Their virulence is greater than that of the bacillus coli. Like it they do not set up or maintain any urinary affection unless aided by an accessory lesion, but they very rarely cause bacteriuria, and the alkaline cystitis or pyelitis caused by them is severe in its symptoms and grave in its consequences.

**Proteus of Hauser** (*Proteus vulgaris, Urobacillus liquefaciens septicus*).—Experiments on animals at the hands of Krogius, Schnitzler, Bastianelli, and Melchior have shown that an intense and even a fatal cystitis may be produced, without the assistance of any predisposing cause, by injecting into the bladder a pure culture of the proteus vulgaris.

Like the pyogenic cocci this bacterium decomposes urea and causes *alkaline* cystitis. Probably it cannot take root and flourish in man without the aid of a predisposing agent, but it is nevertheless the most virulent of the common bacteria of urinary infection. Yet it has been found once in a simple bacteriuria.

**Gonococcus.**—It has long been known clinically that a gonorrheal inflammation may extend from the anterior to the posterior urethra and thence to the bladder, and that the gonorrheal prostate may form a base whence repeated incursions into the bladder are quite possible. Yet the proof of the existence of gonococcal cystitis has been singularly slow in appearing, and the cases reported are remarkably few. Thus far we only know that the gonococcus alone, unaided by any predetermining cause, may cause an acute cystitis; that in such cystitis the urine is acid; and that this purely gonorrheal cystitis recovers or is replaced by a secondary mixed infection so that the gonococci, if still present, can no longer be found. Hence that striking clinical condition, acute gonorrheal cystitis, may be accepted as purely gonococcal in origin; while nearly all the subsequent chronic manifestations in the bladder are due to secondary infection by those bacilli coli and pyogenic cocci that everywhere follow in the wake of the gonococcus to perpetuate the inflammations inaugurated by it.

**Bacillus Typhosus.**—It is only within a few years that the importance of the bacillus typhosus in urinary infection has been appreciated. According to the recent studies of Richardson [1] and Gwyn,[2] typhoid bacilli appear in the urine during the second and third weeks of the fever. Typhoid bacteriuria occurs in from 15% to 30% of all cases of typhoid. Grave pyelo-nephritis is rare, and while the bacilli usually spontaneously disappear from the urine they may remain for

[1] J. of Exper. Med., 1898, iii, 349, and 1899, iv. J. of Mass. Ass'n of Boards of Health, 1899.
[2] Johns Hopkins Hosp. Bull., 1899, x, 109.



years. Typhoid bacilluria is peculiarly amenable to treatment. The urine is acid and closely resembles that of bacillus coli bacteriuria.

### Routes of Invasion

Bacteria may reach the interior of the urinary tract by one of four routes (Rovsing):

1. From the urethra—ascending invasion.
2. From the kidney—descending invasion.
3. By irruption of a neighbouring focus.
4. By the circulation—indirect invasion.

Scientists are still at odds as to the predominating importance of any one route over any other. For the first observers, Pasteur, Bouchard, and others, the urethra was all important. Then Rovsing, Melchior, and Bastianelli, while still attributing the majority of cases to the urethra, recognised a renal origin for some cases. Albarran, Hallé, and Legrain hint that descending infection is more common than is generally believed. Infection through the blood or the lymphatic current has been made by most authors the special attribute of the tubercle bacillus, but Reymond [1] and Van Calcar [2] believe that the bacillus coli passes directly out of the rectum and in through the bladder when there is constipation or other intestinal obstruction. In the present state of our knowledge it is not possible to reconcile these opposing views, and in our consideration of the various routes of invasion it will be more practical to confine ourselves as strictly as possible to the clinical aspect of affairs.

**Urethral or Ascending Invasion.**—The three methods by which microbes may ascend from the urethra are:

1. Through instrumentation.
2. By extension upward of a urethral inflammation.
3. By spontaneous ascension of the urethral bacteria.

