Sleeping with the Enemy

or

The Curses of Venus: Terrifying Tales of Sexually Transmitted Infections

by

Ellen Støkken Dahl

Translated by Lucy Moffatt

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Agent: Inga Semmingsen

inga.semmingsen@osloliteraryagency.no

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“Forty-eight days ago I graduated from the faculty with distinction;

but a distinction is one thing and a hernia is quite another”

Mikhail Bulgakov, *A Young Doctor’s Notebook*

“Swallow this.”

Evil dead II (Sam Raimi, 1987)

Chapter 1–Year of the Flood

A bit about gonorrhoea

> *"They ate, they drank, they married wives, they were given in marriage, until the day that Noah entered into the ark, and the flood came and destroyed them all."*
>
> Luke 17:27

This is where it happens.

My office is spacious with pale walls, but it contains so many things that it feels a bit cramped and chaotic. Over there in the corner is the massive leatherette gynaecologist's chair. Right beside it, along the short wall, is the examination table and, next to that, a trolley with an array of sterile instruments and equipment for taking samples.

Beneath the room's sole window, which is equipped with both blinds and curtains to ensure my patients' privacy, stands the desk with its prehistoric PC, which I switch on, and a printer that rarely works the way it's meant to. On the other side of the room is a movable screen and a sink with a mirror above it.

Elsewhere on the wall, a selection of anatomy posters reveal what lies beneath the skin of the human sex organs.

As I sip on my bitter coffee and review my patient list, I notice that the scrub top I picked out earlier today is covered in ink-stains from a pen that accidentally ended up in the laundry with it, but it's too late to change now. Instead, I do up the top button of my coat as I open the door and call in the first patient of the day.

"Come on in," I say, and Jørn sits down on the chair I point him towards.

Before I even have a chance to ask what I can help him with, he reveals that he has "a pretty huge problem *down there*" and is hoping it'll be possible to do something about it.

"What kind of problem?" I ask.

"I think it's easiest if I just show you," Jørn answers.

Jørn's a straight talker, I'll give him that. He stands up, thrusts a hand down his trousers and starts pulling out unfeasible quantities of tissues from the depths. It's an absurd moment because there seems to be no end to it. He produces long, interconnected strips of paper and small, crumpled clumps. He's clearly picked up the paper at different places, adding more as needed – like sandbags shoring up a dilapidated dam. Some of the paper has little red hearts printed on it. I picture him sitting on an embroidered chair at his Grandma's house and dashing off to the toilet in the hall every other minute, terrified that the dam is about to burst, unleashing a gruesome flood on the intricate floral pattern. Some of it looks like those serviettes made from recycled paper that get wrapped around hot disposable

cups with plastic lids, while the smallest, most repulsive clumps, must once have been snow-white Kleenex.

But regardless of their origin, every scrap of paper is filthy, soaked in a yucky, yellowish-green fluid.

"I see," I say. "And I agree – this is a *huge* problem."

"What's the matter with me?" Jørn asks. He stands hunched over the wastepaper bin, which is now full to overflowing with soiled paper. "And can you fix it?"

I feel a fizz of excitement but force myself to dial down the enthusiasm a notch or two. It doesn't do to be *too* gleefully eager when you're dealing with other people's unpleasant ailments. But I *am* eager, because I think I know what the problem is – and of course I can fix it.

Sexually transmitted infections, or STIs, are my favourite diseases. What with the discharge, the sores and the grim atmosphere that hovers over them, STIs are the medical world's equivalent of a spine-chiller. They scare us silly! Taking a deep dive into the facts about STIs – and especially their history – gives me a delicious, icy sensation of terror-infused pleasure similar to the thrill I get from reading a true crime or watching a good horror film. Part of me wants to look away, to shut my eyes, and yet I can't get enough of them. I just have to watch, I just have to read on. It's become an obsession.

It's great being a doctor when your patient has an STI because when you work with diseases that are *so* steeped in shame, you can achieve an awful lot with

very little. A good conversation or a bit of warmth and understanding are often just as important as the medicine. Patients with STIs frequently feel isolated and keep their suffering to themselves. Many are ashamed of how they acquired the infection and they're terrified of passing it on.

But STIs aren't about morality. Catching one says nothing about who you are, about whether you're a good or bad person. Catching an STI is a common consequence of sex – an activity that we humans are, after all, programmed to enjoy. Anyone can contract them and getting infected is often as much about good or bad luck as about the choices you make.

Another thing I like about this work from a purely medical point of view is that the patients almost always turn out fine. Nowadays, we have medicines that work. We can, for example, live good, normal lives with HIV, we can get rid of chlamydia, and I'm pretty sure Jørn will be absolutely fine.

"Don't be afraid," I say. "We'll work this out."

"You really think so?"

"Yes," I say. "It'll be okay."

Jørn wells up. "Thanks," he blurts out, meeting my gaze for the first time.

"Of course, I'll need to examine you and run a few tests before I can tell you anything else," I say. "But my guess is that you have gonorrhoea."

The truth is I'm more than ninety per cent sure that Jørn has gonorrhoea but I also firmly believe that the Hippocratic Oath should include a provision stipulating

that doctors should never make diagnoses based solely on stained tissues. That's why I ask Jørn to step behind the screen, take off his trousers and pants, and lie down on the examination table. Jørn obeys and spends a long time standing behind the screen. Several solid, sickening clumps of tissue drop into the bin, like pebbles in a pond.

A Flood of Semen

It's delightful when names make sense, and gonorrhoea is one of those diseases whose name describes the way it behaves in the human body. I learned this from a dictionary of medical etymology that I bought to make myself look clever when I first started studying medicine just over ten years ago.

The Ancient Greek word *Gonorrhoia* is a compound of *gónos* meaning "seed" or "semen" and the suffix "-rhoia", meaning something that "runs" or "flows" over. The word diarrhoea shares the same suffix – although in that case, as we all know, the substance that's overflowing is very different. When you put "gónos" and "rhoia" together, you get a word that means "a flood of semen" – and it really isn't a bad name at all: The fluid flowing freely in poor old Jørn's pants really *does* look like a flood of semen: it's liquid, and it's seeping out of his urethra. It isn't semen, though; it's discharge.

The Ancient Roman physician and philosopher Galen was the man who came up with this fitting name while working as a doctor almost two thousand years ago. He was the first person to apply the word gonorrhoea to discharge from the penis.

Since Galen used this particular word, it's easy to assume that he was observing the same disease we see today – that gonorrhoea is at least as old as its name. But while that's possible, it's difficult to say for sure. The Bible also contains descriptions of dripping urethra, but it's risky to use ancient texts as diagnostic tools. Two thousand years ago, Galen must have wrestled with *something* that caused men's penises to drip and seep, we do know that; what we can't know for sure is whether the discharge was caused by the disease we call gonorrhoea.

A contemporary colleague of Galen's, Aretaeus of Cappadoccia, described gonorrhoea as a continuous, non-painful flow of semen. The fluid was "thin, cold, colourless and unfruitful" – so pretty different from classic gonorrhoeal discharge, which is yellowish-green owing to the large amounts of pus. Women could also suffer from the same disease, he said, but "their semen is discharged with titillation of the parts, and with pleasure and an immodest desire for connection with men." Women may be sinful souls, but the last time I checked, sexual arousal was not synonymous with gonorrhoea.

Other diseases such as chlamydia, mycoplasma and trichomoniasis can also cause urethral discharge, but gonorrhoea is in a class of its own when it comes to dripping. In fact, gonorrhoea patients drip so copiously that one of the nicknames for the disease is "the drip". Still, in the absence of a time machine, we can't be certain which disease it actually was that the ancient texts were describing. And it all gets even more muddled when you bear in mind that doctors and scientists were

once convinced that several different venereal diseases – including gonorrhoea and syphilis – were simply different sets of symptoms for the same pestilence.

