report: Probable Adhesive Pericarditis

Present: 128

Discussion: R. E. Mattison
R. W. Koucky
L. G. Rigler
C. J. Watson
O. H. Wangensteen
R. M. Johnson

Gertrude Gunn
Record Librarian

* * * * * *

II. OUR GUEST TODAY

PHILIP S. HENCH, Mayo Foundation

* * * *

III. MOVIE

Title: "Behind the Shadows"

Released by: National Tuberculosis Association

* * * * * *

IV. ABSTRACT

GOUT

I. J. Pass

Definition

Gout is "a disease occurring in paroxysms and marked by a painful inflammation of the fibrous and ligamentous parts of the joints, deposits of urate of sodium in and about the joints, and an excessive amount of uric acid in the blood."

History

Gout is one of the oldest described diseases. It is one of the humorous diseases in medicine, the subject of much satire and ridicule. Kings and tyrants have boasted of having it and others have suffered the tortures of the damned with it. In the arts, gout, although a most painful and troublesome affliction, is rarely depicted as a tragic affair, but as a source of satirical and caricaturistic wit.

Hippocrates (460-370 B.C.) is given credit for being the first to describe the disease, but that was in man. "Podagra," as gout was then called, was a term used before 500 B.C. to designate an affliction of the joints of beasts of burden. Before Hippocrates had used the term podagra for gout in man, there was an affliction of the joints commonly seen in hunters and later considered to be same as gout. It was known as the "Disease of Diana" after Diana the Goddess of the Hunt. In Laconia a temple was dedicated to Artemis Podagra and here the people afflicted with the disease prayed for relief. Another mention before Hippocrates' time is made by Hieron, a tyrant ruler of Syracuse from 478 to 467 B.C., who mentioned bladder stones in individuals suffering from gout.

The term "podagra" is derived from the Greek "agra" meaning a seizure and "pous" (pod) meaning foot. Although gout and rheumatism were confused even after Sydenham's classical description, the earliest Greek writers were far more accurate than those of the Renaissance, as they confined the term "podagra" to joint afflictions of the foot alone. For seizures in other joints of the body, they used the appropriate prefix--as " "chiragra" for the hand, "pechyagra" for the elbow, "gonagra" the knee, "dentagra" the teeth, etc. The term gout was not used until the thirteenth century. It was derived from the Latin word "gutta" (drop), from the drop of evil humor distilled into the joints. James Russell Lowell, who was severely afflicted with gout, in his humorous way did not agree with the Latin origin of the word. The "disease," he says, "derives its name from the patient's inability to go out. The ordinary derivation from gutta is absurd, for not only is the German form gicht deduced from gehen but the persons incident to the malady are precisely those who themselves or their ancestors for them, have kept just this side of the gutter."

Hippocrates not only described gout as an entity but recognized that eating and drinking had something to do with its cause, that there was a rich man's and a poor man's gout, that eunochs never had the disease, and that women seldom had it before the menopause. Galen (131-200 A.D.) spoke of the hereditary nature of the disease. He added that venery was probably a cause of gout, and as the effects of wine were already appreciated, there arose the aphorism that "podagra is the daughter of Bacchus and Venus." Celsus (first century A.D.) and Aretaeus, the Cappadocian (about 200 A.D.) gave good descriptions of gout. Aretaeus recognized the intermittent nature of the disease, as is shown by his story of a man, severely afflicted with podagra, who won an Olympic race during a remission.

The modern history of gout begins with Sydenham, who distinguished gout from other arthritic afflictions. Wollaston analyzed the tophaceous deposits and found them to consist chiefly of sodium urate. There followed the classical clinical descriptions of Heberden and Scudamore. The chemical history of gout, as disorder of metabolism, dates from Alfred Garrod's work, especially from his discovery of uric acid in the blood.

No disease has such a predilection for eminent men. Among medical men, to name only a few, are Sydenham, Harvey, Linne and Widal. Amongst the men in pure sciences are Leibnitz and Newton, and among the historical figures are found such men as Alexander the Great, Louis XIV of France, the two Pitts, and Benjamin Franklin. Charcot said "the distinguished statesmen become victims of gout."

"The geography of gout is that of the habits of mankind." It is a disease of temperate climates.

Incidence

Gout was evidently much more frequent formerly than at present. In Germany, France, America and even England, the home of gout, the number of those afflicted with the typical disease is constantly decreasing. That its incidence was decreasing was observed as early as 1881 by Charcot. Relative to the frequency of latent gout, there are, however, widely divergent views. There are some who frequently make a diagnosis of gout. On the other hand, there are others who go so far as to deny the very existence of the disease. This wide discrepancy is undoubtedly due in part to the differences in criteria for the diagnosis of gout, in part to the actual variation in the frequency of the disease in different strata of society. Thus, Herrick and Tyson saw 6 cases of acute gout in one year of office practice, whereas only 5 cases were seen in a similar period in large arthritic clinic of the Presbyterian Hospital of New York City. There seems, moreover, to be as wide fluctuation in the incidence of gout reported from the same hospital for different decades. Williamson found 116 cases of gout (0.399% of admissions) for a 6 year period (1914-

1920) in Cook County Hospital, Chicago. Volini and O'Brien could find only 18 cases admitted to the same hospital during the past 5 years (1930-1935). On the other hand, at the Philadelphia General Hospital, only 47 cases of gout were recognized in approximately 414,296 admissions from 1905 to 1929, whereas in 146,992 admissions from 1929 to 1935 there were 30 cases.