1. The passage of an instrument into a clean bladder is a frequent cause of cystitis. The gentle passage of a smooth, soft, clean instrument through a normal canal into a healthy bladder never causes cystitis. Perhaps bacteria are carried into the bladder by every instrument. Perhaps numerous pathogenic germs are often introduced in this manner. But experiment and experience unite to proclaim that the healthy bladder is thoroughly able to sweep itself clean of these enemies. Whether the germs come from a dirty catheter, or from an inflamed or a normal urethra, the bladder may be absolutely protected from them by the prophylactic injection of nitrate of silver (p. 218). But this is not enough. The bladder may

[1] Guyon's Annales, 1893, xi, 253 and 343.
[2] *Ibid.*, 1899, xvii, 1253.

be thus protected, but not so the posterior urethra. Not to mention the gonococcus, any of the specific bacteria enumerated above can take root in the prostatic portion of the canal if only the soil is sufficiently harrowed to receive the seed. Contusion or abrasion by the rough passage of an instrument often suffices, and if the bacteria are at hand, if a posterior urethritis is lighted up, if this posterior urethritis extends to the bladder and even to the kidneys, the heavy, rough, unskilled hand of the surgeon is to blame. No man with a general experience can fail to see that an infinite gentleness with a modicum of cleanliness spares the posterior urethra many woes into which it is plunged by the proud possessor of an infinite cleanliness with only a modicum of gentleness. It is not for me to depreciate cleanliness—the cleaner you are the better; but, if I may be allowed the phrase, the gentler you are the best. A gentle catheterization followed by an antiseptic irrigation does not cause any inflammation, unless the prostate is already inflamed or considerably congested, unless the vesical powers of resistance are greatly lowered, or unless there are gonococci about.

2. Infection of the urine by direct extension of an anterior urethritis backward is met with clinically only in gonorrhea and stricture.

3. Whether the bacteria of the uninflamed anterior urethra can ascend to the bladder in face of the urinary stream is a question not yet definitely decided. Certainly they may ascend a short distance along the anterior urethra, for they have been found, in a certain proportion of cases, at varying depths in the canal. Paladino-Blandini[1] found that if pure cultures of the staphylococcus aureus, the bacillus typhosus, and the "bacillus of green pus" were placed within the meatus urinarius of the guinea-pig, these bacteria could usually be found in the kidney at the end of twenty-four hours in the male, and at the end of forty-eight hours in the female. These experiments suggest that perhaps even immobile bacteria may travel against the urinary stream in small numbers, at least, and that the bladder and even the kidney may thus be invaded at any time. But before accepting such a theory, with all its startling possibilities, we must await confirmation of Paladino-Blandini's experiments by other observers.

With stricture of the urethra the combination of obstruction and infection is particularly favourable to the ascent of bacteria and the production of inflammation.

**Renal or Descending Invasion.**—Without stopping to debate the question whether or not the healthy kidney can transmit living bacteria in any numbers without injury to its secreting structure, we may accept as proved clinically, notably in the case of the typhoid bacillus, the fact that bacteria may enter the urine in great numbers from a kidney clinically sound. There is strong evidence for the belief that in the course of the various infectious diseases, even in tuberculosis, bacteria are commonly transmitted by the kidneys without leaving any appreciable trace of their passage through those organs. When the kidneys are inflamed their bacterial output is still less doubtful.

[1] Guyon's Annales, 1900, xviii, 1009.



It has been shown, moreover, by Carle, Posner and Lewin, Lesage and Macaigne (*cf.* Melchior, Rovsing, Van Calcar, Albarran, Hallé, and Legrain), that any coprostasis or constipation enhances the virulence of the bacillus coli in the intestine and causes these germs to leave the intestine in great numbers. A certain number of these bacteria reach the urine, presumably by excretion from the kidney. It is the prevailing tendency nowadays to attribute the spontaneous infection of prostatics to this cause, and hence great stress is laid upon keeping their bowels clear to avoid this possible source of danger. Direct extension of a parietal inflammation from kidney to ureter and from ureter to bladder occurs only in tuberculosis.