Scottish surgeon John Hunter, who was born in 1728, was so convinced that gonorrhoea and syphilis were one and the same disease that he staked both honour and life on proving it. He used a lancet to extract pus from the dripping penis of some poor wretch (think Jørn in 18th century clothing), then punctured *his own penis* with the same lancet. His aim was to show that people contracted syphilis after being given a nice little dose of the "flood of semen".

When Hunter later died of cardiovascular disease, many believed this proved that he had indeed contracted syphilis, because the disease can settle in the blood vessels and heart, silting up the circulatory system. The truth of the matter is that his cardiovascular problems were probably caused by other factors. They are after all and even in Hunter's day, only a tiny minority of cases were down to syphilis. Or he may have acquired syphilis via some other route. Perhaps he had used himself as a guinea pig on more than one occasion because there was something he was hoping to prove.

Nowadays we know that John Hunter was wrong. Gonorrhoea is not a subspecies of syphilis but an entirely separate disease that is caused by infection with *Neisseria gonorrhoea* bacteria, familiarly known as *gonococci.*

Taking Home the Applause

"You want me to lie down *there*?" Jørn asks. "On *that* table?"

“Yes,” I say.

“But the paper’s wrinkled,” Jørn says. “It looks like someone’s already been lying on it!”

And he’s right. Someone has lain there. The paper has been used: I just forgot to replace it.

“Ugh!” Jørn says. “Lying down in someone else’s...”

He still hasn’t taken off his boxers, which were once white but are white no more.

“Sorry,” I say.

He nods.

I remove the wrinkled paper and discover that the roll has run out. I have to leave the office to fetch a new one.

“Wait here,” I say, crossing my fingers that Jørn won’t make a break for it while I’m away. “I’ll be right back.”

As I wander down the corridor to the store room, stained Birkenstocks slapping stickily on the linoleum, I think about gonorrhoea’s many nicknames. The drip is one, as I said before. “The clap” is another. There’s some debate about where this name comes from, but the most credible theory is that it originated in France in the 1370s. *Les Clapiers* was the name of a Parisian district so packed with brothels that it was only too easy to take the applause home with you. Other theories are: a) that it derives from an old-fashioned treatment for gonorrhoea, which involved slapping

or clapping the penis hard and rhythmically; or b) that gonorrhoea caused a "clapping sensation" (whatever that might mean) in the urethra. A more common description of the sensation gonorrhoea sufferers experience when urinating is that it feels like peeing barbed wire.

Gonorrhoea is no rarity, but its incidence in Norway has fluctuated. It was common in the 1930s and 1940s, peaking at 11,195 cases in 1946 before access to antibiotics dammed up the drip. In the 1970s and early 1980s, a fresh wave of gonorrhoea swept the country, peaking at nearly 15,000 cases in 1975. Things got so bad that the Norwegian Directorate of Health ran a poster campaign in 1976 with the slogan "Thirty-six Norwegians will catch gonorrhoea tonight," to raise people's awareness of the crazy contagion rates and encourage them to be better at using condoms. By the end of the 1990s, the condition had become rare again, with fewer than two hundred cases that year. Now, unfortunately, cases are on the rise again, with 1,705 in 2019. But while this is a cause for concern, it's worth noting that, at just 4.6 new cases per night, the level is well below the figure seen in the 1970s.

Gonorrhoea is shockingly contagious. The risk of contracting the disease from a single bout of vaginal intercourse (for those who practice it) is seventy per cent for people with vaginas and thirty per cent for people with penises. For comparison, the risk of infection with chlamydia is ten to twenty per cent per sexual contact for both parties, while for HIV – a virus many are terribly afraid of – the risk is very much lower, at around 0.1 per cent. And gonococci aren't in the

least bit picky: they thrive in most of the body's mucous membranes and can cause infections in the rectum, the vagina, the throat, the urethra and the eye. Since vanishingly few people use condoms during oral sex and gonorrhoea less often produces symptoms when infection is via the throat, gonococci in the throat can serve as a silent reservoir of disease.

Gonococci infect through direct contact between mucous membranes, so it doesn't take much imagination to see how they might end up in a throat or an anus. But the disease can also be transmitted indirectly. In some studies, gonococci have been found in the anuses of men who haven't had anal intercourse. Infection via the eye can occur when a person gets gonococci on their fingers after touching forbidden, dripping things and then rubs their eyes. It's also possible for babies to become infected with gonorrhoea of the eye during birth, through direct contact with their mother's mucous membranes.

Unfortunately, gonococci have a particular talent for causing inflammation and eating their way through the cornea. Luckily, we see very few gonorrhoeal eye infections these days, but they were very common in the 19th century: a full ten per cent of European children were infected with gonorrhoea of the eye during birth, and in three per cent of those cases, this resulted in blindness.

The German gynaecologist and venereologist Carl Credé (who had a splendid white beard and is best known for Credé's manoeuvre – a method for removing the placenta from birthing women more rapidly) came up with a smart solution for gonorrhoeal blindness in 1879. He started dripping solutions containing silver

compounds into the eyes of all new-born children . Silver kills bacteria and had already long been used for medical purposes at this point. In 1882, the practice of dripping silver-based solutions into new-borns' eyes – known as Credé's prophylaxis – was introduced as a standard measure in Norwegian hospitals. Between 1958 and 1984, gonorrhoea was so common in Norway that this procedure was mandatory and carried out on all new-borns.

Probing Penises

When I totter back into my office carrying a heavy roll of paper over my shoulder, Jørn is still standing behind the screen in the corner, trousers down. I fasten the roll to the frame of the examination table and pull out a sheet of glossy, unused paper, which I lay out on the table.

"Now you can lie down," I say.

Jørn lies down on his back, eyes tight shut.

"It'll be just fine," I say. "But I'm going to need you to pull down your boxers too."

"Oh, okay" says Jørn, tugging them down his thighs. "Like that?"

"Great," I say.

I direct the glaring lamp at his noble parts and take a closer look at Jørn's huge problem. Out of his urethra seeps the same thick yellowish-green fluid that soiled all those tissues.

I put on some gloves (there are limits to my love for STIs) and examine his groin. Beneath the skin, I detect some hard, tender bumps – enlarged lymph nodes. The lymph nodes are the immune system's sentry posts. When the immune system is activated – as it most definitely is when a person has a powerful, pus-inducing infection of the urethra – the lymph nodes enlarge and grow tender.

The next thing I do is palpate Jørn's scrotum.

"One serious consequence of contracting an STI in the penis," I say, "is that the bacteria can climb further up into the body."

"Into the body?" Jørn asks.

"You really don't want that to happen," I say. "Take a look at this."

I point to the anatomy poster on the wall that shows a cross-section of a penis and the male pelvic organs. Jørn reluctantly follows my finger with his gaze.

"First they climb through the urethra and push their way through the prostate," I explain. "Then they come out into one of the two spermatic cords, which run in a loop through your pelvic area, and then they find their way to one of the epididymes."

The epididymes are two small organs that each lie on top of one of the testicles, like slugs on a stone.

"And what happens there?" asks Jørn.

"Once they're in there, they can cause an infection called epididymitis. If that happens, one side of the scrotum will swell up – it can get really enormous and it hurts if you squeeze it."

When I give Jørn's scrotum a gentle squeeze, I find that his testicles are smooth and normal in size; next, I check the epididymes for lumps and bumps, but everything seems to be in order.