Hench reports that at the Mayo Clinic the number of cases recognized has shown an unusual increase: from no cases in 1915 to 4 to 8 cases for each of the next 6 years, to 10 to 16 cases from 1922 to 1925, to 75 cases in 1925, and over 100 cases in 1930. In 1931 he stated that for the preceding summer, 5% of a consecutive series of rheumatic diseases had gout.

In the University of Minnesota Hospitals Out-patient Clinic, among approximately 3,000 cases of arthritis, there have been perhaps half a dozen cases of gout (Wetherby).

Of cases of gout coming to necropsy, Gudzent and Holzmann found 77 in 32,089 examinations made at the Charite' in Berlin; Futcher, 59 in 18,000 autopsies at Johns Hopkins Hospital.

Sex

Gout is much more common in men than in women. Lichtwitz gives as the ratio 7 males to 1 female. In Williamson's series of 116 cases, there was only 1 woman; among Cohen's 47 cases, there were but 3 females. After the menopause, women seem to become far more liable to the disease.

Age

The average age at the onset of gout is between 40 and 50 years. The following table shows the onset by decades for two series of cases:

	Williamson 1920 116 cases	Cohen 1936 63 cases
0 - 10 years	0%	0%
10 - 20 "	0	6
20 - 30 "	15	19
30 - 40 "	37	37
40 - 50 "	29	27
50 - 60 "	18	9
Over 60 "	1	2

The impression that the usual age of onset is between 50 and 60 years is probably erroneous. It may probably be attributed to the average delay of 15 years in the diagnosis of gout.

Young people usually escape gout; it is rare before puberty. However, there are notable exceptions reported. Allbutt feels that these are more frequent than is supposed. Several observers have declared that the younger of large families are more liable to gout than the elder.

At the other end of life, first attacks of gout have been reported in the very aged: Garrod, in a patient of 70; Ebstein, in one of 80.

Seasonal incidence

There are definite seasonal exacerbations of gout. Alexander of Tralles (6th century A.D.) speaks of cold as promoting gout. Allbutt noted the more common occurrence of gout in the spring and autumn. He says "one may suppose men to be more convivial and to take on the whole less exercise in mid-winter." Williamson's series may be considered fairly representative. The frequency of attacks in his 116 cases was as follows:

Spring	40%
Winter	22
Fall	20
Summer	18

Heredity

Most older authors speak of the strong hereditary transmission of gout. Among these are Aretaeus (second century A.D.) and Alexander of Tralles (sixth century A.D.). Some claim that a hereditary history is to be obtained in every case. Scudamore obtained a definite hereditary history in 331 cases out of 522. A perusal of the works of Heberden, Garrod, Roberts, Duckworth, Ebstein and Brugsch leads one to believe that a definite gouty history may be obtained in about three-fourths of all cases. This view is at some variance with the experience of American investigators. Williamson found only a questionable history in 12 of 116 cases; Cohen in 8 of 44 cases. Hench comments that his cases, like those of Futcher, "seem to have earned rather than inherited their gout."

The discrepancy may lie in the very liberal definition of gout by some authors. For example, Lichtwitz says that "when investigating the constitutional and hereditary factors the inquiry should not be limited to gout. In the family, as well as in the personal histories of the gouty, very frequently are discovered migraine and various manifestations of angiospastic diathesis; also asthma, spasmodic vomiting, urticaria, acute articular rheumatism, and arthritis without urate deposits." Furthermore, early workers called many other conditions: angina pectoris, arteriosclerosis, uremia, etc. gout (irregular and retrocedent gout).

Lead

Lead has been accused of producing gout in some cases. Although Skagge is said to have observed gout in lead workers in 1764, and Garrod in 1857 pointed out that it was quite common among lead workers in England, gout has not been considered a usual complication of lead poisoning. Hubner states that it is really not an uncommon symptom in older workmen and that it may even be seen in young men. His description is that of typical gout. Allbutt describes several cases which he ascribes to the lead from the lead pipe used to convey a private water supply, and one due to lead in the patient's snuff.

In Williamson's series, only 9.4% gave a history of contact with lead; only 3 of 47 cases in Cohen's series had been exposed to lead. In none of them, however, was there outspoken plumbism. Aub remarks that although lead intoxication and a gouty diathesis may be related, he has never observed the association.

Eating and drinking

That excess in eating and drinking is important in the causation of gout has been recognized since earliest times. Allbutt comments that "perhaps there is no more deeply rooted empirical axiom in all medical etiology." From Roman history, we learn that during the severity of life during the republic, gout was almost unknown; but that under the Empire, as luxury and indulgence increased, gout became one of the most frequent of the maladies of the upper classes.

"Whether or no excess of rich animal foods alone, without wines or beers, can produce gout is uncertain; perhaps hereafter a dry America will let us know" (Allbutt). Garrod said that "were alcoholic drinks unknown, there would be no gout."