**Irruption of a Neighbouring Focus.**—Apart from those self-explanatory cases in which a fistulous or an exstrophied bladder becomes infected, the opening of an extraneous abscess into the urinary channels is rare. Perinephritic, perityphlitic, and, in the female, pelvic abscesses, may so rupture.

**Invasion from the Circulation.**—In spite of the contentions of Van Calcar and a few others, the great body of authors is united in denying that bacteria reach the urinary channels by emigrating from the general blood stream through the wall of ureter or bladder. It is generally admitted that only the tubercle bacillus may gain access from the lymphatics.

### Accessory Causes of Inflammation

From a consideration of the facts briefly noted in the preceding paragraphs one is tempted to wonder not that the urinary organs become infected, but that they escape the infection that forever threatens them both from above and from below. If every infectious disease, every inflammation, every constipation sends its myriad of bacteria through the kidneys; if every colony of germs deposited within the meatus sends its scouts upward as far as the kidney, it requires some stretch of the imagination to call the upper urinary tract aseptic, and it encourages a belief in the bountiful dispensations of Providence to find that the bladder and kidneys are not perpetually in-

flamed. Yet there is another equally important side to the picture. We have seen the perpetual incursions of the enemy: let us look at the measures Nature has taken to repel them—or, to use a more striking metaphor—we have seen the perpetual sowing of the seed: let us now consider the soil, its natural fertility, and the means by which it is rendered more or less fertile, remembering that however rich the seed it cannot grow upon a barren spot.

In its normal condition the urinary tract is an unfavourable soil. The walls of the channels are smooth, protected by a thick layer of epithelium, and constantly irrigated by the urinary stream. It is probable that the renal epithelium possesses a bactericidal power, and it is quite possible that the vesical epithelium possesses the same power to a less extent (Van Calcar). Certainly the bladder shows a marvellous resistance to infection so long as its mechanical functions are not interfered with. Many and many a man lives for years with a kidney pouring down a continual stream of foul pus and bacteria into his bladder, and yet, so long as that organ can perform its functions properly, so long as there is no obstruction to urination, the bladder suffers even less from the putrid stream flowing through it than would the integument under similar circumstances. But let an obstruction to urination arise, let the bladder become overstrained and congested in its fight against a stricture, let a pool of residual urine collect behind a hypertrophied prostate or a contracted neck, and inflammation at once results.

We have seen that most bacteria are not of themselves able to cause a urinary inflammation. Indeed, the gonococcus and the tubercle bacillus are quite unique in their capacity for causing an inflammation without any accessory lesions, and yet even they are not above availing themselves of accessory lesions when these exist. *No other bacterium can take root and multiply in the urinary tract unless the soil upon which it flourishes is congested.* Congestion is the plough that prepares the soil to receive the seed. This congestion may be acute or chronic. If acute it may be perpetuated by the bacteria once they have lodged. It appears under several clinical forms, of which the chief ones may be enumerated.

Bacteriuria is commonly perpetuated by a renal or a prostatic congestion (p. 364).

Cystitis is usually kept up by the congestion of retention (prostatic hypertrophy, stricture) or of stone or tumour. It may be set up by trauma.

Pyelo-nephritis is usually kept up by the congestion of retention, stone, or tumour. It may be set up by trauma.

The predisposing causes of bacteriuria are so special that they are best considered in the section devoted to that disease. The predisposing causes of cystitis and pyelo-nephritis are retention, stone, and tumour. That stone and tumour should cause congestion, ulceration, and in various ways undermine the resistance of the epithelium is no strange matter. But these conditions are special and none too common, while retention of urine is such an all-pervading cause of inflammation that it must be broadly though briefly considered here.



**Retention.**—The cause of retention of urine is commonly a urethral obstruction, and the urethral obstruction is commonly due to organic stricture, to contracture of the neck of the bladder, or to hypertrophy of the prostate. It is true that "stricture and prostate" does not cover all the ground. Thus retention may be due to vesical paralysis without any obstruction whatever, or the obstruction may be ureteral and not urethral (but such an obstruction gives special symptoms), or even if the obstruction be urethral, it may be due to a thousand things—to congenital tightness of prepuce or meatus, to stone, to spasm, to tumour; but, allowing all these exceptions, it is my belief that 95% of all cases of retention are due to urethral stricture, to contracture of the neck of the bladder, or to prostatic hypertrophy. These are the cases that we meet in practice, with a well-defined set of congestive and inflammatory symptoms affecting the upper urinary organs throughout.

