This booklet provides information and answers to your questions about this condition.
Gout is a disease in which crystals form in your joints. An attack of gout can be extremely painful and continuing crystal formation can cause long-term joint damage. In this booklet we’ll explain what causes gout, how an attack can be treated, and what can be done to get rid of the crystals and stop further attacks. We’ll also suggest where you can find out more about living with gout.

At the back of this booklet you’ll find a brief glossary of medical words – we’ve underlined these when they’re first used.
What’s inside?

3  Gout at a glance
4  What is gout?
4  What are the symptoms of gout?
6  What causes gout?
9  What is the outlook?
10  How is gout diagnosed?
   – What tests are there?
11  What treatments are there for gout?
   – Treatments for acute attacks of gout
     – Non-steroidal anti-inflammatory drugs (NSAIDs)
     – Colchicine
     – Steroids
     – Other treatments for acute attacks
   – Ongoing treatments to reduce urate
     – Allopurinol
     – Febuxostat
     – Other urate-lowering drugs
     – Treatment of joint damage
17  Self-help and daily living
   – Healthy lifestyle choices
   – Complementary medicine
19  Research and new developments
21  Glossary
22  Where can I find out more?
24  We’re here to help
Gout can be very painful. Fortunately, there are a number of treatments available that can ease the pain and others that can reduce the risk of further attacks or even get rid of the problem altogether. With suitable treatment, gout is unlikely to result in permanent joint damage.
What are the symptoms of gout?

An attack of gout can cause very painful symptoms, including:

- extreme pain that comes on quickly in the affected joint (often the big toe)
- affected joints feeling hot and very tender to the touch
- affected joints looking swollen with the overlying skin often shiny and red.

Gout can cause long-term severe and irreversible joint damage.

What causes it?

Gout is caused by too much uric acid, or urate, in your body. Urate is produced by the breakdown of chemicals called purines in your body; most of which are made naturally by your body but some come from your diet. People with gout often have kidneys that are unable to flush out extra urate through urine, and when it builds up above a critical level it can form crystals of sodium urate, particularly in your cartilage. Occasionally these crystals escape from the cartilage and trigger sudden painful inflammation of the joint lining.

What treatments are there?

Initial treatments for acute attacks include:

- non-steroidal anti-inflammatory drugs (NSAIDs)
- colchicine tablets
- steroids (injected into the joint or muscle, or as tablets)
- applying ice packs and resting the joints.

Longer-term treatments aim to lower urate levels and reduce the risk of further attacks. These include:

- allopurinol or febuxostat (which reduce the amount of urate your body makes)
- uricosuric drugs (which increase the amount of urate your kidneys get rid of).

How can I help myself?

Try the following to reduce your risk of an attack:

- Lose weight if you’re overweight.
- Have a balanced diet and avoid eating excessive amounts of purine-rich foods (for example offal, oily fish, yeast extracts).
- Avoid dehydration by drinking plenty of water.
- Avoid drinking too much alcohol.
- Include fruit, vegetables and other foods rich in vitamin C in your diet.
- Avoid fructose (this is often present in fizzy drinks).
What is gout?
Gout is defined by having sodium urate crystals in your joints. These may be present for years without you knowing they are there, but on occasions they can trigger an attack of gout. Gout is an intensely painful form of arthritis. Attacks of gout usually come on very quickly, often during the night. Doctors describe this sudden development of symptoms as ‘acute’. Gout is the form of arthritis we understand the best, and this has led to a range of therapies to treat acute attacks and control the condition.

Gout is the most common type of inflammatory arthritis, affecting 2.5% of adults in the UK. It affects more men than women and can affect men of any age. Women rarely develop gout before the menopause. It gets more common with increasing age in both men and women, affecting 15% of men and 6% of women over the age of 75. Gout has been getting more common in recent decades in many countries, including the UK. This is mainly because we are living longer, and more people are getting overweight or obese.

At one time it was thought that gout was caused simply by overeating and drinking too much alcohol. While this can make attacks of gout more likely, it’s not the whole story.

Gout is caused by chemical processes that take place in your body. A substance called urate builds up, sometimes because your kidneys aren’t able to get rid of it quickly enough. Over a period of time this leads to urate crystals forming in and around your joints. Once there are a lot of crystals in your joints some of them can trigger a sudden painful episode of severe joint inflammation (an ‘attack’) which usually settles back to appear normal within a week. If untreated these attacks get more common and spread to involve new joints.

