Synopsis of Causation

Urinary Calculus

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Disclaimer

This synopsis has been completed by medical practitioners. It is based on a literature search at the standard of a textbook of medicine and generalist review articles. It is not intended to be a meta-analysis of the literature on the condition specified.

Every effort has been taken to ensure that the information contained in the synopsis is accurate and consistent with current knowledge and practice and to do this the synopsis has been subject to an external validation process by consultants in a relevant specialty nominated by the Royal Society of Medicine.

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1. **Definition**

1.1 A urinary calculus (or stone) is a structure which forms and grows in the **urinary tract** by the precipitation and deposition of substances previously in solution in the urine.

1.2 Almost 90% of calculi contain calcium salts. Some contain one “pure” calcium salt, such as calcium oxalate; more commonly there are mixtures of calcium salts, or mixtures of calcium salts and other compounds. Calculi also contain proteins and other organic material forming the matrix in which crystals of calcium etc. are embedded.

1.3 About 5% of calculi consist of uric acid. The remaining 5% have assorted, rare causes including congenital errors of metabolism.

**Figure 1: Urinary Tract**
2. **Clinical features**

2.1 Urinary calculi are common; at least 1 person in 10 will be found to have a urinary calculus at some stage of life. Men are affected 3 times as commonly as women, so the lifetime risk for a man is at least 15%. Many stones cause no symptoms. Some are found incidentally on x-rays taken for unrelated reasons, so many must be undetected, and the true prevalence will be higher than these figures indicate.

2.2 Most calculi cause problems between the ages of 20 and 50.

2.3 Symptoms depend on the site of the calculus in the urinary tract. Most of the calculi that cause symptoms are in the kidney or ureter.

2.3.1 **Calculus in the kidney.** Many calculi cause no symptoms and are found incidentally. Most such calculi are in the kidney. A small calculus in the kidney may never grow or move, and never cause trouble.

2.3.2 Some stones will remain in the kidney and grow. They may reach several centimetres in size. They will cause a dull pain in the loin, bleeding in the urine (which may be “frank” - i.e. visible to the patient - or “microscopic” and detected only on testing the urine) and an increased risk of urinary infection. Kidney stones are dealt with either by non-invasive disintegration by shock waves generated outside the body and focused on the stone (lithotripsy), or by “key-hole surgery” through a single puncture in the loin (percutaneous nephrolithotomy).

2.3.3 Shockwave disintegration has much reduced effectiveness for stones greater than 2cm in diameter, and the success rate falls rapidly as size increases beyond this. Such stones are better dealt with by mechanical disintegration during “key-hole surgery” than by shock wave disintegration, although the latter may be needed to “mop up” fragments left after the former.

2.3.4 Small kidney calculi may leave the kidney in the flow of urine. If they are very small (say, less than 3mm in diameter) they may pass unnoticed down the ureters, into the bladder and out along the urethra with the next act of passing urine. Calculi between 3mm and 6mm in diameter, and which leave the kidney, will probably eventually pass through the urinary tract but are likely to be held up temporarily at some point in the ureter. These are dealt with in section 2.3.5. Calculi between 6mm and about 1cm can leave the kidney but will be held up in the ureter and may not progress, requiring removal, as in section 2.3.5.

2.3.5 **Calculus in the ureter.** Calculi in the ureter will always have started in the kidney and be en route to the bladder. As outlined in section 2.3.4, calculus may be held up at one of the narrow points of the ureter. This causes obstruction to the flow of urine in the ureter, leading to increased muscular activity in the ureter in an attempt to move the calculus onward, which in turn causes an extremely severe pain usually known as “renal colic” or, more correctly “ureteric colic”. This pain starts in the loin and radiates round the front of the abdomen, down to the groin and genitalia. Most calculi less than 6mm in diameter will pass if the pain can be controlled. Calculi that do not progress are dealt with surgically, usually by endoscopic removal.
2.3.6 Occasionally, the obstruction to urine flow in the ureter will cause infection. This combination of obstruction and infection is very dangerous and needs emergency surgical treatment. Emergency treatment is also needed in the rare cases in which the patient has only one kidney, where obstruction of the sole ureter will quickly lead to renal (kidney) failure.

