GETTING TO KNOW

by Ken Flieger

GOUT

Say the word **"gout"** and some people will think of a bloated king surveying the remains of a sumptuous feast, wine glass in hand, swollen foot propped on a pillow—looking for all the world like the dismal product of a grossly overindulgent life.

There are a couple of flaws in that conventional image. We know, for example, that **gout** doesn't afflict only the privileged classes and that women, too, are susceptible, though a lot less than men.

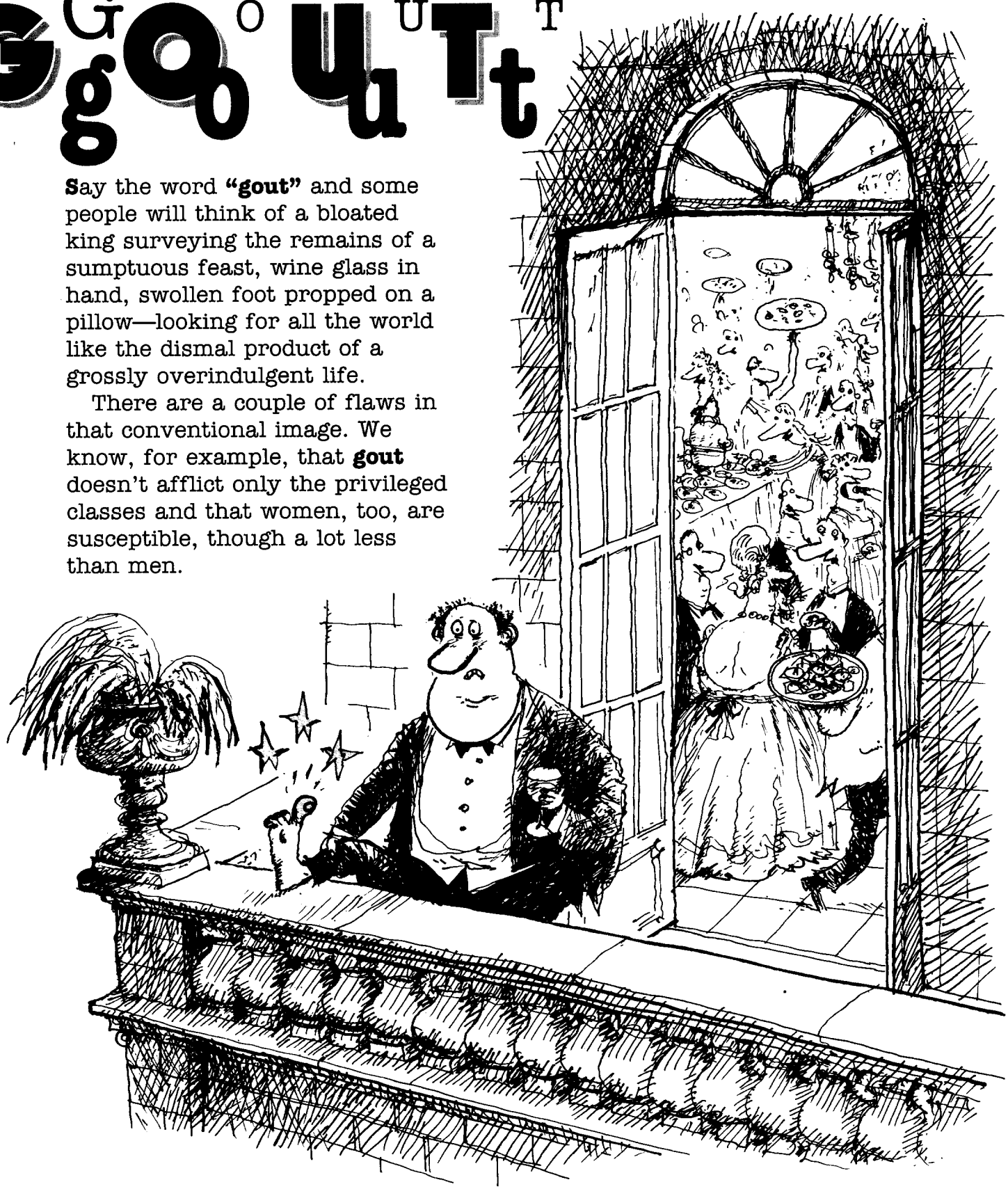


ILLUSTRATION BY RICHARD THOMPSON JR.

THE BIG TOE, HEEL, INSTEP AND ACHILLES TENDON ARE AMONG THE PLACES THAT GOUT ATTACKS FIRST.