"Painful?" I ask, just to be on the safe side.

Jørn shakes his head.

From the trolley behind me, I select a long, narrow metal instrument with a flattened tip – a spatula – and remove it from its packaging.

"What're you going to do with that thing?" Jørn asks.

"Take a sample of your discharge."

Jørn blenches.

"It won't hurt," I assure him, although I'm guessing Jørn won't believe me. Like many of the men I encounter, he's probably afraid that tests for STIs (which mostly involve peeing in a cup) will be invasive and painful procedures that involve inserting probes deep into his penis.

"I'm going to need a fresh sample," I say. "It won't go in very far. There's no need to be afraid."

"Okay," Jørn says, shutting his eyes. "Just do it. Get it over with."

I insert the end of the spatula just inside his urethral opening and collect a drop of discharge which I smear in a thin layer on a small glass slide. After that, I take two sample swabs with absorbent tips, pick up a little discharge on each of them and stick them into test tubes with screw tops.

"See?" I say. "That went brilliantly!"

Jørn doesn't say a word, but I assume he's relieved.

"You can get dressed again now," I say. "Then I'll go and see if I can't confirm what's the matter with you."

Naturally, I don't tell him that his fate *might* have been to have a probe inserted deep into his penis if the disease had run its course in an age without functioning antibiotics.

A bougie for use in the urethra is a long – slightly longer than a penis – thin instrument with a rounded, often spherical tip. The word bougie is French for candle, and is used because bougies look a little bit candles (or anything else that's long and thin for that matter) and because, over the ages, these instruments have not just been made from materials like wood, metal and plastic, but also from wax.

With its blunt head and narrow body, the instrument can be inserted into the urethra (or any other natural body opening) using firm movements and a good deal of pressure, without any great risk of cutting or punching out new and unwished-for openings. It follows the urethra's natural course until it comes to a halt. And when it does that, the doctor can – in the style of a plumber – blast their way onwards by removing the blockage and expanding narrow sections.

Gonorrhoea of the urethra unleashes a battle between the immune system and the aggressive gonococci. This battle is what causes the inflammation. The immune system's aim is to remove and destroy the invaders, but in the heat of battle, the body's own cells also come to harm. And damaged mucous membranes

can heal in new ways. The urethra may become narrower or – in a worst-case scenario – become blocked. And you don't need to be a doctor to see why that's an undesirable outcome.

Before antibiotics became available, many gonorrhoea patients suffered from blocked urethras. Clearing plugged pipes was a procedure that was carried out regularly at Oslo University Hospital, back in the days when the entire venereal diseases unit was wreathed in barbed wire. Sadly, I don't think the wire was intended to keep people out.

Bougies were also used not just to remedy the consequences of gonorrhoea, but to try and cure the disease itself. In an article from 1913, two surgeons describe a fancy new trick. It was already common knowledge that gonococci couldn't handle high temperatures: In laboratory experiments, they perished at forty degrees Celsius, but higher temperatures would apparently be even more efficacious – and confined areas of the body, such as the interior of the urethra, could cope with much higher temperatures than the body as a whole. In fact the surgeons were of the view that maintaining a temperature of forty-five degree in the urethra for more than six hours would be brilliantly effective and cause little damage. They placed one hollow silver bougie inside another; the outer bougie only had an aperture at one end, while the inner bougie had holes at both ends, so that water could flow through it freely, then run into the outer bougie. They heated the water to a few degrees above forty-five, to compensate for cooling during its passage through the apparatus, then they connected the two bougies to rubber tubes that would carry

the water both in and out, creating a steady flow and a constant temperature. After that they stuffed the bougie into the penis and left it there. Higher temperatures, the authors mention by way of warning to others keen to try this method, might cause severe burns in the patient's urethra.

It takes a lot make my jaw drop, but I feel a bit sick at the idea of heating up a urethra for six hours straight. And it's not just because it sounds hideously uncomfortable; there's also something about this gradual heating process that reminds me of cooking, of being slowly tenderized in a sous vide. After all, our flesh is no more resistant to heat treatment than that of pigs or cows.

Strong as Gonorrhoea

The best thing about gonorrhoea – even this horrible disease has a silver lining – is that I don't always have to wait for the results of tests I've run. Sometimes I can make the diagnosis myself.

Carrying the little glass slide containing Jørn's discharge, I cross the corridor and let myself into the lab, where the microscope is. I remove the protective fabric cover, switch it on and sit down on the stool in front of it. All the equipment I need to demonstrate the presence of gonococci is old hat. Albert Neisser, who discovered them (hence the name *Neisseria* gonorrhoea), examined discharge in almost precisely the same way as I'm about to back in 1878, when he discovered the bacteria in the pus and discharge from thirty-five men and women with typical STI symptoms.

The first thing I do is run the slide through a little flame. The aim is to dry the discharge and fix it to the glass without allowing the smear to be damaged by the flame. Then I open a little glass bottle with a dropper and cover the dried patch of discharge with a dye called methylene blue. I rinse off the excess dye in the sink, then dry the glass slide with a tissue, making sure I don't rub off the dried discharge. The blue dye makes it easier to see the microscopic world of the microbes.

Venereology, the study of sexually transmitted infections, is one of the fields where it pays to make regular use of the microscope, and I enjoy the practicality of it. Among others, it allows me to diagnose gonorrhoea and pelvic inflammations caused by other STIs, enabling me to distinguish them from yeast infections and bacterial vaginosis, aided by discoveries that were made several hundred years ago.

At low resolution the image looks chaotic – full of dots and patches in varying sizes and various shades of blue. But as I zoom in, I can easily distinguish between the different cells and bacteria. I see the skin cells from Jørn's urethra, which resemble fried eggs, and a carpet of white blood cells, which have turned up to deal with the ongoing infection. They are small and spherical, with a big multi-lobed nucleus. I'm not just interested in the white blood cells – a sure sign that Jørn's urethra is severely inflamed; if I'm lucky, I'll also be able to find the gonococci themselves.

Bacteria have markers that enable us to distinguish them from each other when we examine a specimen under a microscope. The *gonococcus* is a *coccus*, a

type of bacteria that is shaped like a sphere, unlike the *bacillus*, which is shaped like a rod. Gonococci are not perfectly round, but look a bit like coffee beans.

Another marker is the way they position themselves in relation to one another. Some bacteria arrange themselves in chaotic clumps, known as clusters, while others arrange themselves in rows or chains. For example, I can easily see the difference between streptococci – cocci in chains – and staphylococci – cocci in clusters.

Gonococci, however, are happiest in pairs; they are what is known as diplococci. The word *diplo* is Greek for double, and I find these pairs everywhere in Jørn's smear. I observe them lying tightly packed together, two by two, with the smooth curved end facing outwards and the flat end facing inwards. Some of them are still outside the white blood cells, but most of them are in the bellies of these cells, which have come along to eat them up. Unfortunately for Jørn, the white blood cells' strategy isn't wildly successful.

I respect gonococci. Perhaps it sounds odd to say I respect such a harmful bacteria, but since few other bacteria work harder, more intelligently and with greater determination than the gonococci, I feel they deserve it. But they scare me too. There's no doubt about it.

Whereas our bodies consist of millions of cells, bacteria are single-cell organisms. They are simple yet fully alive. Just like our own cells, just like all other life on earth, they contain genetic material which serves as the recipe to

produce precisely them. The genes are bathed in fluid and packed in a thin, moist cell membrane of fatty substances that holds everything together. I like to picture it as a tiny little malevolent water balloon, which is keen to settle in the urethras of unlucky people like Jørn.