What are the symptoms of gout?
Urate crystals cause inflammation, meaning the affected joint becomes intensely painful, red, hot and swollen (see Figure 1). The skin over the joint often appears shiny and may peel. Attacks typically affect the big toe and usually start at night. The symptoms develop quickly and are at their worst within 12-24 hours of first noticing anything is wrong. Any light contact with the affected joint is painful – even the weight of a bedsheet or wearing a sock can be unbearable.

Although gout most often affects the big toe, other joints may also be affected, including other parts of the feet and ankles, knees, elbows, wrists and fingers. If several joints are inflamed at once this is called polyarticular gout. It’s very rare to have gout in joints towards the centre of the body such as the spine, shoulders or hips.

Urate crystals can also collect outside of the joints and even be seen under the skin, forming small, firm white lumps called tophi (see Figure 2). These aren’t usually painful but can be embarrassing and disfiguring, and can get in the way of
Gout most commonly affects the big toe. The joint becomes red, hot, swollen and extremely painful.

Urate may collect under the skin, forming small white pimplies (tophi), but these aren’t usually painful.

Figure 1: Gout affecting the big toe

Figure 2: Tophi caused by urate collecting under the skin

Tophi on the ear

Tophi on the hand
clothing. Sometimes they get inflamed, break down and leak pus-like fluid containing gritty white material – the urate crystals themselves.

**What causes gout?**

Gout occurs in people who have levels of urate in their blood that are persistently above a critical level which allows urate crystals to form. About two thirds of the urate in our bodies comes from the breakdown of purines which are naturally present in the cells of our bodies. The other third comes from the breakdown of purines in some foods and drinks.

Having urate in your blood doesn’t mean you’ll definitely develop gout – it’s normal and healthy to have some urate in the bloodstream. When urate levels start to build up, your body usually gets rid of any excess urate through your kidneys into your urine; however, if your body is making too much urate or your kidneys are unable to remove enough urate, then urate levels start to rise. If the level goes above a certain point (the saturation point), it’s possible for urate to form crystals of sodium urate. These crystals mainly form in and around joint tissues, especially joints at the ends of your legs and arms, such as your finger and toe joints.

Figure 3 shows a joint with urate crystals. The crystals gradually build up in your cartilage and other joint tissues over years. You will not know this is happening. When there are a lot of crystals in your joints some of them can spill out into the joint cavity (the space between the bones). This process is called crystal shedding. The hard, needle-shaped
crystals touch the soft lining of the joint (the synovium) and make it very inflamed very quickly. The inflammation process breaks down the crystals that have become loose inside the joint, and the attack gradually settles over a few days or weeks, depending on how many crystals spilled out.

Apart from causing sudden attacks of inflammation, a build-up of crystals can eventually lead to tophi forming in and around your joints. These hard tophi can grow and cause pressure damage to your cartilage and bone. This is just like the damage caused by osteoarthritis and can cause more regular, daily pain when you use the affected joints. At this stage the condition is often called chronic tophaceous gout. Some tophi may be seen and easily felt under the skin, but by this time the unseen part of the tophi in your joints and deeper tissues are usually quite extensive.

Several factors can affect the level of urate in your body:

- The genes you’ve inherited may make it more likely that your kidneys don’t flush urate out of your body as well as they should, even though your kidneys are otherwise completely normal and healthy. This is the most common cause, especially when there are several family members affected.
• The bigger the body the more urate is produced each day, so if you’re overweight or obese it could cause your body to make more urate than your kidneys can get rid of.

• If you have high levels of cholesterol and fats in your blood (a condition called hyperlipidaemia), high blood pressure or late-onset (type 2) diabetes, your kidneys won’t be able to get rid of urate as well as they should, so all these conditions tend to be linked with raised urate levels. This combination of problems is often called metabolic syndrome.

• Kidney disease may mean that your kidneys aren’t able to process urate as well as they should.

• Some tablets such as diuretics (water tablets) reduce your kidneys’ ability to get rid of urate effectively.

• Rarely, if you have a chronic blood disorder that causes your body to produce too many blood cells, the level of urate produced by the breakdown of those cells may be higher than your kidneys can cope with.

Where a particular cause can be identified (such as kidney disease or regular use of diuretics), the condition is called secondary gout. However, most gout is primary and is usually due to a combination of factors, for example, through having inherited kidneys that aren’t very good at getting rid of urate and then becoming overweight.

If you’re prone to gout and have urate crystals in your joints, several things can encourage urate crystals to shake loose from your cartilage and trigger an acute attack. These can include:

• a knock or injury to the affected joint
• an illness, such as pneumonia or flu, that makes you feverish
• an operation – this also puts your temperature up a little
• overeating and drinking too much alcohol
• dehydration.