But still there's a good deal right with that picture. It correctly reflects that:

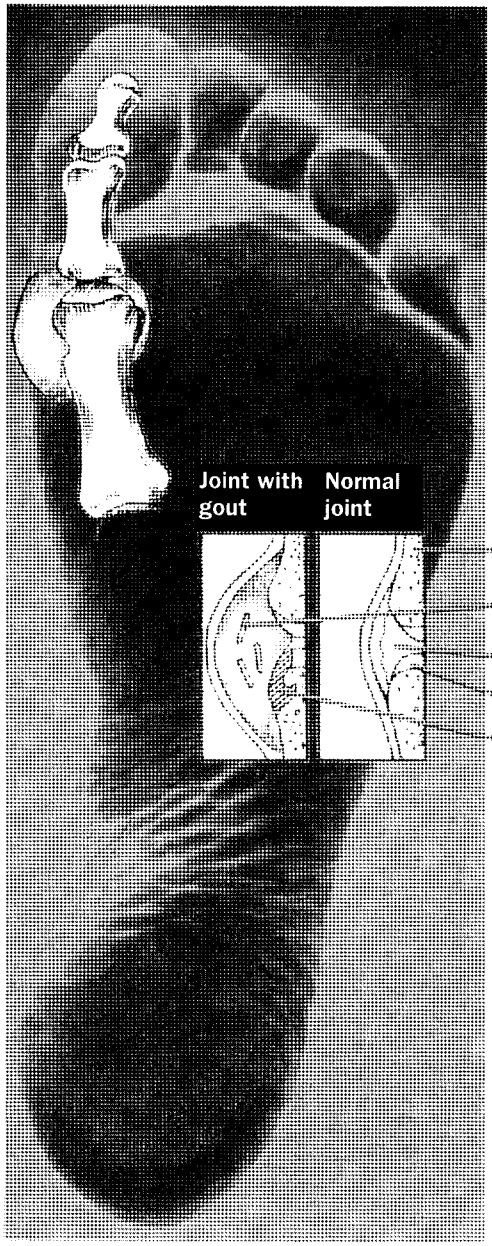
- About 90 percent of people afflicted with gout are men over 40.
- Obesity in general, and in particular excessive weight gain in men between ages 20 and 40, has been shown to increase the risk of gout. In fact, about half of all gout sufferers are overweight.
- Alcohol abuse and so-called "binge" drinking are associated with gout, as is eating purine-rich foods such as brains, kidneys, liver, sardines, anchovies, and dried beans and peas.

In addition, careful scientific surveys have shown that occupational exposure to lead, the use of certain drugs to control high blood pressure, some surgical procedures, family history (possibly a genetic predisposition), and trauma are all linked to an increased risk of gout. Indeed, the prevalence of gout—the number of gout sufferers for each 100,000 people—is rising rapidly in the United States and other developed countries. Some authorities believe the increase is related to higher living standards.

Our fanciful image of a gouty Henry VIII (or other bloated monarch) can't show, however, the one common denominator that ties together this mixed bag of risk factors: failure of the metabolic process that controls the amount of uric acid in the blood. For most people, the process works just fine. But in some 1 million Americans, uric acid metabolism has gone seriously haywire. As a result, they suffer from gout.

And suffer they do. An Englishman, Thomas Sydenham, writing in the 17th century, left this unfortunately all-too-accurate description of a typical attack of gout:

The victim goes to bed ... in good health. About two o'clock in the morning, he is awakened by a severe pain in the great toe; more rarely in the heel, ankle, or instep. The pain is like that of a dislocation. [It] becomes more intense ... . So exquisite and lively meanwhile is the feeling of the part affected, that it cannot bear the weight of the bedclothes nor the jar of a person walking in the room. The night is passed in torture ... .



How Gout Gets Going

After several years of abnormal uric acid deposits, uric acid crystals can build up in joints and surrounding tissues. They form large lumpy deposits called tophi, which, if left untreated, can damage joints.

(Source: Arthritis Foundation)

A Crystal Culprit

In spite of the agony and havoc it can cause, uric acid is a normal constituent of the human body. Ordinarily about one-third of the uric acid in our system comes from food, especially foods like those noted earlier that are rich in purines. The rest we produce ourselves through ordinary metabolism.

The body converts purines to uric acid.

ACUTE GOUT IS TREATED WITH DRUGS THAT BLOCK THE INFLAMMATORY REACTION.

The level of uric acid in the blood fluctuates in response to diet, fluid intake, overall health status, and other factors. Men normally have somewhat more uric acid than women do (although the difference begins to narrow after menopause), and in both sexes it tends to increase with advancing age.