2.3.7 **Calculus in the bladder.** This will cause irritation of the bladder, leading to a frequent and urgent desire to pass urine, bleeding in the urine (which may be visible or detected only on testing) and recurring attacks of cystitis. Treatment is by **endoscopic removal**. Bladder stones are rare in the Western world.

2.3.8 **Calculus in the urethra.** It is very rare for a calculus to become stuck in the urethra. One which does so causes extreme pain and inability to pass urine, resulting in urgent treatment by surgical removal.

2.4 The dominant clinical feature of urinary calculi is their tendency to recur, often despite appropriate treatment.
3. **Aetiology**

3.1. In the Western world, most urinary calculi originate in the kidneys (although they may not cause symptoms until they pass from the kidneys into the ureters or bladder). The aetiology of most urinary calculi is, therefore, that of kidney (renal) calculi.

**Aetiology of calculi in the kidneys**

3.2. Calculi begin in the kidneys as tiny plaques of urinary solutes on the lining of the kidney. These plaques grow by further deposition of urinary constituents on their surfaces. Growth rates vary widely. At one end of the scale, some calculi take years to increase in diameter by a few millimetres; at the other, they may double in size every few months, especially in the presence of low urine volume, biochemically abnormal urine, or urinary infection.

3.3. Thorough and detailed investigation identifies an underlying cause for calculus formation in over 90% of cases. In routine clinical practice, investigation is often limited and many cases are wrongly described as “idiopathic”.

3.4. **Low urine volume** is the most important factor contributing to calculus formation and growth. There is a lower limit to the amount of urinary solute that has to be excreted each day, so a low urine volume inevitably causes concentrated urine. Some substances will be present in concentrations at the upper limit of their solubility, leading to precipitation of crystals, which aggregate into calculi. A low urine volume results from an oral fluid intake insufficient to maintain a normal 24-hour urine output of, ideally, at least 1.5 litres in a temperate climate. A low oral fluid intake is an occasional cause of low urine volume, especially in old age or in occupational or other circumstances which limit the facility to drink frequently. Much more common is the situation where extra-urinary fluid loss is excessive. This can occur in situations of chronic diarrhoea or after some forms of bowel surgery, but excessive fluid loss by sweating is by far the most important cause. Oral fluid intake needs to exceed perspiration losses by at least 2 litres to maintain satisfactory urine volume.

3.4.1 Physical effort in a hot environment can cause losses of several litres of sweat a day. From the results of a questionnaire survey of 406 male workers in several occupations in Asia, outdoor work in a tropical environment appears to be associated with a fivefold increase in prevalence of urinary stones compared to indoor work. In this study, there was no increased risk of stone formation from sedentary occupations carried out in the tropical environment. Another study of machinists, engaged in moderate physical activity, showed that 236 workers in a very hot environment had a stone prevalence about 3.5 times that of 165 workers similarly engaged in a normal temperature.

3.4.2 Low urine volume (which is virtually synonymous with dehydration) is thought by clinicians to be the sole cause of calculus formation in 10% of cases, and a significant contributor in 50%. A survey of the epidemiological records of US military personnel from the 1980s showed a doubling of the annual incidence of stone formation in army aircrew. It was speculated that dehydration was the most likely cause, partly on the basis of a quoted study of Royal Navy personnel from 1968, which showed an increased incidence of urinary calculi in
personnel, such as engineers and cooks, working in hot environments. None of these studies is prospective, and the reliability of questionnaire surveys or record reviews is always questionable. However, the results support the strongly held clinical impression of experienced urological surgeons that, among “stone formers”, people who have lived or worked in tropical or other hot environments are over-represented, as are those who admit to low fluid intake.