Similar attacks can be caused by a condition called acute calcium pyrophosphate crystal arthritis (acute CPP crystal arthritis), which was sometimes previously called ‘pseudogout’. In this type of arthritis it’s calcium crystals that are deposited in joint cartilage rather than urate crystals. Acute CPP crystal arthritis affects the knee and other joints more than the big toe and is most common in people with osteoarthritis. You may be more at risk of gout attacks if any of your parents or grandparents had it.

See Arthritis Research UK booklet Calcium crystal diseases including acute CPP crystal arthritis (pseudogout) and acute calcific tendinitis.
What is the outlook?

Attacks can vary from person to person. Some people only have an attack every few years, while others have attacks every few months. In time, though, attacks tend to happen more often and new joints are affected.

Although acute attacks of gout are very dramatic, the inflammation goes down fairly quickly and the attacks themselves probably don’t cause long-term joint damage. However, a continued build-up of urate crystals and formation of hard tophi can damage your cartilage and bone, leading to long-term (chronic) arthritis.

With modern treatments and possibly some changes to your diet and lifestyle, this type of damage can usually be prevented by bringing urate levels in your tissues down below the point at which crystals form.

Lowering your urate levels will prevent new crystals forming and slowly break down the crystals that are already there. It may take as long as two to three years of treatment to completely clear your body of urate crystals, but once they’re gone then the risk of acute attacks of gout and of further joint damage from tophi is removed.

Because gout is associated with metabolic syndrome, you should pay special attention to your cholesterol levels, blood pressure and diabetes. If these are treated effectively it can help reduce your urate levels.

As well as the threat of causing attacks of gout, persistently high urate levels can lead to other health concerns. For example:

- High urate levels can cause inflammation and furring of the lining of arteries and lead to increased risk of heart disease (for example, angina, heart attacks, atrial fibrillation, heart failure) and stroke.
- The crystals in your joints can cause long-term low-grade inflammation in between the acute attacks without you having any symptoms.
- High levels of urate can make chronic kidney disease worse.
- There is growing evidence that high urate levels increase the risk of cancers, especially prostate cancer.

Having acute attacks and eventually chronic joint symptoms due to gout is bad enough, but our increasing recognition of these additional health problems has made us realise that gout is a potentially serious condition that needs to be treated appropriately and not ignored.
How is gout diagnosed?

What tests are there?
A diagnosis is often based on your symptoms and an examination of the affected joints, but your doctor may suggest the following tests:

A blood test can measure the amount of urate in your blood. The critical serum level of urate (the saturation point) is around 360 μmo/L (equivalent to 6mg/dl).

This is within the normal range for men, and for older women, so being informed that your blood test is ‘within normal limits’ is irrelevant – you need to know if it is above or below this critical level. A raised level of urate strongly supports a diagnosis of gout but can’t confirm it – not everyone with a raised level of urate will develop crystals in their joints, and it’s possible for urate levels in the blood to be normal at the time of an acute attack.

X-rays of joints will reveal joint damage if you have long-standing and poorly controlled gout. However, x-rays are rarely helpful in confirming the diagnosis because they’re usually normal in the early years of having gout. Ultrasound of joints can be used to detect earlier signs of gout, and can be useful where the diagnosis is uncertain.

Synovial fluid examinations involve taking fluid samples from a joint through a needle and examining them under a microscope for urate crystals. This test can confirm the diagnosis but isn’t always practical – it can be difficult and sometimes uncomfortable to draw fluid from a small joint such as the big toe. However, it may be possible to identify a few crystals in a sample taken from your knee, even if you’ve not yet had an attack of gout there. A fine needle inserted into a tophus under your skin can also be used to identify urate crystals.
What treatments are there for gout?
There are two main parts to treating gout (see Figure 4). These are:
• treating the acute attack of inflammation when one or more joints are very inflamed and painful
• ongoing treatment to reduce the level of urate in your blood and get rid of urate crystals.

Treatments for acute attacks of gout
The two most commonly used drug treatments for acute attacks of gout are non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine.

Non-steroidal anti-inflammatory drugs (NSAIDs)
Acute attacks of gout are often treated with oral NSAIDs, which can ease pain and possibly reduce some of the inflammation. Examples include ibuprofen, naproxen and etoricoxib.
Like all drugs, NSAIDs can sometimes have side-effects, but your doctor will take precautions to reduce the risk of these – for example, by prescribing the lowest effective dose for the shortest possible period of time.