Higher-than-normal amounts of uric acid in the blood, a condition called hyperuricemia, is quite common and only rarely warrants medical treatment. On the other hand, sustained hyperuricemia is the primary risk factor for gout. It's safe to say that, while not all people with hyperuricemia develop gout, virtually everyone with gout is hyperuricemic. It works this way:

At normal and even somewhat elevated levels, uric acid stays in solution in the blood. It moves through the circulation, gets filtered by the kidneys, and is excreted in the urine. When, however, blood uric acid levels rise above a certain concentration (which varies with temperature and blood acidity), it forms needle-like crystals that lodge in or around a joint.

In response to irritation caused by uric acid crystals, the skin covering the affected area rapidly becomes tight, inflamed, swollen, and red or purplish. These classical signs of inflammation, together with sudden and extreme pain (just as Thomas Sydenham described), strongly suggest an acute attack of gout. The diagnosis is confirmed by laboratory finding of uric acid crystals in fluid taken from the affected joint.

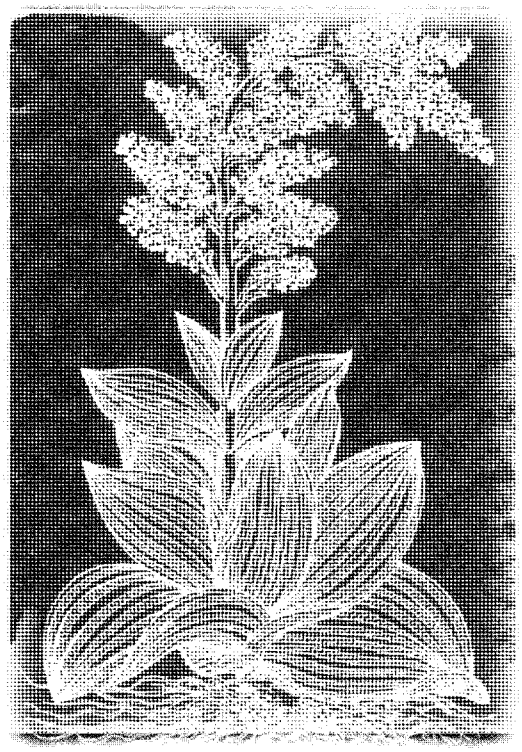
Why is the big toe the most common site for an initial gout attack? Perhaps because first, the extremities are a bit cooler than other parts of the body, and uric acid crystals form more readily at lower temperatures; and second, normal walking and standing subject the feet to considerable stress. Together, these factors might explain why the big toe, heel, instep, and Achilles tendon are among the places that gout attacks first. Other targets, especially in untreated patients who have recurrent attacks of gout, are the knee, elbow, wrist, fingers and, less often, the shoulder, pelvis, spine, and internal organs.

Gout is classified as a form of arthritis because it is initially and predominantly a disease of the joints. Other similar conditions exist; one called "pseudogout" is somewhat milder than true gout and is caused by calcium rather than uric acid crystals. Infection or trauma to the affected area can mimic gout and mislead both patients and health professionals. Accurate diagnosis is essential for appropriate treatment.

Without treatment, an initial acute attack of gout will run its painful course within several days or a few weeks, by which time all outward evidence of the disease disappears. The next acute attack—50 or more percent of gout sufferers will have a second attack—may not occur for months or years. Subsequent attacks, however, are likely to be more frequent, more severe, and more destructive to joints and other tissue unless the problem is treated. Over time, uric acid crystals accumulate in the body, causing gritty, chalky deposits called tophi that are sometimes visible under the skin, particularly around joints and in the edges of the ears. Tophi may also form inside bone near the joints, in the kidneys, and in other organs and tissues, causing permanent damage. Advances in treatment, fortunately, have made this kind of chronic gout extremely rare.

Treatment

As with most illnesses, effective treatment of gout depends on a correct diagnosis. Gout can be unequivocally diagnosed by telltale uric acid crystals in joint fluid. But appropriate treatment is often started after a "clinical" diagnosis based on painfully obvious signs and symptoms and other relevant factors, such as the patient's uric acid level, age, weight, gender, diet, and alcohol use. If this picture adds up to a strong suspicion of gout, treatment can be started with the immediate goal of



autumn crocus

GOUT IS CLASSIFIED AS A FORM OF ARTHRITIS BECAUSE IT IS INITIALLY AND PREDOMINANTLY A DISEASE OF THE JOINTS.

arresting the acute attack.