Williamson notes that of his cases, 92 of 101 were well nourished. Of 116 cases, 4 were teetotalers, 41 drank moderately, the rest heavily, many to excess. Sixty-two per cent of Monroe's cases admitted habitual use of spirits. Hench remarks that the new liquor laws already have augmented the series of cases at the Mayo Clinic.

Trauma

It has long been supposed that the site of an outbreak of gout is often determined by a strain or injury. Garrod and others have described the great toe as an area of stress. Allbutt suggests that the attacks on the great toe are in part because of its cold and distant place. A blow or a sprain may predis-

pose to gouty involvement. Hench records gouty attacks precipitated by operative procedures. Cohen noted acute attacks following tonsillectomy and acute appendicitis.

Diagnosis

The classical picture of gout is perhaps well known: it has been well described by Sydenham (from personal experience), Heberden, Allbutt and others. The following is from Allbutt:

"We passed down to a bed on which a man was lying with a cradle over his legs. Unwelcome as we were to the timorous patient, we lifted the coverings from the cradle and displayed two great toes; the right blazing with acute gout, the left also smitten but not so severely. He prayed us not to touch even the bedstead. He was a big florid man, rather fat, and by occupation a college butler."

Such cases are increasingly rare. But is gout correspondingly rare? The answer depends largely upon the rigidity of the criteria applied. Some will accept cases as gout only if the patients have (1) increased blood uric acid, (2) tophi, or (3) so-called characteristic punched-out areas in the roentgenogram, and only if they give a history of attacks of acute arthritis, preferably in the great toes.

Hench, Vanzant, and Nomland reviewed in 1928, 100 cases of proved gout. They found that the so-called proofs of gout were uncertain in their presence or in their time of appearance. In 28%, the blood uric acid was not elevated. In one case, this was true even though many tophi were present and the gout had begun 30 years previously. Tophi were not found in 66% of the cases. In only 22 cases were the roentgenograms of themselves characteristic of gout. A third of the patients had not had involvement of the great toes. Many of the patients claimed that they had never touched any form of alcohol. From their study, they felt that the history of repeated attacks of acute arthritis with complete remission gives the earliest clue to the diagnosis of gout.

Diagnostic criteria of gout

1. Joint involvement

Classically, gout is supposed to attack the great toe. This is often the case. Scudamore found that of 516 cases, the great toe of one foot only was attacked in the first seizure in 314; in 27, both great toes. In Williamson's series, 88% involved the great toe; the right four times as commonly as the left. Hench, Vanzant and Nomland found similar classical involvement in 38 of 100 cases. However, attacks may commonly involve other joints, and may even be polyarticular from the start. Scudamore reports first attacks involving insteps, ankles, knees, heels and hands. A common experience is that of Hench who describes a man who suffered numerous attacks of gout involving the great toe, the ankles and the region of the Achilles tendon. "When a toe was attacked, a diagnosis of sprain, infectious arthritis or neuritis was made, and treatment consisted of strapping or of the removal of foci of infection."

Several investigators, notably Williamson, Lambert and Hench, have noted that the great toe when affected by gout is most tender medially rather than underneath as is the case in infectious arthritis.

2. Tophi

The tophus is the most certain of the diagnostic criteria of gout. These may appear before the first attack, or after 15 or more years of repeated attacks. They may develop painlessly and are often unnoticed. In its first stage, the tophus is usually soft and fluid in its consistency. Later, it fills with crystalline sodium urate. The formation and disappearance of tophi is independent of the occurrence of attacks. Tophaceous deposits begin in the synovial membranes, cartilages, ligaments and adjacent tissues. They may also appear in the helix of the ear, in various bursae, particularly that over the olecranon, subcutaneous tissue and less commonly in the eyelids, larynx, bronchi, auditory ossicles, aorta,

corpora cavernosa, etc. Finally, in the words of Sydenham, "the whole body is converted into a tophus." Tophi may become so large that they produce severe deformity of joints. Rarely the tophi may become so prominent as to ulcerate the skin. "We used to hear of gouty forefathers who had the advantage of being able with their chalkstones to mark up their points at whist."

The occurrence of tophi varies greatly in various series reported. Williamson found tophi in 66 of 116 cases (56%); Hench, Vanzant and Nomland, 44 of 100 cases (44%); Schnitker and Richter, 39 of 55 cases (71%).

3. Blood uric acid

Since the time of Garrod, the view that uric acid is a poison and is the toxin of gout has been firmly rooted in the minds of most physicians. In gout, uric acid is retained in the body. Garrod attributed this retention to kidney damage. It is true that with renal insufficiency there is commonly retention of uric acid, also blood uric acid is often elevated in leukemia and polycythemia, but these conditions are rarely associated with gout. There is no recognizable relationship between hyperuricemia and the gouty attack. Hench found several patients with tophi and even specific roentgenograms for gout who did not have an increase of uric acid in the blood. (Normal blood uric acid, 2 to 4 mgms. %.)