NSAIDs can cause digestive problems (stomach upsets, indigestion, or damage to the lining of the stomach) so NSAIDs should be prescribed along with a drug called a proton pump inhibitor (PPI), which will help to protect your stomach.

NSAIDs also carry an increased risk of heart attack or stroke. Although the increased risk is small, your doctor will be cautious about prescribing NSAIDs if there are other factors that may increase your overall risk – for example, smoking, circulation problems, high blood pressure, high cholesterol or diabetes. If you’re on some other medication, including water tablets or warfarin, or if you have other medical conditions, such as chronic kidney disease, it may mean that you shouldn’t take an NSAID, but your doctor will advise you on this.

If you’ve had an attack before, be on the lookout for early signs of another attack and take your prescribed treatment as soon as possible. The earlier you start treating an acute attack the better.

Colchicine
Colchicine is made from the crocus plant. It’s not a painkiller but it’s often very effective at damping down the inflammation caused by the crystals touching the joint lining. As with NSAIDs, colchicine tablets should be taken as close as possible to the beginning of an attack, and certainly within the first 24 hours of the attack starting, otherwise it may not be effective. Your doctor may let you keep a supply so you can start taking them at the first signs.

The recommended dose of colchicine is 0.5 mg two to four times per day, depending on your size, age and whether you have other health problems. Some people are unable to take colchicine because they have side-effects such as nausea, vomiting or diarrhoea. For this reason it’s best to start at a low dose and only increase it if there’s no upset. You shouldn’t take colchicine at the same time as NSAIDs. Colchicine can interact with

See Arthritis Research UK drug leaflet Non-steroidal anti-inflammatory drugs.
many other drugs (including statins for high cholesterol) but your doctor will advise whether this means you should avoid colchicine or temporarily adjust your other medications.

You can also take colchicine in the longer term at a dose of 0.5 mg once or twice a day to reduce your risk of having attacks in the future. However, like NSAIDs, colchicine won’t reduce the urate level in your blood, so it won’t help to get rid of the urate crystals or prevent long-term joint damage.

**Steroids**

If an acute attack of gout doesn’t improve with NSAIDs or colchicine or if you’re at risk of side-effects from these drugs, your doctor may prescribe a steroid injection into the joint or muscle, or a short course of steroid tablets (usually no more than a few days).

**Other treatments for acute attacks**

Putting an ice pack on the affected area can reduce some of the swelling, heat and pain. They’re very safe, but make sure that you don’t put the pack directly onto your skin to avoid burning or irritating your skin. You can buy reusable cooling pads from sports shops and chemists, or you can use a pack of frozen peas, wrapped inside a damp towel. You should always use ice packs alongside any drug treatments your doctor has prescribed.

Resting the painful joint also takes some of the edge off severe pain. A cage over the affected foot or knee to take the weight of the bedclothes at night can help.

**Ongoing treatments to reduce urate**

The drugs given to ease an acute attack don’t get rid of the urate crystals in your joints or reduce the level of urate in your blood. Drugs are available that can lower urate levels and get rid of urate crystals in your body. Traditionally, the use of these urate-lowering therapies was reserved for people with more severe gout, for example if they were having frequent attacks, or if they had tophi, kidney stones, evident signs of joint damage or high urate levels.

Recent guidance advises that when people are first diagnosed with gout that an explanation about urate-lowering therapies is included in the information that they receive about gout. People with gout can then be involved in deciding whether or not they wish to go onto urate-lowering therapy at an earlier stage.

The reasoning for this line of thinking is that urate crystal deposits are very widespread even at the time of someone first experiencing an attack of gout. High urate levels can cause long-term joint damage and can also be bad for other aspects of your general health. The majority of people who suffer from gout will have multiple attacks and the condition is easier to treat the earlier you start.
You may still have acute attacks when you first begin urate-lowering treatment, so you may wish to take daily NSAIDs or colchicine to dampen down inflammation while your urate level is brought down. This tactic is called ‘prophylaxis’ (preventative treatment) of acute attacks. You’ll continue to be at risk of acute attacks for at least six months and probably longer, until all the crystals are dissolved away. It can take as long as two to three years to clear your body completely of urate crystals.

Urate-lowering drugs are usually very well tolerated, but you might have to stop using them if you have side-effects such as a rash or indigestion (dyspepsia). Aside from this, you should continue to take them until your doctor tells you to stop.

If you miss doses of your urate-lowering medication, especially in the early stages, this can cause your urate level to go up and down, which can trigger acute attacks.

It’s also important to consider other ways of reducing your urate levels. For example, it’s important:

• to lose weight if you’re overweight
• to avoid foods which are high in purines
• not to drink too much alcohol.