The most incredible thing about gonococci as far as I'm concerned is located on the outer surface of the cell membrane. Gonococci are covered in hair-like outgrowths known as pili, which they use like grappling hooks. The gonococci throw out a long thin pilus to get a firm grasp on a mucous membrane we'd much rather they didn't attach themselves to, in the vagina or the urethra for example. Then they retract the pilus – a bit like the way a retractable vacuum cleaner lead is sucked back up when you press the right button. This pulls the gonococcus towards the membrane; it's like the world's most repulsive ski lift.

The gonococci's pili are fantastic (for them not us): their movement is driven by one of the world's most powerful biological motors, enabling the gonococcus to haul 100,000 times its own weight. For comparison, if I were capable of hauling 100,000 times *my* own weight, I'd be able to drag thirty-five blue whales around. And while I've learned that few things are impossible if only I work hard enough, I'm obliged to acknowledge that "strong as gonorrhoea" is a title that is far beyond my reach: I would barely be able to haul 0.016 per cent of the weight of just one blue whale.

In the interior of the vagina and the urethra, there's a constant flow of liquid, which might wash away the bacteria if it weren't for these pili. As it is, the

gonococci dig in their claws like leeches and can't be flushed out by either urine or discharge.

Once the gonococci have taken hold, they clamber through the thin outer layer of the mucous membrane and establish themselves deep inside. There, they divide over and over and over again, seemingly following the biblical injunction to go forth and multiply. The human body quickly realises that something's brewing and summons the white blood cells, which are experts at gobbling up and disarming bacteria.

When the immune system makes its entrance, that's normally bad news for our bacterial foes – but not for gonococci. They deal extraordinarily well with anything the human body can throw at them.

The white blood cells start off briskly, gobbling up the gonococci like candies. That's why you'll see masses of small coffee-bean-shaped diplococci inside the white blood cells when looking for gonorrhoea. But this feeding frenzy fails to halt the infection for two reasons: first, many of the gonococci manage to sneak off; the powerful grip of the pili makes it more difficult for the immune cells to eat them up. Secondly, many of the gonococci that are swallowed survive. Although the gluttonous immune cells have bacteria-killing proteins in their stomachs, the gonococci are able to cut them to pieces. And if any of the gonococci do get injured, they are geniuses at surviving through co-operation: they borrow genes from each other and repair their own damaged parts.

The gonococci that survive continue dividing inside the immune cells, ultimately becoming like Trojan horses. When, at last, the immune cell dies of exhaustion and the cell membrane dissolves, an ocean of healthy gonococci floods out, ready to wreak even *more* havoc.

Normally, the immune system becomes better and better at combating a disease over time because the white blood cells recognise bacteria they have met before and can go on the attack harder and stronger next time they cross paths. The problem with gonococci is that they're good at playing dress-up. They switch around the proteins that are on display, making it impossible for the immune system to recognise them from one time to the next.

"It *is* gonorrhoea," I say, back in the office with Jørn. "I've seen it."

I feel a bit like a mediaeval bard declaiming tales of the gruesome, deadly powerful and cunning gonococci – the bacteria that have transformed the inside of his urethra into a bloody, muddy battlefield; the bacteria that could have blinded him or blocked his urethra, but which I am now about to destroy single-handedly.

"What now?" Jørn asks. "How do I get rid of it?"

"You'll get treatment."

"What kind of treatment?" Jørn asks.

Here, I'm fortunate enough able to inform Jørn that he will be getting antibiotics, rather than having a bougie filled with hot water jammed up his urethra. Nor will

he have to endure any of the other peculiar treatments my long-deceased colleagues once provided.

In the 17th century, many physicians turned to herbal medicine. Intricate, almost witch-like recipes involving water lilies, strawberries, syrup of violets, dandelion, parsley fern and poppy seeds were to be taken "at every hour of the day and night."

If the magic brew failed to work, men could try to ease their afflictions by peeing with their penis dipped in a bucket of warm cow's milk. Towards the latter half of the 19th century, gonorrhoea was treated with cubeb, a type of Indonesian pepper, and copaiba, a balsam extracted from special South American trees. Copaiba – which we now use to manufacture varnish and biodiesel, among other products – was mixed with liquorice to mask the sharp taste.

If that didn't help either (and, I can reveal, it did so only rarely) the physicians' creativity ran riot as they tried everything from experimental surgery to penis baths that lasted days at a time.

But enough of that. I repeat: Unlike the poor wretches of times gone by, Jørn will get antibiotics.

"Antibiotics?" Jørn repeats, wrinkling his nose.

"Yes," I say. "Is something the matter?"

"Will that work?" Jørn asks.

"Yes," I reply.

"Are you sure?"

Modern patient that he is, Jørn has used the time I spent on lab work to do a spot of googling. He's read several articles about so-called "super gonorrhoea," a multi-resistant bacteria that can cope with all and any of the antibiotics we use to try and kill it. And now he – like me, I confess – is pretty frightened. He asks if there's any chance that he'll never be well again.

"I'm feeling a bit stressed here," he says.

"But there's no need," I say. "It'll be fine.

We haven't yet seen any cases of super gonorrhoea in Norway, but there is definitely cause to fear for the gonorrhoea patients of the future.

Since we first started treating them with antibiotics, those tough, hard-working gonococci have managed to achieve resistance to no fewer than six different antibiotic classes, one after the other. Unlike syphilis bacteria, which we have treated with the same medicine for many decades, gonococci adapt so well and so rapidly that it's a struggle for us to keep up. One of the samples I took from Jørn's discharge is a culture sample that can be used to check whether Jørn's particular bacteria are antibiotic-resistant and, if so, *which* types of antibiotics they are resistant to, so that his treatment can be tailor-made if necessary.

I hope and believe that the standard gonorrhoea treatment Jørn will get today will work. But one day – a day that is uncomfortably close at hand – I will no longer be able to answer anxious patients with the same calm reassurance as today. Antibiotic resistance is a long-predicted health crisis, and one of the greatest

challenges of our age. In order to slow its development, we must be sparing; we must handle antibiotics with common sense and respect. Because if super gonorrhoea spreads, if these bacteria become commonplace, we may well see the return of bougies and buckets of warm cow's milk.

Soon I'll be blending antibiotic powder with liquid anaesthetic then slowly injecting the mixture into Jørn's buttock muscle. In a few days, I hope, test results on the samples I took will confirm that Jørn has gonorrhoea and that his gonococci are among the treatable types – and that will be the end of Jørn's dripping nightmare.

For now, at any rate. But there's a chance he may catch it again – and that's why we health workers also have to put in some "preventative work" in the form of clear, clinical communication.

Taking a deep breath, I say in a grave voice: "You do know it makes sense to use condoms, don't you, Jørn?"

When he looks down and doesn't answer, I launch into an explanation of how condoms – which were once made from pig gut or sheep bladder and fastened around the middle with a ribbon (Jørn's cheeks take on a greenish tinge at this point) – actually protect against gonorrhoea.

"Gonorrhoea is transmitted through contact between mucous membranes," I say, "and the condom works like a barrier! The bacteria's grappling hooks can't get a grip."

“I know how to use condoms,” Jørn says irritably.

“Would you like to take some with you when you leave?” I ask, pointing to a brimming jar on my desk.

Jørn takes a few before saying that he knows it makes sense to use them. “I do *try* to be good,” he says.

“I’m sure you probably do,” I say. “But you have to remember that gonorrhoea is highly contagious.”

“I can tell you one thing for sure,” Jørn says. “I’m definitely going to get better at using them now because there’s no way I want to go through all this again.”

“We’ll see,” I say as I show him out of the office. “We’re here if you need us.”