**Effect upon the Bladder.**—The first effect of any urethral obstruction is that the bladder has to strain in order to empty itself. This strain implies congestion, and as the obstruction in the cases with which we are dealing is always chronic and usually progressive, so is the strain and with it the congestion, chronic and progressive. The bladder is pushed to the last extremity. In struggling to overcome the obstruction it undergoes compensatory hypertrophy (p. 341); it gives all its reserve force to this end, and if other enemies appear in the shape of an invading horde of bacteria it is no longer in so fit a condition to expel them as it should be. Yet it usually escapes infection until its muscle has been sufficiently overcome to permit the accumulation of a pool of residual urine. This is the burden that cannot be borne. The bacteria now arriving find a safe harbour in the residual pool. Here they settle and multiply. They have leisure to work at the congested bladder wall, and if they are ammoniogenic the irritating changes they produce in the urine aggravate the state of affairs to a marked degree. Clinically a chronic alkaline cystitis is infinitely more severe than a chronic acid cystitis, unless that cystitis be tubercular.

From this brief review it will be observed that the *bacteria are*

*not the most essential agents of infection.* They can do no harm in the urinary tract until some adventitious cause comes to prepare the soil for their growth. Unless the predisposing cause is there no inflammation can occur. While the predisposing cause persists an inflammation may indeed be cured, but there is no assurance that it will not relapse. So long as the predisposing cause exists there is danger. To cure the patient absolutely and permanently of his inflammation the predisposing cause must be removed. This done, the bladder will quickly dispose of the bacteria. This is the quintessence of urinary therapeutics: *to prevent inflammation avoid trauma; to cure inflammation relieve retention.*

**Effect upon the Kidneys.**—The effects of retention upon the kidneys may be summed up briefly. These organs become congested with the bladder, partly by a reflex nervous mechanism, but chiefly by the heightened urinary pressure transmitted from the bladder to the ureter, the pelvis and the kidney itself. In these cases of "stricture and prostate" that we are studying the pressure increases slowly through a space of weeks or months. Hence there is commonly little or no distention of ureters or kidneys until absolute retention is reached. But all the while there is a chronic congestion, causing in the ureters an inflammatory sclerosis comparable on a small scale to that met with in the bladder, while in the kidney the sclerosis resulting from long-continued congestion takes the form of a chronic nephritis (p. 555). The kidneys are thus permanently damaged. A certain proportion of the excreting epithelium is destroyed, and the remainder is forced to excessive work by this very loss. Thus congestion is piled on congestion and the kidneys are as ready as the bladder to become bacterially inflamed. If now an inflammation flares up in the residuum of the *bas fond*, the kidney falls an easy prey to it. Yet even here evidence of a conservative effort on the part of the kidneys is not wanting. The kidney does not—it has been already noted—become dilated until the state of chronic complete retention has been reached, and it is a noteworthy, clinical fact that until the kidney becomes so dilated and pouched that it has its own *bas fond* it does not usually become infected by ammoniogenic bacteria. It may fall a prey to bacillus coli infection of a light catarrhal sort in its early days of congestion, but it is not attacked by the bacteria of ammoniacal cystitis until it provides a special nook for their multiplication. Thus we frequently encounter cases of the most violent ammoniacal cystitis complicated by pyelo-nephritis, it is true, but acid pyelo-nephritis of a comparatively benign character. Occasionally the reverse of the picture is seen: a pouched, suppurating, disintegrated kidney pouring its multitude of bacteria through the bladder, which latter is affected little or not at all, because it is fully able to empty itself.