If you have other features of metabolic syndrome (high blood pressure, high lipids, diabetes), good control of these will also help to reduce your urate levels.

**Allopurinol**

Allopurinol is the most commonly used urate-lowering drug. It has been available for many years and is normally effective and very well tolerated if taken correctly. It works by reducing the amount of urate that your body makes and is usually taken once a day.

Your doctor will measure the level of urate in your blood and will probably start you on a dose of 100 mg a day. If your urate level hasn’t come down enough after a month, your dose will be increased by 100 mg. You may need several dose increases of 100 mg roughly each month until you’re at the right dose that keeps your blood urate level well below the saturation point. The maximum dose of allopurinol is 900 mg but most people reach the target urate level by taking a dose somewhere between 200–500 mg. Allopurinol is available as 100 mg and 300 mg tablets so you won’t need to take a lot of tablets if you need a higher dose.

Allopurinol is broken down and excreted through your kidneys, so if your kidney function is impaired you may be started on a lower dose (50 mg) and the dose increased more cautiously. Once your urate level is well below the saturation point (well under 360 μmol/L or 6 mg/dl), you should continue on that dose of allopurinol and have blood tests every 12 months or so to make sure that your urate level is within the desired range. The main reason not to start with a large dose is that lowering urate levels quickly can actually trigger an acute attack. This is
probably because the crystals in your cartilage become smaller as they start to break down, which allows them to shake loose more easily and shed into the joint cavity. Bringing urate levels down slowly by gradually increasing the dose of allopurinol is much less likely to trigger an acute attack.

Increasing the dose gradually is also less likely to result in side-effects such as a rash, headaches or nausea. If you do develop any side-effects soon after starting allopurinol, you should stop taking the tablets and see your doctor, who will advise whether you should restart the tablets and what special care you should take. Very rarely some people can develop a severe skin rash, fever and become very unwell (so-called allopurinol hypersensitivity syndrome).

If you’re not having any side-effects, it’s important to keep taking allopurinol. The most common reason for allopurinol not working is the patient not taking the drug regularly or at the correct dose.

Allopurinol can affect some other tablets, especially warfarin and azathioprine. If you have to take either of these drugs for any reason, you must tell the doctor who prescribes them that you’re also taking allopurinol. The dose of the other drug may need to be adjusted.

See Arthritis Research UK drug leaflets Allopurinol; Azathioprine.
Febuxostat
Febuxostat is a more recently introduced drug that also reduces the amount of urate made in the body. However, unlike allopurinol it’s broken down by your liver and is therefore particularly useful if you have kidney problems and can’t take a high enough dose of allopurinol.

Febuxostat comes in just two doses. The starting dose is 80 mg, which is quite strong and may trigger acute attacks, so it’s recommended that you take a daily NSAID or colchicine for at least six months to help protect against this. If your urate levels haven’t lowered after a month, the dose of febuxostat can be increased to a maximum of 120 mg daily.

There have been concerns about people with some heart conditions taking febuxostat. Current research suggests it’s probably safe for these people, but more research is being carried out. If you’re not sure whether you should be taking febuxostat, talk to your doctor.

See Arthritis Research UK drug leaflet Febuxostat.

Other urate-lowering drugs
Uricosuric drugs, which include sulfinpyrazone, benzbromarone and probenecid, work by flushing out more urate than normal through your kidneys. These drugs may not be suitable if you’ve had kidney stones or similar disorders. They’re not widely used in the UK, but they may be a useful alternative if allopurinol isn’t suitable for you.

Other drugs for treating acute attacks and for lowering urate are in development now and it’s likely that new drugs will become available in the future. If you’re unable to tolerate or be treated successfully with allopurinol, febuxostat or uricosuric drugs you may need to see a hospital specialist (rheumatologist) for further advice.

Treatment of joint damage
If gout has already caused joint damage, the treatment will be the same as for osteoarthritis, including:

- losing weight if you’re overweight
- daily exercise (both muscle-strengthening exercise and general aerobic exercise)
- reducing strain on the affected joint (for example, by pacing your activities and wearing the right footwear)
• painkillers (for example paracetamol, codeine)
• NSAIDs
• anti-inflammatory creams and gels
• topical capsaicin cream
• steroid injections into the painful joint
• surgery, including joint replacement.

See Arthritis Research UK booklets Osteoarthritis; Osteoarthritis of the knee.