Chapter 2 – A Sore Point

A Bit about Herpes

"O'er ladies' lips, who straight on kisses dream,

Which oft the angry Mab with blisters plagues,

Because their breaths with sweetmeats tainted are."

Shakespeare, Romeo and Juliet

Helene's genitals have been really painful for the past few days. Now, as she sits leaning well back in the gynaecologist's chair, the cause is clear. Her genitals are red and swollen, and her skin and mucous membranes are scattered with small clusters of tense, angry blisters, and sharply defined sores.

I touch her outer labia gently with a gloved finger, move them apart and see that there are more sores in the vestibule, the area between the inner labia where the urethral opening and vagina are located. The sores are weeping, and the fluid is mixed with discharge, which is thin, pale yellow and streaked with blood.

Helene starts in pain when I accidentally brush against one of the sores.

"That hurt," she says.

"I know," I say. "Sorry."

"No problem."

I take out a swab with an absorbent tip and say I'm going to take a sample.

“This will feel a bit uncomfortable,” I add, before gently running the swab over the blisters and sores, absorbing some of the fluid seeping out of them. A few blisters burst and Helene whimpers. This disease really can be hellishly painful.

“I’m pretty sure it’s a herpes outbreak,” I say, as I pop the swab into a little screw-top test-tube, cover up Helene’s genitals with a towel and lower the gynaecologist’s chair so she can slide down off it and onto the floor.

Helene rummages around behind the screen and says nothing as I wash my hands. But when she emerges and sits beside my desk, she looks as if someone has died.

“I expect you’ve heard of herpes, haven’t you?” I ask.

“Yes,” she replies.

I tell her what it is anyway: a disease that causes a rash of blisters on the skin and mucous membranes. The blisters burst and become sores, then dry out and form little scabs which eventually fall off. Herpes is caused by a viral infection. There are two slightly different variants, both of which cause the disease, known as herpes simplex virus Types I and II. When I examine a patient with genital herpes, as now, I can’t tell them immediately which virus they’ve been infected with – only the test can reveal that that – but I can tell them that they are not alone in having this infection.

“Herpes is really common,” I say. “More than half of us – maybe as many as 80 per cent – pick up a herpes Type I infection on our mouths in childhood, and

almost one in three young adults are infected with herpes Type II, on the genitalia."

Type I has become a more common cause of genital herpes in recent years, probably because of oral sex, and this type may cause a slightly milder version of the disease, with fewer outbreaks, whereas Type II rarely causes oral herpes. That said, both herpes types can cause infections in totally different areas of the body.

"Some really unlucky people get herpes of the eye," I say. "Or of the finger – typically if they're both unlucky and dentists."

"Why on earth would I care about dentists' fingers?" Helen interrupts me.

I'm about to say that she's quite right, I ought to stick to the point, when she abruptly bursts into tears. I push a box of tissues towards her and Helen helps herself to three.

"I can't have herpes," she sniffles.

"Why not?" I ask

"No one I've slept with has it."

"How do you know?"

"Because," Helene says, waving at her groin, "I'd have seen it!"

"No," I say. "You wouldn't."

One dark and stormy night just over a week ago, a loved-up couple – Helene and a handsome lawyer we'll call Mohammad – were out on their third date.

If you met Mohammad on the street and asked him if he had herpes or accused him of passing it on to Helene, he'd deny it – and believe he was telling the truth. Mohammad can't remember ever having had blisters on his skin and he certainly didn't have them when he was out with Helene. Unfortunately, though, that doesn't mean he doesn't have herpes.

"One of the odd things about herpes," I say, "is that most of the people who have it don't know they do."

While as many as one in four Americans have herpes Type II antibodies, which means that they have been infected and are carriers of the virus, only one in forty have actual outbreaks involving blisters.

"And the trouble with that," I continue, "is that people can also pass it on without realising."

When Helene and Mohammad slept together, the herpes virus climbed off the unwitting Mohammad's skin and onto Helene's. The friction between their bodies caused little tears in Helene's mucous membranes to form, and that made it easier for the herpes viruses to find their way in.

It took a few days for Helene to notice anything. To start off with, there was a prickling sensation in her thighs, as if someone was tickling her, and then it developed into an itchy discomfort. As her skin cells were destroyed by the herpes virus, the first blisters formed.

Always and Forever

Although the herpes viruses initially cause skin problems, it's the nervous system that ends up being their new forever home. The word herpes comes from Ancient Greek and means "to creep", perhaps because of the prickling sensation on the skin before an outbreak. But the name also describes one of the virus's crucial traits.

"When people get infected, the herpes viruses seek out the nerve endings in the skin," I tell Helene. "Then they creep from the skin further into the body, using the nerves as rope ladders. In the end, they reach the neuron cell bodies that lie clustered together just outside the spinal cord, in what's known as a ganglion, and this is where they settle down. If the person contracts a genital infection, the sacral ganglion in the pelvic region is the one that gets a visit. If they're infected in the mouth, the same thing happens to the throat ganglion. The herpes cells deactivate the host cell's ability to die so they can stay there forever."

"Surely not forever?" Helene protests.

"Yep," I say. "Herpes follows you to the grave."

"But I can't stay like this for the rest of my life!"

"Don't worry, you won't."

As long as the viruses are chilling out in the neurons, they don't cause any problems – and they aren't contagious either. But now and then, they wake up and

decide to go on a jaunt. Climbing along the same nerves they used to clamber inside, they creep out onto the surface of the skin.

"Once they're out on the skin, they can cause a new outbreak of blisters and sores in the same place where the person was infected, or they can be passed on to others."

"But this hurts so badly," Helene says. "Am I going to get loads of these outbreaks?"

"It varies," I say. "But what you're suffering now, your first outbreak, will be the worst. It can hurt like hell and last for weeks on end. Some people also experience fever and muscle pain – the kind you get with flu or other viral infections – and end up getting pretty ill. And some have such sore genitals that they have trouble peeing."

"You've got to be kidding me," Helene says.

"No", I say. "But I'll give you some medicine: pills containing antiviral substances that will shorten the duration of the outbreak and ease the pain."

"But they don't get rid of the disease?"

"No. You'll still have herpes," I say. "And now and then you may have a fresh outbreak. Although they may be painful and annoying, they usually get milder and less frequent as time goes by. A lot of people never suffer any further outbreaks at all and others only feel a slight itching when they do happen."

I tell her that herpes outbreaks often happen when people are ill, which is why oral herpes is often called a cold sore. Other common triggers are damage or friction to the skin, menstruation or sunbathing."

Helene, who hasn't answered, takes another three tissues out of the box and blows her nose.

"The important thing now," I say, "is for you to avoid infecting yourself."

"What do you mean?" Helene asks.

"Don't touch your genitals and then rub your eye – because you may infect yourself with the virus. Wash your hands."

"Oh my God," she says. "Am I going to have to worry about infecting my own eye for the rest of my life?"

"No, no," I say. "You can only infect yourself right now."

The herpes virus works a bit like a vaccine against itself. When people get infected – in the mouth, say – antibodies are created that make it impossible for them to be infected with the same virus again on a different part of the body – the genitals, for example.

"But during the first outbreak," I say, "the body hasn't yet managed to produce those antibodies. So all your mucous membranes are fair game. Later you'll be protected. Unless you contract the other virus type. You can catch both, although luckily there's a certain amount of cross-protection. Being infected with one of the viruses mostly protects you against serious outbreaks of the other."

Helene looks at me with tears in her eyes.

“This will get better quickly once you’ve started taking the tablets,” I say. “I’ll write you out a prescription for some analgesic gel that you can try out too – and your skin will heal nicely without any scarring. Is there anything else you’d like to ask about?”