Acute gout is treated with drugs that block the inflammatory reaction. One of the oldest agents known to be effective against acute gout is colchicine, which comes from a common European plant, the autumn crocus, and is marketed in this country primarily as a generic drug. An English clergyman, Sidney Smith, said a century and a half ago that he had only to go into his garden and hold out his gouty toe to the plant to obtain a prompt cure. This may have been an exaggeration, but a rapid response to colchicine suggests that the patient does indeed have gout.

This old, powerful remedy is now used less often than it once was because it can be quite toxic, causing nausea, vomiting, diarrhea, and stomach cramps when taken by mouth and severe (even fatal) blood disorders when taken intravenously. Moreover, modern agents, specifically nonsteroidal anti-inflammatory drugs (NSAIDs) are highly effective against acute gout and less toxic than colchicine. To treat an acute case of gout, the first choice of many physicians is the NSAID Indocin (and other brands of indomethacin). Naprosyn (naproxen) is another NSAID commonly used in acute gout.

Steroid drugs, such as Deltasone (and other brands of prednisone) and Acthar (and other brands of adrenocorticotropic hormone), may be used if NSAIDs fail to control an acute attack. Steroids may be taken by mouth or by injection into the bloodstream or muscle.

Drug treatment usually relieves the symptoms of acute gout within 48 hours. Subsequent treatment, which may well be lifelong, is aimed at preventing further attacks by controlling uric acid in the blood—keeping it below concentrations at which crystals can form. Two main treatment approaches are used, in some cases simultaneously.

One approach is to slow the rate at which the body produces uric acid. Zyloprim (allopurinol) has been approved for the treatment of gout and is frequently prescribed for gout patients who have uric acid kidney stones or other kidney problems. Side effects include skin rash and upset stomach, both of which usually subside as the body becomes used to the drug. Zyloprim makes some patients drowsy, so they need to be cautious about driving or using machinery.

The other approach to controlling gout following an initial acute attack is to increase the amount of uric acid excreted in urine. Two so-called uricosuric drugs commonly used for this are Benemid (probenecid) and Anturane (sulfinpyrazone), both approved by FDA for gout treatment. In addition to lowering blood uric acid levels, these drugs help dissolve deposits of uric acid crystals around joints and in other tissue. Zyloprim is also used to dissolve tophaceous gout in uric acid over-producers. Uricosurics can cause nausea, stomach upset, headache, and a potentially serious skin rash.

Drugs to control uric acid levels may, paradoxically, prolong an acute attack. For this reason, Benemid, Anturane and Zyloprim are not used during the acute stage of gout. They may, in fact, induce gout flare-ups during the early part of long-term use. Accordingly, colchicine in a dose low enough to avoid toxic side effects is sometimes prescribed to prevent acute attacks during this phase of treatment.

Common-Sense Measures

Better understanding of what gout is, what causes it, and how to treat it has perhaps dispelled some of the traditional myths about what has been erroneously called "the disease of kings." Then, too, folk wisdom about gout, coupled with good science and medicine, points to measures that prudent people can take to prevent or at least lessen the severity of the condition.

Many authorities and the Arthritis Foundation, which supports research and public service programs relating to gout, advocate weight control as a logical aid to gout prevention. They point out, however, that people who are overweight should get professional guidance in planning a weight-reduction program, because fasting or severe dieting can actually increase uric acid levels.

Experts generally agree that people with gout can eat pretty much what they want, within limits. People who have kidney stones caused by uric acid may need to avoid purine-rich foods. But this problem can usually be handled effectively with drug treatment.

Curbing alcohol use and avoiding "binge" drinking can reduce the likelihood of acute attacks. So can drinking six or eight glasses of water a day, which dilutes uric acid and aids its removal by the kidneys. Some medicines—in particular the thiazide diuretics ("water pills") used to control high blood pressure—tend to increase uric acid levels. A gout patient taking one of these drugs may have to switch to another type of diuretic or blood pressure medicine.

Finally, although uncommon, it might be helpful to find out if an environmental or occupational exposure to lead is playing a role in a patient's problem with gout.

While a cure for gout—a treatment that gets rid of the condition once and for all—isn't on the horizon, reliable and effective ways of diagnosing gout and keeping it under control constitute one of the more impressive success stories of modern medical science.

There may be no sure-fire way to keep a person from having that first agonizing attack, but prompt treatment can minimize the risk of further attacks and virtually rule out the damaging and crippling effects of chronic gouty arthritis. ■

Ken Flieger is a writer in Washington, D.C.