Folin, Derick and Berglund studied the relationship between uric acid and gout. They felt that the blood uric acid was quite constantly elevated in gout; however, they could not relate the blood uric acid levels to the attacks of gout. They found that the acute attack of gout is not accompanied by any change in the usual uric acid level in the patient's blood. Intravenous injection of uric acid does not bring about an attack; moreover, when during the height of an attack, uric acid is injected intravenously, causing considerable increase of the uric acid content in the joint fluid of the acutely inflamed and swollen joints, the symptoms are not aggravated.

The blood is capable of holding in suspension far more uric acid than has ever been met with in gout, so there must be some unascertained factor which promotes its deposition in the gouty state. While hyperuricemia is not restricted to gout, the deposition of urates in the tissues in and around the joints is entirely confined to this disease. In the absence of tophi, a diagnosis of gout is only presumptive. It therefore appears that the fixation of uric acid in the tissues and its conversion into sodium urate has a much greater etiological significance in the problem of gout than the mere presence in the blood of uric acid in amounts that vary relatively little from the normal.

The purine bodies, adenin, guanin, hypoxanthin, xanthin and uric acid, result from the transformation of the nucleoproteins of the food and tissues. Foods, rich in nucleo-proteins, may be avoided, unfortunately not with uniformly salutary results. Experimentally, the purines of the diet appear to be of great etiological significance in gout. Schlotthauer and Bollman have shown that turkeys in whom blood uric acid values of 15 mgm. % was obtained on diets high in purines developed definite gouty tophi on their feet. Perhaps the condition in birds is not entirely comparable to gout in man.

Krafka has suggested that since the chief endogenous source of uric acid is the extruded nuclei of normoblasts in the maturation of red blood cells, any factor which causes stimulation of erythropoiesis becomes thus a potential etiological agent in gout. He points out that gout has frequently been found associated with pernicious anemia and with polycythemia. The disappearance of old-fashioned gout he would attribute to the discontinuance of the formerly common medical practice of bleeding.

4. X-ray findings

The presence of punched-out areas of rarefaction in the articular

ends of bones is considered as strong evidence of gout. Unfortunately, these may appear in other conditions such as infectious arthritis. Moreover, verified cases of gout may show entirely non-specific joint changes. In Williamson's series, 14 of 42 cases had characteristic findings. Of 28 cases without characteristic x-ray findings, 21 had tophi!

5. Cutaneous manifestation

Some attach great importance to the manifestations of gout in the skin. The commonest sign described is a dry eczema. Hyperkeratosis occurs around the nails of both the hands and feet, as well as of the elbows and knees. These look as if the skin were powdered with grey cigar ash. Cmunt has described eruptions characteristic of gout. Painful edema along the tibia is said to occur frequently.

6. Diagnostic aids

There are several other criteria which may be considered as evidence of gout. Delicati of Bath drew attention to the presence of ground down teeth in a not infrequently almost perfect set. He also mentions ulnar deflection of the terminal joint of the index finger (Coates' sign).

Recently Lockie and Hubbard have made a contribution to the diagnosis of gout which may prove of great value. They found that on diets high in fat and low in carbohydrate patients with gout developed attacks of gout within a few days. On this diet, the hyperuricemia increased and the amount of uric acid excreted in the urine decreased. The variations in the clinical symptoms were not directly dependent on changes in the uric acid concentration of the blood. Attacks were noted before the increase in the blood uric acid occurred in some instances and disappeared while the hyperuricemia was still present in others.

The feeding of high purine diets has proved of very little value in the diagnosis of questionable cases of gout.

Classification

Acute Gout

The onset of acute gout is usually sudden. There may be a prodromal period during which dyspnea, capricious appetite, pyrosis, constipation and attacks of sudden diarrhea, mostly at night, occur. The tongue is heavily coated and there is a disagreeable taste in the mouth. There may be a feeling of heat and numbness in the muscles with muscular twitching. Pain usually begins during the night, commonly in the big toe. This may be accompanied by chills and fever. The temperature may go to 102 or 103°F. The pain grows worse or the chilliness subsides. It may be of vice-like or of the gnawing type. The affected part becomes exquisitely tender. This lasts about 24 hours and as the pain subsides swelling and redness appear. The opposite limb or many joints may be affected simultaneously. There is usually a polymorphonuclear leucocytosis during the acute manifestations. The sedimentation rate is increased during the acute attack. With the subsidence of the swelling the skin desquamates.

Chronic Gout

Chronic gout may be said to be present when tophi are present and when joint deformities appear. The separation of acute and chronic gout is difficult. Indeed, Lichtwitz feels that gout is always a chronic disease. Acute gout, he says, is simply the stage of the disease characterized by explosive attacks.

Abarticular or irregular gout

The older physicians considered that "gout is protean in its activities and no part of the body is immune from attack by the gouty poison." Many disorders in gouty persons were accordingly considered abarticular or irregular gout. These included bronchitis, eczema, migraine, conjunctivitis, nephrolithiasis and others. Rolleston warned that abarticular gout is a danger-

ous subject because of the tendency to regard almost any manifestation in gouty persons as gout. There can be little doubt of the existence of abarticular gout since tophi have been demonstrated in the bronchi, in the larynx, in the heart, etc. However, it has undoubtedly been much overemphasized in the older literature.