Helene doesn’t answer.

“It’ll be just fine,” I say, as I fiddle around with my PC, preparing to write out the prescription.

“No it won’t!” Helene bursts out.

“What’s bothering you?” I ask.

“You said I can pass it on without realising.”

“Yes, I did – among other things.”

“So I can never have sex again,” Helene says. “Ever!”

“I never said that!”

“But if I have these viruses in my body forever and don’t know when I’m contagious,” Helene says, “that means I can infect anyone anytime.”

“You’re most contagious during visible outbreaks, but you may be contagious anytime,” I confirm.

According to one study, people with herpes have the virus on their skin around 18 per cent of the time, often without realising it. The risk of contagion per sexual contact is between three and four per cent.

“But that means I can never sleep with anyone again. Don’t you see? This is herpes – it’s absolutely disgusting. I can’t go around passing it on to people.

Old Disease, New Shame

When I work with and write about sexually transmitted infections, I have an agenda, an aim. My aim is not to make people like discharge and blisters; it is to encourage us, as a society, to take a slightly less melodramatic view of these diseases.

No one is particularly keen on stinky respiratory secretions or the substance that emerges from a diarrhoea patient's backside. Crap and phlegm are both pretty revolting, but few people with diarrhoea or pneumonia think there's something intrinsically wrong with them, or believe they should be ashamed of the illness and how they caught it. No one keeps pneumonia or stomach upsets secret; instead, these ailments are treated as practical, medical problems. The patients and their doctors take simple measures to ensure that they recover and protect others from contagion. As a result, the issue causes no additional anxiety or emotional stress.
I wish we took the same view of STIs, but unfortunately, as soon as genitals and sexuality are in the picture, shame comes sneaking in too.

To my mind, it would make more sense if the shame we felt about the different STIs was proportionate to the seriousness of the disease – if we were less ashamed about the common diseases. But attitudes to herpes confirm that there's no logic to shame.
Herpes is incredibly common – in fact it's more common to be infected with a herpes simplex virus than not. The disease is rarely much of a problem: most

people don’t even know they have it, and most of those that do experience only mild symptoms. It is almost never dangerous. The absolute worst that can happen is that the herpes simplex virus can, in vanishingly rare cases, cause inflammation of the brain in people with oral infections; or it can cause severe illness in babies who are infected by their mothers during birth. In practice, though, this isn’t something a person with a herpes infection needs to worry about. Yet some patients leave my office in a state of panic, anger and grief when I give them the diagnosis.

Oddly enough, perhaps, herpes is one of the diseases we are most ashamed of, and of course Helene has picked up on this shame. Herpes is a dirty word. Herpes is the monster under the bed. I’ve seen those of us who are disgusting enough to have herpes or nasty enough to pass it on subjected to death threats or urged to kill themselves on internet forums and in comments sections.

One of the strangest things about this herpes shame is that it’s such a new emotion. Herpes has been with us for a long time. To give you an example: the Roman emperor Tiberius – who ruled around two thousand years ago – is said to have forbidden kissing for a period because he got so sick of seeing blisters on people’s lips. Up until a certain point in history, we certainly managed to view herpes as a common, practical problem: the blisters and sores are no fun and we’d much rather not have them, but in our era, shame about the blisters has become a bigger problem than the blisters themselves. Some people believe their sex life, indeed their entire life, is over. It’s the end of the world.

How did we get here?

The shame kicked in properly in the 1960s, when new laboratory techniques enabled scientists to differentiate between different herpes viruses. Even though both herpes simplex variants cause herpes – blisters and sores on the skin – we started talking about them as if they were very different diseases. Type II became associated with genital infection and with that, herpes the STI (which had actually been there all the time) was born. In public discourse, a distinction was drawn between the terrible STI and the innocent cold sore. And that split was also accompanied by emotions.

In a Slate article titled “How Herpes Became a Sexual Boogeyman”, we can read how conservative forces – aided by sensationalistic media coverage – transformed an innocent blister virus into “the new sexual leprosy”, as a Time Magazine cover put it in 1980.

The news stories about herpes were a powerful shot across the bows for sexual freedom. Penicillin had dealt with syphilis, and various medicines were being used to cure gonorrhoea. Although we now know that HIV was smouldering away in the not-too-distant future (the American HIV epidemic started in 1981), no one had heard of HIV or AIDS back then. The risk of acquiring a serious disease from sex was lower than ever before, not to mention that the Pill had come on the market, giving women better control of their own fertility at last.

Some people got scared. Would there be no end to the general public's promiscuity? Should people be allowed to do what they wanted with their bodies, without any fear for the consequences?

The new (ancient) herpes suddenly became seen as the defender of chastity. American pastor Billy Graham declared: "We have the Pill. We have conquered VD with penicillin. But then along comes Herpes Simplex II. Nature itself lashes back when we go against God."

In 1973, a Time Magazine article wrote, "Unlike the basic herpes simplex, which strikes indiscriminately, Type II appears to exercise moral judgment—tending to afflict primarily the sexually promiscuous

This notion, the idea that genital herpes was linked to sexual promiscuity, took root in the popular imagination, where it remains to this day. Regardless of whether they have Type I or Type II, people with genital herpes feel guilty, while people with oral herpes get away with simply having a cold sore. People like Helene blame themselves and are ashamed of what they've done; of how irresponsible they've been. And they become terrified of passing it on.

And the thing about shame is that it's immune to facts about the disease. Doctors and health professionals have been repeating the same thing since well before the fear of herpes took root during the 1970s and 1980s, and we continue to repeat it today: Herpes is not the end of the world.

But it doesn't work. People don't listen to us.

Herpes Isn't the End of the World

"What makes you think you can never sleep with anyone again?" I ask. "Think of all the people who already have herpes – do you suppose all of them have given up sex?"

"But they don't know they have it, do they?" Helene says.

"A lot do, a lot don't," I say. "And since it's so common to be infected with one of the viruses, it's pretty likely that any person or people you choose to sleep with in future will already be infected and protected, right?"

"But I can't know that," Helene says.

"No," I say. "Like so many other things in life, this isn't something you can know or control absolutely. Condoms go a long way towards protecting against infection. They don't cover all the skin of course, but it's better to use them than not."

"What are you actually trying to say?" Helene asks. "Is the moral of the story here that I can sleep with whoever I want to, despite knowing that I might infect them with herpes? That doesn't sound right to me."

"I don't know what the moral is," I say. "This isn't a fable, it's your life. And the situation you're in is one you have in common with an awful lot of other people. I'm trying to give you the best possible information so that you can make the choices that feel right for you. On the one hand, herpes is common, it's mostly harmless, it's a disease lots of people have anyway, and most of the people who catch it don't notice a thing. I think those are pretty reassuring facts to know. On the other hand, as you're experiencing now, people can end up with irritating

blisters and sores, either once or several times. Not to mention all the negative emotions you're burdened with."

"I don't know what I'm going to do," Helene says. "Do I have to tell people before I go on another date? I don't know what I'd say."

"With some STIs, like chlamydia and gonorrhoea, you have an obligation to tell your partners. Cases have to be tracked and traced so that everyone who needs treatment gets it. But there's no such obligation with herpes. You get to decide what to say and who to say it to. Lots of people choose to talk to others about it, so they can work out how to deal with the risk together. Others choose to avoid sex during outbreaks and use condoms. And others again hope for the best and carry on as usual."

"But I feel so disgusting," Helene says. "This is the absolute worst."

"I know it isn't easy," I say. "But it's not a huge or dangerous or unusual thing. You just need to give yourself a bit of time to digest it."