Self-help and daily living

Healthy lifestyle choices
There are a number of changes you can make to your diet and lifestyle to help ease attacks of gout. The most useful things you can do are:
• losing weight if you’re overweight or obese
• reducing the foods you eat which are high in purines
• avoiding excess alcohol, especially beer and spirits
• avoiding dehydration by drinking plenty of water.

Weight loss
Losing weight sensibly and gradually, if you need to, is the most effective dietary treatment for gout because it can greatly reduce the urate levels in your body. The larger someone’s body is, the more urate is produced.

Weight loss should be gradual and combined with daily exercise. Extreme weight loss or starvation diets increase cell breakdown in your body, which can raise urate levels.

The best way to lose weight is to have a low-fat, balanced and nutritious diet and to exercise regularly, preferably daily. If you are new to exercise or haven’t exercised for a while, it might be a good idea to talk to your doctor before exercising. A sensible and sustainable approach is to start off an exercise regime with fairly short, but frequent workout sessions, and then to gradually build up the length and intensity of your exercise sessions. Aerobic exercise in which you get out of breath is particularly good for burning calories.

If you regularly burn off more calories than you consume on a daily basis you will lose weight. Determination and motivation are key factors in losing weight. It helps to find a sport or exercise you enjoy which you will keep doing. Some people find joining a leisure centre or sports club can help as meeting new people can be fun and motivational.

Lifestyle choices are not the main reason why most people get gout. However, someone who has a healthy lifestyle and also takes prescribed medication will give themselves the best chance of lowering urate levels and this will reduce the likelihood of having attacks of gout.

We don’t recommend Atkins-type weight-loss diets for people who are prone to
gout. These diets include a lot of meat and are therefore high in animal proteins, which are high in purines and which break down to produce urate.

**Fluids**

Drinking plenty of water may reduce your risk of an attack and of urate forming crystals in joint tissues. If you have kidney stones, you may need as much as 3.5 litres (6 pints) a day. Even if you don’t have kidney stones, you should aim for at least 1 litre (2 pints) of fluid a day.

You can include some other fluids besides water in this total but not beer or other alcoholic drinks. However, many soft drinks contain large amounts of sugar, in the form of fructose. Sugary fizzy drinks and fruit juices can be high in sugar and fructose content. Keep these to a minimum as fructose sugar is likely to increase the level of urate in your blood. Diet soft drinks don’t appear to increase the risk of gout.

There’s some research which suggests that drinking coffee regularly may help by increasing the amount of urate your kidneys get rid of. This doesn’t appear to be due to caffeine but to some other factor that we don’t know about yet.

Drinking a glass of skimmed milk every day may help to prevent attacks of gout.

**Alcohol**

Drinking too much alcohol, especially beer and spirits, may increase your urate levels and your chances of having a gout attack. A moderate intake of wine doesn’t appear to increase the risk. It’s important for many reasons to drink alcohol only in moderation, especially if you have gout. New Government guidelines state that men and women should not drink more than 14 units of alcohol a week.

Fourteen units is equivalent to six pints of beer which is 4% alcohol by volume (proof); or six 175 ml glasses of 13% proof wine.

The Government guidance warns against ‘saving units up’ to drink in one go, and strongly advises you to make sure you have alcohol free days every week.

Your doctor may suggest you drink well below these weekly limits if you have gout.

The charity Drinkaware and the NHS Choices website are reliable sources of information on alcohol. Remember that units are calculated from the strength of the drink as well as the quantity.

**Other diet tips**

Limiting your intake of foods that are particularly high in purines may be helpful, whether or not you need to lose weight. These include:

- **red meat and offal** – for example beef, kidneys, liver, sweetbreads
- **oily fish** – for example anchovies, fish roes, herring, mackerel, sardines
- **foods rich in yeast extracts** – for example Marmite, Bovril, Vegemite.

Aim to reduce the amount of protein you get from meat. Try replacing one portion of meat or fish a day with other sources of protein, such as beans, eggs, pulses or low-fat dairy products.
Vitamin C encourages the kidneys to get rid of more urate, so a diet rich in vitamin C may be helpful. This is another reason to make sure your diet includes plenty of fruit and vegetables. There’s some evidence that cherries may be particularly beneficial – either the fruit or the juice, fresh or preserved.

**Complementary medicine**
There’s little evidence for many of the other natural or herbal remedies and supplements available for gout. These include celery seeds, garlic, artichokes and saponins (natural compounds found in peas, beans and some other vegetables).

Generally speaking, complementary and alternative therapies are relatively well tolerated, but you should always discuss their use with your doctor before starting treatment. There are some risks associated with specific therapies.