3.5. The duration of exposure to a hot environment needed to cause calculus formation was the subject of a US military study of Department of Defense personnel deployed in Iraq and Kuwait in 2003.\(^7\) One hundred and eighty-two personnel developed symptomatic, radiologically proven calculi during a 6-month period. Taking the onset of deployment in the Middle East as time zero, the mean time to presentation with symptomatic calculi was 93 days, with a standard deviation of 42 days. This time is very much shorter than clinicians have generally supposed. The study provides unprecedented and rather surprising information, which is especially relevant in the current context, and the limitations need to be considered as follows.

3.5.1 Firstly, the incidence and prevalence of calculi were unknown. The estimated deployed population of 150,000 (personal communication with the first author) was in constant flux. The study involved one hospital. Although this was the only tertiary referral hospital, there were 3 combat support hospitals, other smaller medical units, and medical and paramedical staff attached to larger military units, all of whom will have treated less complicated cases of renal colic. Meaningful calculations of the expected incidence and the actual incidence are impossible. In terms of expected incidence, the usually accepted figure for the annual incidence of symptomatic stones in middle-aged Caucasian males (a different population from the study population) is 1%; so in a stable population of 150,000 such men, an expected 750 cases would present in 6 months. For the study incidence to be higher than this figure, the hospital involved would have had to receive only one-quarter of all symptomatic stone patients. It may be that the incidence in the study was indeed lower than expected, because of the different population and the measures of forced hydration (see below). Lack of data on prevalence and incidence does not in itself detract from the significance of the results in terms of time to presentation.

3.5.2 Secondly, the study population was probably not representative of the total population of those with symptomatic stones. It is likely that personnel treated outside this unit had smaller stones, most of which passed with conservative treatment, and were therefore more likely to be of more recent formation. If this selection bias had any effect it would be towards the stones in the study population having greater size (and, therefore, longer time since formation), resulting in an overestimate of mean time to presentation rather than an underestimate.

3.5.3 Thirdly, the study assumes that at “time zero” (i.e. date of deployment) in the Middle East, no stones were present. This cannot have been the case, as some personnel would have had undetected, asymptomatic calculi at deployment. Such stones would have been developing for some time, some of them for a long time. If there were a significant number of such stones, the results would be biased in favour of a shorter time to development. However, a history of recurrent stone problems, or known bilateral stones, prevents acceptance for
military service; pre-deployment medical screening precluded deployment of those with symptomatic stone disease, and those with stones treated in the previous 30 days (who were at risk of having remaining fragments) were excluded from the study. These factors would combine to reduce the prevalence of asymptomatic stones in the study population at deployment to well below that in the general population.

3.5.4 Fourthly, the paper states that measures were in place to ensure forced hydration. This was done with bottled water, some of which contained high concentrations of calcium. If personnel were compliant with forced hydration, it could be argued that calculus formation was due to a high calcium intake, not to dehydration. It is impossible to assess the likelihood of this being the case because: the rate and degree of compliance are not known; bottled water was supplied by “3 dominant suppliers” and had calcium concentrations ranging from “average” to “hard” (over 3 times average); and the authors have no information on which products or mixtures of products subjects had consumed. It could, reasonably, be argued that development of a calculus was a result of service in the relevant environment for an appropriate length of time, and whether this was due to dehydration or to mandatory measures to prevent it, the calculus was still a result of that service (unless it could be shown that the “forced hydration” involved fluid with a low calcium content).

3.5.5 Fifthly, the only remotely similar study is one quoted by the authors from 1945, involving US personnel deployed for 18 months in an undisclosed desert environment. The mean time to presentation with stone disease was virtually 11 months (standard deviation 5 months). These are longer intervals than those in the recent study, but there is no information on the climate (not all deserts are hot, especially at night), nor on any hydration measures, and the diagnostic and treatment facilities available at that time were very different from those available in 2003.