Helene has started crying again and she hasn't yet received a prescription, an information leaflet or a farewell smile and a firm, supportive handshake. We aren't even close to finishing this consultation, which should have ended on a positive note several minutes ago. And I'm getting frustrated.

"This is all part of the game if you're going to have sex," I say. "It's a risk everyone's aware of."

But I can’t get through to her. She doesn’t even calm down when I tell her that herpes shame was thoroughly overblown in the 1960s by conservative forces eager to rein in sexual freedoms.

“It doesn’t matter where the shame comes from,” Helene says. “Only that it’s there.”

Sadly, it is undoubtedly there, and it affects the way we speak and think about the disease.

As if on autopilot, Helene grabs some fresh tissues from the box and wipes away her tears.

I can choose to stub this entire consultation out like a cigarette smoked down to the filter, and light a new and more successful spark with another patient instead. I can stamp the prescription and make Helene get up out of her chair and leave. I know, of course, that she’ll be fine in the end, but even so, that’s not what I do. I so want her to listen to me. To stop crying. So I start to improvise, to babble. I search for the right thing to say, the key that’ll fit the lock.

Herpes is pretty exciting,” I say. “Some scientists are using the herpes virus to attack deadly brain cancer.”

Helene keeps quiet.

“And the sub-variant of the herpes virus we contract may be able to indicate where in the world we’ve lived and travelled.”

No response.

“Okay,” I say and try again. “It isn’t just more common to have herpes than not. We humans have also lived with it for an awfully long time.”

Helen continues to dab at the skin beneath her eyes with her tissue, as close to her lower eyelashes as she possibly can. She catches the black tears before they get a chance to trickle down her cheeks.

“You said,” she says, “that we’ve had herpes for ages, but we only started being ashamed of it fifty years ago. But what good is that to me?”

“Can’t you guess?” I ask.

“Guess what?”

“Can’t you guess how long we – humans I mean – have had herpes? It’s a really long time. Don’t you want to give it a shot?”

Helene gazes at me, eyebrows raised.

“Go on, just guess,” I insist. “How long do you think we’ve been struggling with these rotten blisters and sores?”

“I don’t know,” Helene says. She’s stopped crying and now her eyebrows are leaning in towards each other. “How long have we had it?”

“Guess!”

“Okay,” says Helene. “Did we have it in the Middle Ages?”

“Further back!”

“In Ancient Greece and Rome, then?” Helene says. “The Stone Age?”

“You’re way off the mark.”

Helene shrugs. “Go on, then, tell me.”

“We don’t know which virus you’ve got yet,” I say. “Your test needs to be analysed first. But if you’ve got Type I, you’ve acquired a virus that’s been following us for six million years. And if it’s Type II, then it’s a virus that’s followed us for 1.6 million years. We had herpes before we were even human beings at all.”

“Seriously?” Helene says.

But How Do We Know How Old the Herpes Viruses Are?

In 2006, a group of scientists discovered that humans aren’t the only animal species to be plagued by blisters and sores. First, a herpes virus was found among our closest relatives, the chimpanzees, and later among a whole host of other primates. Every primate has its own virus, which is slightly different from the rest. But we humans are – as far as we know – the only primates to be blessed with two. Scientists who are trying to form a hypothesis about how and when a particular species originated start off by drawing a family tree, or a phylogenetic tree to use the posher term. They take a group of species that exists today: primates, say, birds in the sparrow family, flowers, coral reefs or – as in this case – different variants of the herpes simplex virus, and imagine that they once had a shared ancestor.

They draw the tree by studying the genetic material, the DNA, of all the different herpes simplex viruses, then mapping the similarities and differences between them. Differences in their genetic material tell scientists something about how

many mutations the species have undergone since they branched off from one another on the family tree. The more similar the genetic material is, the less time has passed since they split apart from each other. Armed with this information, the scientists can sketch out a hypothetical family tree showing the evolution of herpes, from the original common herpes ancestor to today's diverse selection of herpes types.

The two herpes simplex viruses that affect humans have many shared genes. But when scientists compared the two with the chimpanzee variant, they were in for a surprise. Oddly enough, Type II herpes simplex was more similar to the chimpanzee virus than our Type I, which means that Type II is likely to be a closer relative of the chimpanzee virus than of the other human virus.

We can therefore assume that Type I split off from the chimpanzee virus long before Type II did, simply by looking at differences and similarities in the two viruses' genetic codes. But in order to find out how long ago the virus types parted ways, scientists must use what's called a molecular clock. A traditional molecular clock is a molecule that mutates at a constant rate. It may be a bit of genetic material or a protein. By counting the number of differences in the chosen molecule in two different species, scientists can gain some insight into how much time has passed since the two species split away from each other.

Haemoglobin, the protein that carries oxygen in red blood cells, has been used as a molecular clock since 1962. By counting differences in the haemoglobin of, say, mice and men, scientists were able to say something about how long it was since

we split away from mice on the tree of life, since the time between each mutation is roughly constant. Nowadays it's commoner to use a computer algorithm as a molecular clock – as the scientists studying the evolution of herpes did.

After drawing the family tree, counting differences and calculating how much time has passed between the different points of divergence in the tree, scientists need a way to make the time absolute. We have the number of years, but we want to know the specific dates. When did the divergences happen? If one or more divergence points on the tree can be dated, it's easy to find all of them, since the molecular clock has given us the temporal distance between the splits.

In order to calibrate the tree like this, we have to start out from something we already know. Fossils are often used for calibration because they can give us information about the age of species for which we have them. There are no virus fossils, but in the case of the herpes simplex viruses, we can use the fossils of early humans and other anthropoid apes.

The original virus has since branched out in different species along with its hosts, and the hosts – humans and apes – also have a common ancestor. Fossils of early humans and early chimpanzees, and previous hypotheses about when the various primates diverged on the tree of life provide a basis for calculating how long the various herpes viruses have co-evolved with their hosts.

The American scientists proposed a hypothesis that herpes Type I has been with us since our branch of the family tree split away from that of the chimpanzee, six million years ago. At that point, our common ancestor was infected with a virus

that has since mutated and co-evolved with its hosts, into the chimpanzee virus and the human virus, respectively.

In the case of herpes Type II, however, they believe that our forefathers became infected after close contact with the chimpanzees' ancestors around 1.6 million years ago – long after we had parted ways on the tree of life.

*

"Shit!" says Helene. "Have we had herpes that long?"

"Yes," I say. "So you're not just part of a huge community now, you always have been. Herpes is older than us, probably even older than the emotion of shame."

"How weird," Helene says.

"Our ancestors lived with sores and blisters for millions of years," I say. "And I think it's a bit much to ask you to bear the burden of all that accumulated shame, don't you?"

Helene gets up, picks her jacket off the back of her chair and I send her on her way with a prescription and a gentle handshake.

"It'll be fine," I say one last time.

I hope she believes me.

Chapter 3 - Figs and Cauliflowers

A Bit about Genital Warts

"In order to buy some slave boys

Labenius sold his garden

But now he has only

An orchard of figs."

Martial, Epigrams vii, 71

"Hairy legs and forearms

May suggest machismo,

But the doctor smiles as he removes

The figs from your smooth anus."

Juvenal, Satires ii, 11

When I was a medical student, I learned that genital warts typically look like cauliflowers and that's not a bad comparison. It's the surface structure – scattered with little dots, like tiny wee cauliflower florets squeezed up close together – that does it.

Gunnar, who has accepted my apology for the late start to our appointment, is sitting in the gynaecologist's chair, butt bared beneath the spotlight. Great clusters

of cauliflowers sprout around his anus like some ghastly fairy ring in a patch of moss.