In many cases the risks associated with complementary and alternative therapies are more to do with the therapist than the therapy. This is why it’s important to go to a legally registered therapist, or one who has a set ethical code and is fully insured.

If you decide to try therapies or supplements you should be critical of what they’re doing for you, and base your decision to continue on whether you notice any improvement.

**Research and new developments**
Ongoing research is greatly increasing our understanding of what triggers gout and how new therapies may be developed to treat it. A preliminary study funded by Arthritis Research UK confirmed that the majority of people with gout could be greatly helped and their quality of life improved by providing full information on gout and involving patients in decisions concerning their treatment package. This includes urate-lowering drugs, together with dietary and weight loss advice, rather than simply treating the symptoms of acute attacks. This has led to an ongoing full-scale clinical trial in general practice to find out the most effective package of treatment for people with gout.
Danny loves to stay fit and active. He surfs, swims, cycles, takes part in triathlons, and plays and coaches rugby. Despite all this physical activity and having a healthy diet and lifestyle, Danny has recently suffered with gout and has had to take a rest from sports.

As well as suffering excruciating pain, Danny has felt frustrated at how the condition is perceived by some people. Danny, 45, has suffered from gout previously, which he treated with non-steroidal anti-inflammatory drugs, but he hadn’t suffered with it for nearly three years.

Danny said: ‘I’ve had three weeks of real pain and it’s been horrible. ‘People don’t want to admit to having this disease as it’s seen as an ‘old man’s condition’ brought on by fine living and too much port and cheese. That misconception means younger people might not open up about their gout and seek help.’

The day before Danny’s attack he had played rugby and his family thought he might have picked up an injury in the game. The next day the pain was intense, and Danny knew it wasn’t a rugby injury.

‘I normally drink lots of water, but recently I hadn’t been drinking as much water as normal. I think that, as well as impact to my foot during the rugby game, may have been a trigger.’

There is a history of gout in Danny’s family, which may make him susceptible to the condition.

He said: ‘Gout can really make life difficult. Through my work I have to inspect building sites and when my gout was bad I wasn’t able to perform my duties on the sites.

‘My foot has really swollen up. There are some days when I find it difficult to get my shoe on and then some days when I have my shoe on, I struggle to take it off.’

Danny is due to see his doctor to review his medication and probably start on urate-lowering treatment. He added: ‘I feel positive about the future and hopeful about being able to stay on top of my gout’.
Glossary

**Aerobic exercise** – any exercise that increases your pulse rate and makes you a bit short of breath.

**Azathioprine** – a drug commonly used to help prevent rejection of transplanted organs. It works by suppressing the body’s immune system and is also used in rheumatoid arthritis to prevent the immune system attacking the joints.

**Cartilage** – a layer of tough, slippery tissue that covers the ends of the bones in a joint. It acts as a shock absorber and allows smooth movement between bones.

**Dehydration** – a condition where the normal water content of your body is reduced. The human body is about two-thirds water. The amount of water in the body only has to be reduced by a few per cent before the chemical balance in the body is affected. Dehydration can be caused by illness, exhaustion or not drinking enough fluids.

**Diabetes** – a medical condition that affects the body’s ability to use glucose (sugar) for energy. The body needs insulin, normally produced in the pancreas, in order to use glucose. In diabetes the body may produce no insulin or not enough insulin, or it may become resistant to insulin. When the body is unable to use glucose obtained from foods, the level of sugar in the blood increases. If untreated, raised blood sugar can cause a wide variety of symptoms.

**Inflammation** – a normal reaction to injury or infection of living tissues. The flow of blood increases, resulting in heat and redness in the affected tissues, and fluid and cells leak into the tissue, causing swelling.

**Menopause** – the time when menstruation ends, usually when a woman is in her 50s. This means the ovaries stop producing eggs every four weeks, and it’s no longer possible to have children. If this happens before the age of 45, it’s known as premature menopause.

**Non-steroidal anti-inflammatory drugs (NSAIDs)** – a large family of drugs prescribed for different kinds of arthritis that reduce inflammation and control pain, swelling and stiffness. Common examples include ibuprofen and naproxen.

**Osteoarthritis** – the most common form of arthritis (mainly affecting the joints in the fingers, knees, hips), causing cartilage thinning and bony overgrowths (osteophytes) and resulting in pain, swelling and stiffness.

**Proton pump inhibitor (PPI)** – a drug that acts on an enzyme in the cells of the stomach to reduce the secretion of gastric acid. They’re often prescribed along with non-steroidal anti-inflammatory drugs (NSAIDs) to reduce their side-effects.
**Purines** – nitrogen-containing compounds, found mostly in nucleic acids – DNA and RNA. The body breaks purines down to uric acid, which passes from the body mainly via the urine, but also via the bowel.