3.5.6 Despite these difficult considerations and reservations, this paper clearly shows that in the context of military service in a very hot environment, urinary calculi can develop to a stage sufficient to cause symptoms within a timescale of a few months at most. It is relevant that the study recorded time to presentation with symptoms, not to stone formation, which would, obviously, be significantly shorter. On clinical grounds, there are good reasons to believe that for every person with a symptomatic calculus there would be at least one (and probably more) who would have a calculus causing no symptoms by virtue of its size or position. Such a calculus formed during, say, 1 or 2 months of service in a very hot environment could grow slowly after return to temperate regions. It would be difficult to refute a claim that even a few weeks of such service had resulted in the formation of the nucleus of a calculus which had subsequently grown after return to a temperate climate, perhaps causing symptoms years later, and which would not have developed at all without the initial period of relevant service.

3.6 Increased calcium in the urine (“hypercalciuria”). This is one of the commonest specific abnormalities found in stone formers. The amount of calcium in the body depends on a balance between absorption of dietary calcium from the gut, use of calcium in the body (especially in bones), and excretion of calcium into urine by the
kidneys. These activities are partly controlled by a hormone produced by the parathyroid glands.

3.6.1 Increased calcium absorption from the gut occurs with parathyroid overactivity, but often may occur without such overactivity, when the exact cause is unknown. In severe forms, too much calcium is absorbed even if calcium in the diet is restricted. In mild forms, too much calcium is absorbed only if the dietary calcium is above normal. The excess calcium is excreted in the urine, predisposing to calculus formation.

3.6.2 Increased calcium loss in the urine, due to increased blood calcium concentration, occurs with parathyroid overactivity. It may occur without such overactivity, caused by a “leak” of calcium from the kidneys despite a normal blood calcium concentration.

3.7. Increased oxalate in the urine (“hyperoxaluria”). This is a common abnormality in stone formers. Mild cases may be caused by excessive dietary intake of oxalate-rich foods such as chocolate, spinach, rhubarb, strawberries and asparagus. More severe cases may be caused by excessive absorption of oxalate from a normal diet, especially in people with inflammatory bowel disease, notably Crohn’s disease.

3.7.1 People with inflammatory bowel disease have an increased incidence of urinary calculi, which can be as high as threefold, and 36% of people with Crohn’s disease have hyperoxaluria.

3.7.2 Rare inherited metabolic defects can cause hyperoxaluria, but these conditions cause stone formation (and other disabling abnormalities) in childhood, so sufferers are unlikely to be accepted for military service.

3.8. Increased uric acid in the urine (“hyperuricosuria”). This occurs in gout, and in people with abnormalities of uric acid metabolism who do not have other features of gout. Sufferers may produce pure uric acid calculi, or calculi with a nucleus of uric acid which is covered by layers of calcium salts. They also are at increased risk of producing stones which contain no uric acid.

3.9. Other causes. These are numerous, but individually rare. They include: low concentrations of magnesium or citrate in the urine; inherited disorders of cystine or xanthine metabolism; urinary tract infection; and treatment with certain drugs. The most common examples of the latter cause are thiazide diuretics. Indinavir, used in the management of HIV, may precipitate in the urine to form stones composed of the drug itself, as can the constituents of proprietary cough medicines taken to excess.

3.10. Low urine volume combined with any of the causes in 3.6 to 3.9 will further increase the risk.

Aetiology of calculi in the ureters

3.11. Calculi in the ureters are always calculi passing from the kidney to the bladder, and so have the same aetiology as kidney calculi.
Aetiology of calculi in the bladder

3.12. In the Western world, many bladder stones probably form by deposition of precipitated urinary solutes on small stones which have formed in the kidneys and passed (often unnoticed) down the ureters to the bladder. Bladder stones can grow to several centimetres in diameter. Growth is accelerated by a low urine output (as in 3.4), urinary infection, and urinary stasis. Stasis occurs when bladder emptying is impaired because of obstruction to the outflow of urine (as in the condition of prostate gland enlargement) or because of damage to the nerve supply to the bladder as a result of spinal injury or conditions such as multiple sclerosis.