Retrocedent gout

Under certain circumstances, gout is supposed to fly from the extremities to some internal viscus, such as the heart, stomach or brain with serious, even fatal, results. If the brain be the seat of its descent, then delirium, coma or convulsions may appear; if the heart, violent palpitation, pain, irregularity of rhythm, dyspnea, perhaps syncope and death; if the stomach, violent vomiting, and so forth; The treatment is "to recall the gout" to its former quarters in the foot by means of hot mustard baths, a bottle of port and so on. This phenomenon probably belongs to what Allbutt has called the mythopoietic period of medicine." Its total disappearance suggests that such conditions as uremia, angina pectoris and apoplexy were called gout.

Allergy in gout

Of recent years, some have accorded gout a place in the great family of allergic diseases. The literature contains many examples of gouty attacks occurring after certain foods. It has been known for a long time that a specific hypersensitiveness may exist for a certain alcoholic beverage: one person may have an idiosyncrasy to beer, another to champagne. Widal, who was gouty himself, was the first to postulate the allergic nature of gout. In himself, he noted a hypersensitiveness to a particular wine and came to the conclusion that the allergen was a protein from the yeast. Llewellyn cites examples of gouty attacks provoked by hives, asthma, typhoid inoculations, and even gnat bites. He says that an eosinophilia not infrequently accompanies the attacks. Lichtwitz has emphasized the frequent findings in the family as well as in the personal history of the gouty, migraine, asthma and urticaria. Recamier said, "Gout and migraine are sisters."

Nephritis in gout

Aretaeus, the Cappadocian, a contemporary of Galen, is given credit for first describing a scanty, deep yellow, cloudy urine during attacks of gout. Thus, early in the history of the disease, attention was directed to the kidneys. Nephritis has been regarded as a common complication in gout. Unfortunately, most of the early references are not only valueless but actually misleading. The diagnosis of nephritis from albuminuria alone is, to say the least, fraught with pitfalls.

Rarely urate deposits in the kidneys may be large enough to produce renal impairment. Uric acid infarcts in the form of whitish streaks in the medulla of the kidney have been described. The usual renal complication of gout is, however, as has been pointed out by Schnitker and Richter, vascular in origin. These authors found vascular disease and hypertension to be more frequent in patients with gout than in non-gouty individuals of the same age. Of 55 cases of gout, 17 (31%) had clinical nephritis. Of these, 5 died in uremia. Four were examined pathologically: 3 had predominantly vascular nephritis (arterio- and arteriolosclerosis), one had glomerulonephritis.

The older works related gout to nephrolithiasis. Garrod wrote: "In later years, gout develops when in early life gravel and calculi are formed." There is some difference of opinion about this relationship at present: Hench found stones in 11% of his gouty patients: Schnitker and Richter consider nephrolithiasis entirely incidental.

Pathology of gout

From a purely clinical point of view, gout appears, from most descriptions, a distinct entity; from a pathological point of view, the distinction is far

from clear-cut. Nichols and Richardson point out that one of the primary causes of degeneration of articular cartilage may be the deposit uric acid crystals in the cartilage in gout. These cause erosion of the cartilage. The constant irritation of the foreign body causes the perichondrium to proliferate and to lay down new cartilage. The final result, they say, cannot be distinguished from the usual osteoarthritis except for the presence of urate crystals.

Kraggs, who has carefully reviewed the Strangeways collection of joints in the museum of the Royal College of Surgeons, probably the largest collection of its kind in the world, feels that "gout is very closely akin to rheumatoid affections." "A urate of soda deposit is the one diagnostic feature of gout, and in its absence it is impossible to distinguish the gouty joint from one that has the characteristics either of osteoarthritis or of rheumatoid arthritis." "In joints that have been affected by gout for a number of years, there are usually found 'mixed' arthritic changes such as lipping, grooving, eburnation, and bony and fibrous ankylosis, with microscopic evidence of degeneration of bone and cartilage. This association of hypertrophic and degenerative signs is present in almost every one of the Strangeways specimens of gouty joints."

One may object that the pathologist is seeing the end stage of the gouty process. It is true; nevertheless, the very similarity of that end process with that of rheumatoid arthritis and osteoarthritis may cause one to suspect a certain degree of overlapping of the clinical symptomatology of gout and of chronic arthritis.

The deposition of sodium urate crystals may occur in many locations other than the joints: in ligaments, tendons, joint capsules, periosteum, tendons, bursae and in subcutaneous and intermuscular connective tissues. More rarely, deposits are found in bronchi, heart muscle, pericardium, pleura, meninges, etc. There is considerable difference of opinion regarding the mechanism of the deposition. It appears that urates are deposited in previously uninjured or, according to others, previously injured, areas. In any case, the urate appears to be definitely irritating, perhaps chiefly of the nature of a foreign body. Moreover, when the deposits become large, they produce necrosis of adjacent structures, commonly ulcerating through to the surface where they may exude a milky or chalky substance.

The gouty bronchitis, the gouty heart disease, the gouty nephritis, etc. of the older clinician were, undoubtedly, simply bronchitis, heart disease, nephritis, etc. occurring in the gouty, However, tophi are certainly occasionally responsible for visceral disease. Hench reports a case of von Mueller and Brogsitter in which a tophus in the bundle of His was responsible for a complete heart block.