“It’s itchy as hell,” he says.

“I bet it is,” I say.

Genital warts or condyloma are harmless but irritating. They can itch and burn and get in the way.

“Don’t they look awful?” asks Gunnar as I paint the warts with a thin layer of acetic acid.

I shrug and say, truthfully, that I’ve seen worse.

After a few seconds, the warts whiten. That’s the effect of the acetic acid. Now that they’re paler, they look even more like cauliflowers than when they were pink and shiny. This trick makes it easier to find all the warts and distinguish them from skin bumps and skin tags.

While it may seem pretty tasteless to compare genital warts to food, it’s hardly a new practice. In around year zero of the common era, people used to call genital warts figs, fici, and they even made their way into contemporary satirical literature, like the poems of Martial and Juvenal, who wrote of fig orchards and smiling doctors picking figs off some poor wretch’s smooth backside.

Both hint at a link between homosexuality and genital warts, in a way that can be read as slightly tongue-in-cheek bullying. But according to historian J.D. Oriel, there was little sympathy on offer for gay men in Ancient Rome. At Roman clinics, the surgeons tended to spare neither “iron or fire” in their treatment of genital

warts, and were apparently unmoved by compassion for their screaming patients, who had only themselves and their "unnatural behaviour" to blame for their plight. Although having something inserted into one's anus makes it easier to become infected with condyloma of the anus, anal sex is not a prerequisite for catching anal warts. And anyone, absolutely anyone, can catch genital warts and other STIs regardless of their sexual orientation. Luckily for Gunnar, my toolkit contains neither iron nor fire. What's more, I certainly don't consider Gunnar to be engaging in "unnatural behaviour," even though he has already told me that he has sex exclusively with men.

Perhaps we ought to be able to take it as read that doctors in the 2020s will be more open and positively disposed to gay people than their Ancient Roman counterparts; unfortunately, though, nothing can be taken for granted. Some doctors still expect to meet only cishet heterosexuals in their consulting rooms. Such assumptions (and, where it exists, hostility) limit the health options available to queer people.

The sexual health of women who have sex with women, and therefore don't need contraception to protect against pregnancy, has almost been forgotten by the health system, because many people assume that there has to be a penis in the picture for there to be any risk whatsoever.

Men who have sex with men but don't dare speak about it openly to health professionals when they seek testing for STIs can also miss out on vital health services. Statistically speaking, men who have sex with men are at greater risk of

contracting rarer STIs such as HIV, syphilis and gonorrhoea (although these have gradually become more common among the heterosexual population too); consequently, the threshold for testing should be lower in their case. And health professionals must bite the bullet and establish what kind of sex their patients have and who they have it with before running any tests. It won't help to test a urine sample if the patient has a gonorrhoeal infection in their throat or rectum. That's just the way it is.

On the other hand, the link between homosexuality and the risk of contracting STIs has contributed to increased stigmatisation and marginalisation of gay men. It's hardly controversial to say that HIV research and the development of lifesaving medicines would have happened more quickly if that terrible disease had largely struck heterosexuals when the wave of HIV hit the USA in the 1980s.

"What actually causes warts?" asks Gunnar.

"Infection," I say. "It's a virus."

Warts in humans are caused by HPV viruses. HPV stands for human papillomavirus – a family of more than a hundred viruses in all, two of which are associated with genital warts, while several others are associated with cancer. Like the herpes viruses, the HPV viruses exploit tears in the body's outer defences, the skin, to make their way further in. They insert their genes into the skin cells, causing them to divide rapidly and create cauliflowers – or figs, if you prefer.

As with herpes, not everyone who contracts the HPV virus ends up with visible warts. It's common for people to have the virus on their skin and mucous membranes and to pass it on unwittingly. HPV transmission is so common that most sexually active people will have an HPV infection over the course of their life; however, unlike herpes, the viruses do not remain in the body forever. In most cases, the body manages to deal with the infection, eliminating it entirely within a year or two.

"Is there anything we can do about this?" Gunnar asks.

"Well, there's always the option of just leaving them be," I say. "Waiting for them to disappear by themselves."

"But they itch – and they're insanely big," Gunnar says.

Another option is that he could paint the warts with corrosive liniment or smear them with immune-modulating ointment himself. The second treatment is effective but hideously expensive, while the first stings and will damage normal skin if you miss the warts.

"Your warts are in a pretty awkward spot," I say. It'll probably be tough for you to reach them considering where they are."

"Sure will," says Gunnar. "I'd rather not do any painting or smearing."

"We can try to freeze them off."

Gunnar gives me two thumbs up. I cover his groin with a towel and fetch a canister of liquid nitrogen.

Doctors have been using cold temperatures to reduce inflammation and pain since the days of Hippocrates. But cryosurgery – eliminating unwanted tissue with cold – was first introduced in the mid-19th century when an English doctor, James Arnott, mixed up crushed ice and salt, which he used to freeze and destroy visible cancer tumours. Arnott's ice mixture reached a temperature of minus 24 degrees Celsius. But the treatment is more effective at lower temperatures, which we can easily achieve today.

When highly compressed in a metal container, nitrogen – which accounts for more than 78 per cent of the air around us – takes on liquid form. The pressure also causes its temperature to sink, and the liquid maintains a temperature of roughly minus 200 degrees. Cryosurgery is simple, cheap and achieves good cosmetic results, creating only minor scarring or other skin damage.

I attach the metal canister of nitrogen to a cylinder head with a trigger for my finger and a thin nozzle that makes it easy to target small objects accurately.

"Here," I say, showing Gunnar how I test the flask by spraying a thin jet of liquid nitrogen onto my own forearm; the hairs become frosty – a bit like grass before sunrise on a winter's day. "Now we'll try to freeze them. Ready?"

Gunnar nods.

I aim at floret after floret of his cauliflowers. They freeze and turn white. Gunnar feels a slight prickling sensation, but it doesn't hurt.

“Hopefully that will get them to retreat,” I say. “But you may need several rounds. And even if they do vanish now, they may still come back before disappearing entirely.”

“Great,” says Gunnar, as he jumps off the chair.

He puts his clothes on behind the screen, then heads for the door.

I’m relieved that the consultation has been so quick. Now I’m only five minutes behind schedule for my next patient. I smile and tell him to come back if he needs anything else. But just as Gunnar is reaching for the door handle, he stops and turns back towards me.

“Hey, by the way,” he says. “I have a bit of a rash…”

“I’m afraid our time’s up,” I say. “I was only meant to look at the warts today.”

“Can’t you just take a quick look?”

“No,” I say. “Sorry, but I can’t.”

Although it must seem odd to Gunnar that I, with my state-funded education, can’t help him with a rash, the fact is that I have to devote my time to the specialist services the clinic offers. To ask me, a worker at a sexual health clinic, to examine a rash, a stress fracture or a sore throat is – unless it’s related to sexual health – a misuse of my time.

“Okay,” he says. “I’ll book an appointment at my regular doctor’s. The thing is, I’ve never had a rash on my palms before, so I was just wondering about it.”

“The rash is on your palms?”

“Yeah,” Gunnar says. “Look.”

He holds his hands up in front of him to show me.

The hair rises on the back of my neck.

“What do you think it is?” Gunnar asks.

“You don’t happen to have had a round, sort of hard sore anywhere on your body, do you?”

“Actually, yes,” Gunnar says.

“On your penis?”

“Yes,” Gunnar says. But it didn’t hurt and then it just vanished so I didn’t give it any more thought.”

“Fuck,” I say.

“What did you say?” Gunnar asks.

“Sit back down,” I say. “And I’ll take a look at that rash of yours.”

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