3.12.1 Some calculi may form primarily in the bladder because of the above risk factors.

3.12.2 A specific increased risk of bladder stone formation occurs in patients with long-term urinary catheters, where the catheter forms the focus for crystal deposition. Such catheters are used on a long-term basis in many patients after spinal injury. Urinary catheters are also used for a few days or weeks in critically ill patients, after major surgery, and after pelvic or spinal trauma. It is clinically recognised that the nucleus of a stone can form on a catheter in such a timescale. The small amount of calculus material may remain behind after the catheter is removed, to form the basis of a stone which may become obvious months or years later. Calculus formation and growth will be accelerated by the risk factors discussed in sections 3.2 - 3.10.

3.12.3 In developing countries, bladder stones are much more common than in the West, and often occur in children. This was the situation in the West until the middle of the 19th century, and is almost certainly due to dietary deficiencies, exacerbated in many developing counties by dehydration due to heat and to difficulties in maintaining sufficient oral fluid intake.

Aetiology of calculi in the urethra

3.13. Calculi in the urethra are very rare. In virtually all cases, they will have formed in the bladder and be passed into the urethra at bladder emptying, so their causes are as for bladder stones. Rare exceptions are calculi which form in the urethra in men who have had urethral reconstruction after pelvic trauma.

Recent research

3.14. Almost all calculi have been found to contain nanobacteria at their cores. This strongly suggests that such bacteria have a causative role, perhaps equivalent to that of helicobacter in peptic ulcer disease. Unlike that disease, urinary calculi are unlikely to be treated successfully with antibiotics. By the time someone presents with a stone, the bacteria at the core are covered by dense layers of calcium salts, which would prevent access by antibiotics. It may be that antibiotic therapy will have a role in the prevention of recurrences in people at high risk, but this has not come into clinical use.
4. Prognosis

4.1. Prognosis for kidney and ureteric stones - acute “stone episodes”. The prognosis is excellent for resolution of an acute episode of pain due to obstruction of the kidney or ureter by a calculus (as in sections 2.3.4 and 2.3.5). With modern techniques of minimally invasive treatment, 99.9% of such episodes will be resolved without open surgery. The main concern is the high risk of further stone problems. The prognosis is serious for an obstructing stone that is not treated. The kidney on the affected side will gradually lose its function, the rate at which this happens being dependent on the degree of obstruction. There are, for obvious reasons, no prospective studies of untreated obstruction. The clinical impression from the few cases of “missed” obstruction, patients who refuse treatment, and from experience in situations where medical care is inaccessible, is that complete loss of function can occur in a very few weeks. In other cases, months of severe but not total obstruction can be followed by complete recovery after treatment. Without any treatment, an obstructed kidney will eventually lose all function. If infection occurs in an obstructed kidney (as in section 2.3.6), septicaemia and death can occur in hours without treatment. Obstruction of a solitary kidney (also 2.3.6) causes acute renal (kidney) failure, which is fatal without treatment.

4.2. Prognosis for kidney and ureteric stone recurrence after treatment. Renal calculi are renowned for their tendency to recur, even after apparently satisfactory treatment. Interpretation of published figures is made difficult by several factors. True recurrence (i.e. new stone formation) is impossible to distinguish from growth of fragments remaining after treatment but initially below the size threshold for detection. Patients declared “stone-free” after treatment may have persisting, undetectable, small fragments which form the nuclei of further stones: this is especially likely after fragmentation of larger stones rather than simple removal of smaller ones. Authors do not always differentiate between patients who pass their first stone without intervention, those who have endoscopic removal of small stones without fragmentation, and those who have fragmentation. Similarly, they do they always make clear which, if any, regimes to reduce the risk of recurrence have been used in the observation period. Compliance is generally very poor, even when the required “treatment” involves only avoiding dehydration or avoiding bottled water with high calcium concentrations. 11