Treatment

Schnitker, in writing of the history of the treatment of gout, remarks that his subject might as well have been entitled "the slow progress of medicine" for "gout is one of the oldest described diseases, and really the treatment of it has changed very little through many centuries." Williams called gout "the reproach of medicine."

During the acute attack of gout, the part affected should be elevated. It should be protected from the weight of the bedclothes by a cradle. Hot applications of saturated magnesium sulphate may give great relief, but sometimes heat aggravates the pain, then cold compresses may soothe.

Morphine may be necessary for relief of pain. The time-honored remedy is wine of Colchicum in 15 to 20 minim doses every 3 or 4 hours during the first few days of an attack. This must be discontinued if diarrhea is provoked. Some prefer the pure alkaloid colchicin. Cohen recommends colchicin, grains 1/60, administered by mouth, three times daily, in the beginning of an attack. As the attack subsides, he gives the drug only every 4th week.

Hench recommends cinchophen (atophan) in doses of 7½ grains (0.5 gm.) from 3 to 5 times daily during the acute attack.

A purine-free diet is recommended during the acute attack. Cohen suggests that this be a liquid diet during the first few days. Hench recommends ample fluids (10 to 12 glasses daily) and sweetened fruit juices. He notes that the injection of small doses of foreign protein has hastened recovery in some.

Most authors insist that treatment must be continued during the free intervals. During this period, as well as in chronic gout, a low purine diet is recommended. Hench includes periodic treatment with cinchophen during the interval regimen. He recommends 7½ grains of the drug, 2 or 3 times a day, 3 consecutive days a week, but not on the other days. In addition, sodium bicarbonate is given in 5 to 10 gram doses daily, but not at the same time as the cinchophen. He says that he has not seen any significant ill effects of cinchophen among his patients with gout. He recommends that salicylates be given in interrupted courses if cinchophen cannot be given.

Cohen considers cinchophen too toxic for use even in gout. He prefers colchicin given for one week out of every four. Lichtwitz gives the patient lemons and administers one-half to one teaspoonful of washed sulphur at night.

Llewellyn has said, "any attempt to formulate a set dietary proves but a snare and a delusion. No regimen is applicable to the gouty as a class, nor even to the individual 'gouty' at all periods of his life history." Gudzent feels that acute attacks of gout are related to dietary idiosyncrasies. He has shown that a number of such patients reacted to skin tests with fish, eggs, milk and wheat, and were definitely improved by abstinence from these foods. One of his patients had a severe attack of gout every time he took milk (purine-free). Perhaps skin tests for foods and elimination diets will become more important in formulating regimen for gouty patients in the future. In the meantime, most authors prefer the time-honored purine-free and purine-low diets.

The purine-free diet is as follows:

Foods permitted: milk, cream, butter, cheese, eggs, white bread, rice, macaroni, sago, tapioca, cabbage, cauliflower, lettuce, watercress, fruit, sugar, honey, jelly, marmalade, potatoes, onions.

Foods forbidden: tea, coffee, coca cola, fish, fowl, meat, glandular organs, brown bread, pears, beans, ale and beer.

The purine-low diet is as follows:

Foods permitted: cereals, potatoes, rice, green vegetables and salads, fresh and stewed fruits, ham, bacon or beef once a week; chicken, lamb or mutton, once or twice a week; simple desserts such as junket, prune or fig whip, orange, lemon, grape, pineapple or apricot gelatin; bread, rice or tapioca pudding, plain vanilla ice cream.

Foods forbidden: meat, broth and extracts; strong tea or coffee; alcoholic beverages; liver, kidney, sweetbreads; rich sauces and gravies; condiments and spices; pastries and fried foods; strong flavored foods; rhubarb.

Recently, Lockie and Hubbard have pointed out that gouty attacks precipitated by ingestion of a high fat, low carbohydrate diet, subsided promptly when the patients were given a low fat, high carbohydrate diet. The latter may prove a valuable addition to the dietary armamentarium against gout.

Spa baths and mineral waters are still widely recommended particularly in Europe.

Bassler has recently recommended oxygen by rectal instillation in chronic gout. He says that in 17 of 22 cases he obtained prompt and striking improvement.

Hench recommends removal of large tophi under local anesthesia. He points out that aside from the question of proving the diagnosis, there are 2 possible indications for removal of large tophi: (1) to prevent ulceration which may occur in fingers and toes if the masses become large enough to interfere seriously with the nutrition of the skin and subjacent parts and (2) to relieve the kidneys if possible. The body tries to absorb and excrete the urates. Small tophi may be completely resorbed. Surgical removal of tophi might be expected to relieve the kidneys of the equivalent of months or years extra work excreting urates, depending upon the size of the tophaceous deposits.

To prevent exacerbations, attention should be given to elimination of trauma. Patients should be cautioned to avoid unusual excesses in golf or walking and to avoid use of tight shoes or other traumatizing agents. Moderate exercise is to be encouraged. Foci of infection are eliminated, not because there is much to indicate that gout is infectious, but because focal infections, tonsillitis, acute dental abscesses, etc. seem to provide sufficient disturbance in metabolism to precipitate certain attacks. Hench recommends that persons who have gout or who are suspected of being susceptible to attacks should have a diet low in purines and a few small doses of cinchophen for a few days before undergoing operative procedures.