**Steroids** – drugs that have a very powerful effect on inflammation. They’re also known as corticosteroids and are similar to cortisone, which is produced naturally in the adrenal glands. Steroids can be taken as tablets or as injections – either into the joint itself or into the tissues around the joint.

**Synovial fluid** – the fluid produced within the joint capsule that helps to nourish the cartilage and lubricate the joint.

**Synovium** – the inner membrane of the joint capsule that produces synovial fluid.

**Urate** – a salt of uric acid, which forms as old cells are broken down and foods are digested within the body. It’s normally expelled in the urine but can sometimes build up and form sodium urate crystals that are deposited in and around the joints or under the skin.

**Warfarin** – a drug used to prevent blood clots from forming or growing larger. It works by ‘thinning’ the blood, making it less ‘sticky’ and reducing the blood’s ability to clot.

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**Where can I find out more?**

If you’ve found this information useful you might be interested in these other titles from our range:

**Conditions**
- *Calcium crystal diseases including acute CPP crystal arthritis (pseudogout) and acute calcific tendinitis*
- *Osteoarthritis*
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**Self-help and daily living**
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- *Allopurinol*
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You can download all of our booklets and leaflets from our website or order them by contacting:

**Arthritis Research UK**
Copeman House
St Mary’s Court
St Mary’s Gate, Chesterfield
Derbyshire S41 7TD
Phone: 0300 790 0400
[www.arthritisresearchuk.org](http://www.arthritisresearchuk.org)
Related organisations
The following organisations may be able to provide additional advice and information:

Arthritis Care
Floor 4, Linen Court
10 East Road
London N1 6AD
Phone: 0207 380 6500
Helpline: 0808 800 4050
Email: info@arthritiscare.org.uk
www.arthritiscare.org.uk

British Dietetic Association
5th Floor Charles House
148–49 Great Charles Street Queensway
Birmingham B3 3HT
Phone: 0121 200 8080
www.bda.uk.com

Drinkaware
Finsbury Circus (Salisbury House)
3rd Floor (Room 519)
London
EC2M 5QQ
Phone: 020 7766 9900
Confidential helpline: 0300 123 1110
Email: contact@drinkaware.co.uk
www.drinkaware.co.uk

NHS alcohol information website
www.nhs.uk/Livewell/alcohol/Pages/Alcoholhome.aspx

UK Gout Society
PO Box 90
Hindhead
GU27 9FW
Email: info@ukgoutsociety.org
www.ukgoutsociety.org

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We’re here to help

Arthritis Research UK is the charity leading the fight against arthritis.

We fund scientific and medical research into all types of arthritis and musculoskeletal conditions.

We’re working to take the pain away for sufferers with all forms of arthritis and helping people to remain active. We’ll do this by funding high-quality research, providing information and campaigning.

Everything we do is underpinned by research.

We publish over 60 information booklets which help people affected by arthritis to understand more about the condition, its treatment, therapies and how to help themselves.

We also produce a range of separate leaflets on many of the drugs used for arthritis and related conditions. We recommend that you read the relevant leaflet for more detailed information about your medication.

Please also let us know if you’d like to receive an email alert about our quarterly online magazine, Arthritis Today, which keeps you up to date with current research and education news, highlighting key projects that we’re funding and giving insight into the latest treatment and self-help available.

We often feature case studies and have regular columns for questions and answers, as well as readers’ hints and tips for managing arthritis.

Tell us what you think

Please send your views to: bookletfeedback@arthritisersearchuk.org
or write to us at: Arthritis Research UK, Copeman House, St Mary’s Court, St Mary’s Gate, Chesterfield, Derbyshire S41 7TD

A team of people contributed to this booklet. The original text was written by Dr Mike Snaith and revised by Prof. Mike Doherty, who have expertise in the subject. It was assessed at draft stage by consultant rheumatologist Dr Mohammed Akil and GPs Dr Lisa le Roux and Dr Julian Barnaby. An Arthritis Research UK editor revised the text to make it easy to read, and a non-medical panel, including interested societies, checked it for understanding. An Arthritis Research UK medical advisor, Dr Ben Thompson, is responsible for the content overall.
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- volunteering
- supporting our campaigns
- taking part in a fundraising event
- making a donation
- asking your company to support us
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To get more actively involved, please call us on 0300 790 0400, email us at enquiries@arthritisresearchuk.org or go to www.arthritisresearchuk.org