4.3. The risk of recurrence. Uribarri and colleagues reviewed 6 large retrospective studies of stone recurrences involving different types of stone. After the initial episode the risk of recurrence was 14% at 1 year, 35% at 5 years, and 52% at 10 years. 12 This review study is often quoted, but may not be relevant to modern practice. The studies were published between 1969 and 1985. Treatment of calculi has been transformed since the early to mid 1980s, when shockwave disintegration, keyhole surgery and endoscopic removal became established. Interpretation of the figures is complicated by the fact that the patients were said to have had no specific treatment after the initial episode, but the authors state that, “…because they attended centres of specialized medical care they probably received advice on non-specific measures such as increased water intake and dietary restrictions.” These measures significantly reduce recurrence rates and are now the mainstay of treatment for most patients.

4.3.1. In 1985, the cumulative risk of recurrence after a “stone episode” was reported as 75% over 20 years. 13 Again, this may not be relevant to modern practice.
4.3.2. In a more recent study, a 5-year follow-up period was evaluated following an initial “stone episode” involving a calcium-containing calculus (which may or may not have needed interventional treatment). In this study, 27% of patients who had no preventive measures had at least one further stone, as detected by radiological imaging and not necessarily symptomatic. The mean interval to “new” stone formation was 2 years. This is probably the most representative figure for current risk in patients with no detectable metabolic abnormality who are given no advice about diet or fluid intake. The risk of such asymptomatic calculi later causing problems is covered in section 4.5.

4.3.3. After shock wave disintegration of kidney stones with a variety of compositions, the “radiological recurrence” rate at 4 years was 14%, with a mean time to (radiological) recurrence of 20 months. However, the mean diameter of the treated stones was only 1.4cm.

4.4. Reducing the risk of recurrence. When a distinct metabolic cause is found, specific drug or dietary treatment may be available, but the risk of recurrence tends to be reduced rather than abolished. Even in the very specific case of parathyroid overactivity, surgical cure of the parathyroid problem is followed by a persisting (though diminishing) risk of further calculi for almost 10 years.

4.4.1. Improvements in minimally invasive techniques for treating renal and ureteric calculi are the most likely developments in the near future. Some of these improvements are likely to ensure better stone clearance, thus reducing the risk of early “recurrences”.

4.4.2. Increased awareness of the need for thorough investigation of stone formers should lead to more frequent identification of underlying correctable causes of calculus formation. This applies especially to first “stone episodes”, which have traditionally been under-investigated. Because of the complex aetiology of stones, and the difficulties of ensuring compliance with dietary and pharmacological regimes, this increased awareness might not translate into proportional clinical benefit.

4.4.3. The most common and useful measure to reduce the risk of recurrence is an increase in fluid intake, to produce a urine output of 2.5 litres per day. Increased fluid intake alone (without any other dietary measures) was associated with reduction of the 5-year cumulative recurrence rate from 27% to 12%, and an increase in the mean time to recurrence from 2 years to 3.2 years.

4.5. Prognosis of asymptomatic kidney stones. In a prospective study of 107 people with asymptomatic kidney stones detected radiologically, the cumulative risk of a painful “stone episode” was almost 50% at 5 years. The risk was proportional to the number of stones at initial detection, and to the number of previous “stone episodes”. In other words, the more such problems people have had, the more they are likely to have. Half of the people who had such an episode needed intervention and half passed the stone without it.
4.6. **Prognosis of bladder calculi.** Bladder stones are also renowned for their tendency to recur, especially in the presence of a urinary catheter (as in section 3.12.2). A high and uniform fluid intake is the main preventive measure."
5. Summary

5.1. Urinary calculi are common. The lifetime risk of calculus development is over 10%.

5.2. Over 90% of urinary calculi contain calcium salts.

5.3. The aetiology of calcium stones is varied and complex.

5.4. There are many specific disorders of the body’s handling of calcium, of oxalate, and of other substances that are recognised causes of calculus formation.