Although the current treatment of gout, as outlined above, is fairly satisfactory, it is admittedly empirical. Even today, we may conclude a discussion of treatment with Sydenham's conclusion of his famous dissertation on gout: "As for a radical cure, this lies like Truth, at the bottom of a well; and so deep is it in the innermost recess of Nature that I know not when or by whom it will be brought forward into the light of day."

Conclusions

1. Gout is one of the oldest described diseases. Descriptions of what is unmistakably gout date back to 500 B.C.

2. Gout is a disease of temperate climate.

3. Gout was apparently much more frequent formerly than at present. Whether gout was formerly very common or whether much chronic arthritis was uncritically termed gout is a matter for speculation.

4. Gout is much more common in men than in women. Women appear to be much more liable to the disease after the menopause.

5. The average age in the onset of gout is between 40 and 50 years.

6. Attacks of gout are more common in the spring and winter months.

7. Older workers found that the gouty individual came from gouty families. Modern authors feel that their patients largely served their afflictions.

8. Undoubted cases of gout have been attributed to lead.

9. Excessive eating and drinking are important in the causation of gout. A high proportion of the gouty are heavy drinkers.

10. Trauma: blows, sprains and even operative procedures may precipitate gouty attacks.

11. The diagnostic criteria of gout include: (a) joint involvement, classically that of the great toe, (b) tophi, (c) elevation of the blood uric acid from the normal values of 2 to 4 mgs. % to 5 to 20 or more mgs. %., (d) so-called characteristic punched-out areas at the articular ends of bone.

12. Additional aids in diagnosis include: (a) dry eczema of the skin and hyperkeratosis about the fingernails and toenails, (b) ground down teeth, (c) ulnar deflection of the terminal joint of the index finger, and (d) precipitation of attacks on high fat, low carbohydrate diet.

13. Cases of acute gout are usually

ushered in by gastro-intestinal upset. The pain in the joint or joints is accompanied by fever with temperature to 103°F and polymorphonuclear leucocytosis.

14. Chronic gout is present when tophi are demonstrable and when joint deformities appear.

15. Gouty manifestations in parts remote from joints, in the bronchi, in the skin, in the kidneys, etc. constitute abarticular or irregular gout.

16. Retrocedent gout probably does not exist.

17. Attacks of gout appear in some patients to be associated with specific hypersensitiveness.

18. The nephritis of gout is predominantly vascular in origin (arterio- and arteriolosclerotic).

19. Pathologically, gouty joints cannot be distinguished from the joints of chronic arthritis except by the presence of sodium urate crystals in the former.

20. Treatment includes the use of colchicum or cinchophen plus purine-free or purine-low diet.

21. Prophylaxis demands avoiding of excesses - be it food or drink or exercise, etc.

22. Axioms of diagnostic value (after Hench). Suspect gout in cases of (1) acute rather severe arthritis of short duration, often coming on not immediately with but within a few hours of minor trauma; (2) acute recurring arthritis with complete remissions or of chronic arthritis with such a history preceding the chronic phase; (3) chronic arthritis with nephritis with or without renal colic or stone; (4) chronic arthritis with peri-articular ulcer or assymmetric swelling.

Bibliography

1. Allbutt, Sir T. C.
Gout
Oxford Med. IV: 80-130.
Oxford Univ. Press, N.Y.C.

2. Bassler, A.
Digestive manifestations of gout.
Med. Rec. 140: 667-669, 1934.

3. Berglund, H.
How much do we know about the relationship between uric acid and gout?
Med. Clin. N. Amer. 8: 1625-1650, 1925.

4. Cain, E. F.
Report of a case of gout.
Proc. Staff Meet. Mayo Clinic 6: 1-5, 1931.
Discussion by P. S. Hench.

5. Cohen, A.
Gout (podagra)
Med. Rec. 141: 456-459, 1935.

6. -----
Gout
Am. J.M.Sc. 192: 488-493, 1936.

7. Cmunt, E.
Cutaneous symptoms and food allergies in atypical gout.
Arch. Med. Hydrol. 14: 146, 1936.

8. Futcher, T. B.
The occurrence of gout in the United States: with an analysis of thirty-six cases.
J.A.M.A. 39: 1046-1050, 1902.

9. Garrod, A. B.
The nature and treatment of gout and rheumatic gout.
Walton and Maberly, London, 1859.

10. Gudzent, F. and Holzmann, E.
Klinische und morphologische Beitrage zum Gichtproblem.
Zeitschr. klin. Med. 106: 117-128, 1927.

11. Hench, P. S.
Remarks on gout presentation of two cases.
Proc. Staff Meet. Mayo Clinic 8: 717-719, 1933.

12. ------, Bauer, W., Fletcher, A. A., Ghrist, D., Hall, F., and White, T.P.
The present status of the problem of "rheumatism" and arthritis; review of American and English literature for 1934.
Ann. Int. Med. 9: 883-982, 1936.

13. ----- and Darnall, C. M.
A clinic on acute, old-fashioned gout; with special reference to its inciting factors.
Med. Clin. N. Amer. 16: 1371-1393, 1933.