5.5. Low urine volume, with concentrated urine, is the single most common, important and correctable cause, either alone or in combination with other risk factors.

5.6. Dehydration, most commonly due to physical exertion in a hot environment, is the commonest cause of low urine volume.

5.7. The period of exposure to dehydration that is needed to initiate calculus formation can be as short as a few weeks. Once initiated, a calculus has an even chance of causing clinical problems within 5 years, but may not do so for very much longer.

5.8. Urinary calculi very commonly recur, even after appropriate treatment. Increased oral fluid intake is the most effective non-specific measure for reducing recurrence.
6. Related Synopses

Peptic Ulcer

Inflammatory Bowel Disease
7. Glossary

Crohn’s disease  A chronic inflammatory disease of the digestive tract that can involve any part of the tract from the mouth to the anus.

doscopic removal  Removal of a calculus (or other “foreign body”) by inserting optical instruments (endoscopes) through natural passageways. In the case of removal of a calculus from the urinary tract, this involves inserting a long, fine telescope into the urinary opening, passing it along the urethra and into the bladder. Bladder calculi can be seen, disintegrated, and the fragments removed. More commonly the calculus is in the ureter, in which case the endoscope is negotiated from within the bladder into the opening of the ureter, and then negotiated up the ureter as far as is necessary to see the calculus. The calculus is then removed under vision, if necessary after disintegration by laser or other means.

gout  A disorder of uric acid metabolism which classically causes acute, painful swelling of joints (not limited to the big toe), due to deposition of uric acid crystals. Other manifestations include urinary calculi, and renal failure independent of stone formation.

Helicobacter  *Helicobacter pylori*, a bacterium that infects the mucus lining of the human stomach.

idiopathic  Of unknown cause.

inflammatory bowel disease  A general term that refers to inflammation of the colon and rectum. Inflammatory bowel disease includes ulcerative colitis and Crohn's disease.

nanobacteria  Dwarf forms of bacteria, mostly 0.05 to 0.2 micrometers, about 1/10 the diameter and 1/1000 the volume of ordinary bacteria.

parathyroid glands  Four small glands lying close to the thyroid gland but not functionally related to it. They release the hormone parathormone into the blood. Parathormone maintains blood calcium concentrations by encouraging absorption of calcium from the gut and encouraging calcium release from bones. Excessive parathormone production causes high concentrations of calcium in the blood, which produce high concentrations of calcium in the urine, leading to precipitation of calcium salts and so calculus formation.
<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
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<tbody>
<tr>
<td>peptic ulcer</td>
<td>Ulcer in the stomach caused by pepsin and acid.</td>
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<tr>
<td>renal colic</td>
<td>Sharp, severe pain in the lower back that radiates into the groin; associated with the passage of a renal calculus through the ureter.</td>
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<td>renal (kidney) failure</td>
<td>The inability of the kidney to manufacture and excrete urine, causing waste product to accumulate in blood plasma.</td>
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<tr>
<td>urinary catheter</td>
<td>A tube of latex or silicone rubber which is inserted into the bladder through the urinary passage, to keep the bladder empty of urine. It is retained in the bladder by a small balloon which is inflated after insertion.</td>
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<tr>
<td>urinary tract</td>
<td>The pathway travelled by urine. It comprises: the funnel-shaped “collecting system” within the kidneys (correctly known as the pelvi-caliceal system); the 2 tubes (ureters), one on either side, which transport urine from the kidneys to the bladder; the bladder itself; and the urethra, which is the tube leading from the bladder to the body surface. Most stones which cause trouble are in the kidneys or ureters when they do so.</td>
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8. References