14. -----, Vanzant, F. R. and Nomland, R.
Basis for the differential diagnosis of gout. A clinical comparison of 100 cases each of gout, rheumatic fever and infectious arthritis.
Tr. Assoc. Am. Phys. 43: 217-229,1928.

15. Herrick, W. W. and Tyson, T.
Gout--a forgotten disease.
Am. J. M. Sc. 192: 483-487, 1936.

16. Knaggs, R. L.
A report on Strangeway's collection of rheumatoid joints in the museum of the Royal College of Surgeons,
Brit. J. Surg. 20: 111-129, 309-330, 425-443, 1932-1933.

17. Krafka, J.
A neglected factor in the etiology of gout.
J. Bone and Joint Surg. 17: 1049-1051, 1935.

18. Lambert, A.
Discussion.
J.A.M.A. 74: 1629, 1920.

19. Lichtwitz, L.
Gout.
Bull. N. Y. Acad. Med. 10: 306-319, 1934.

20. Llewellyn, L. J.
Aspects of rheumatism and gout.
William Heinemann, London, 1927.

21. Lockie, L. M. and Hubbard, R. S.
Gout: changes in symptoms and purine metabolism produced by high fat diets in four gouty patients.
J.A.M.A. 104: 2072-2075, 1935.

22. Nichols, E. H. and Richardson, F. L.
Arthritis deformans.
J. Med. Res. 21: 149-222, 1909.

23. Osler, W.
The principles and practice of medicine. 12th edition. Revised by T. McCrae.
Appleton-Century, N.Y., 404-412, 1935.

24. Pringle, G. L. K.
The incidence and criteria of gout.
Arch. Med. Hydrol. 14: 143-145, 1936.

25. Ray, M. B.
The physical treatment of gout.
Brit. J. Phys. Med. 10: 170-172, 1936.

26. Schnitker, M. A.
A history of the treatment of gout.
Bull. Inst. Hist. Med. 4:89-120,1936.

27. Schnitker, M. A. and Richter, A. B.
Nephritis in gout.
Am.J.M.Sc.192: 241-252, 1936.

28. Volini, I. F. and O'Brien, G. F.
Gout.
Med. Cl. N.Amer. 18: 1355-1366, 1935.

29. Widal, F., Abrami, P. and Joltrain, E.
Les cuti-reactions aux vins chez les goutteux. Presse Med.2:1425-1426, '25.

30. Williams, R.
Gout: the reproach of medicine.
Med. Rec. 135: 143-144, 1932.

31. Williamson, C. S.
Gout: a clinical study of 116 cases.
J.A.M.A. 74: 1625-1629, 1920.

V. GOSSIP

The second annual Harold S. Boquist Lecture will be given today at 4:00 P.M. in the Medical Science Amphitheatre by Dr. Albert Russell, of the United States Public Health Service. The subject will be "Silicosis".....The more he read, the more he became certain that if he did not already have the gout, he would soon succumb. I refer to our good friend Isadore J. Pass, the gentleman responsible for the preparation of this program today. A good fisherman, a good eater, and a good fellow, it was hardly fair to ask him to report on this subject, but as usual, he did a very fine job in spite of the mental suffering it caused him.....Our guest discussion leader today, Philip S. Hench, among his other accomplishments, is an outstanding organist. His interest in arthritis and allied disorders has given him national recognition as a student in this field. We are very glad that it was possible for him to be with us today... ....Surgeon William Thomas Peyton has returned from the frozen north, where he spent his annual vacation in the wilds on a hunting expedition. He will not talk.....Staff meetings will recess during the Christmas Holidays - the last two weeks in December. The annual Christmas Party will be held December 17th. .....Recent investigations disclosed that practically every claim made for tooth paste and tooth powder could be discounted. Part of this study was made in our own dental school. A demonstration of what could be done with a good brush and water, salt or soda, was amazing.....According to the American Medical Association, the three common requests for information from lay persons are: marking babies, cosmetics, and weight control. Marking babies is first. All who can appreciate our trend in enthusiastic therapy should read the letter "Water, Water Everywhere" in last Saturday's issue (Nov. 28) of the Journal of the American Medical Association.. .....It is said that real humility is necessary for the cultivation of a good sense of humor. It is the old question of being able to first laugh at yourself.. .....The Medical School is cooperating with the Minnesota State Department of Health in planning traveling short courses in obstetrics and pediatrics during the coming year. The lecture series will consist of six meetings, a week apart. The plan has been very successful in other states and is part of the government's aid program in public health education.... ...The Minnesota Branch of the Cancer Research Society group will hold its fall meeting Tuesday, December 8th. Anyone interested is invited to attend. .....The annual sale of Christmas Seals, now in progress, is being conducted for the thirtieth time. The seals have never received a more favorable reception. It is interesting to note that in Hennepin County part of the fund is devoted to supplying every physician's office with fresh tuberculin each week, so that he may test all patients in his office by this preliminary screen. It might not be a bad idea for you to buy some and put them on your Christmas packages. In Minnesota the campaign is being conducted by the Minnesota Public Health Association; in Minneapolis and St. Paul by the Hennepin and Ramsey County Associations.....

Adios.