**Instrumentation.**—Rough instrumentation may, by bruising the neck of the bladder, light up a prostatitis and perhaps some cystitis; but the cystitis is only caused indirectly and will be of short duration unless some other cause of inflammation steps in to perpetuate it. The cystitis caused by instrumentation in cases of stricture or of hypertrophied prostate is started by the instrument and perpetuated by the retention. Other forms of trauma rarely figure in the etiology of urinary inflammation.

## BACTERIURIA

The one form of urinary infection that does not lend itself readily to classification is bacteriuria—the presence of great numbers of bacteria in the urine without obvious parietal lesion. It can scarcely be termed an infection of the urine without any lesion of kidney, bladder, or prostate. Such a theory, though it has been generally accepted, is opposed to all that we know of urinary bacteriology. We know that the simple injection of bacillus coli into the bladder does not cause bacteriuria. We know that the simple passage of bacillus coli, even in great numbers, from the kidney does not cause bacteriuria. We know that bacteriuria is often only an initial or a terminal phase of cystitis or pyelo-nephritis. We know, finally, that in many cases the bacteriuria may be temporarily conquered by vigorous treatment only to reappear again as soon as the treatment is remitted. In view of these facts it is not possible to accept the old theory that bacteriuria is due to the presence in the urine of a microbe which multiplies so fast that it is not swept away by the urinary stream. On the contrary, we can only conclude that bacteriuria is a collective term covering several different conditions whose salient characteristic is the rapid multiplication of bacteria, so that they swarm in the urine and are associated with little or no pus to indicate the existence of the local inflammation from which they take their origin. It is not just to restrict the term bacteriuria to those cases in which the urine contains no demonstrable pus whatever, for a little pus may appear in one specimen of urine and be absent from the next.

Bacteriuria has been studied at length by Roberts,[1] Krogius,[2] Rovsing,[3] Jeanbrau,[4] Gassman,[5] and others.

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[1] Brit. Med. J., 1881, ii, 623.
[2] Guyon's Annales, 1894, xii, 196.
[3] *Ibid.*, 1897, xv, 910.
[4] Gaz. des hôp., 1899, lxxii, 653.
[5] Guyon's Annales, 1900, xviii, 148.

**Etiology.**—*Bacteria.*—The bacterial agent in this condition is usually the bacillus coli, less frequently the staphylococcus or the bacillus typhosus. Jeanbrau has collected 67 cases with bacterial reports. In 56 (83.5%) cases the bacillus coli was found, in 7 cases the staphylococcus, and in the remaining cases the proteus, the streptococcus, the bacillus subtilis, and a large coccus. The bacillus typhosus does not appear in this list, but from what we are learning of the frequency of bacteriuria during typhoid fever and the possibility of its continuance, one may foresee that the typhoid bacillus will figure prominently in the statistics of the future.

*Route of Invasion.*—Jeanbrau has collected 29 cases of ascending invasion, chiefly attributed to urethritis, prostatitis, and catheterism. Renal invasion has not been clearly established in any great number of cases. Yet all typhoid bacteriuria must be of renal origin, as are also many of the bacillus coli cases. Invasion through a fistula or from a neighbouring pelvic abscess is too rare to be clinically notable.

*Predisposing Causes.*—In the pathogenesis of bacteriuria three salient facts may be observed:

1. The absence of cystitis (proved every time the cystoscope was employed).

2. The presence in many cases of the symptoms or signs of nephritis or pyelitis (22 cases in Jeanbrau's tables), of prostatitis, or of prostatic hypertrophy (19 cases), of urethritis or stricture (5 cases), of incontinence of urine (2 cases), of vesiculitis, etc.

The predisposing causes of cystitis and pyelitis are notable for their relative unimportance here. Retention was observed only 8 times, stone and tumour but 5 times. But bacteriuria is oftenest associated with an inflammation of the kidney or the prostate. In other words, *bacteriuria is usually the expression of a catarrhal prostatitis or pyelo-nephritis*, though it may be the expression of a catarrh of any part of the urinary tract, provided only that the parietal lesion is sufficiently slight and the bacterium sufficiently prolific.

When bacteriuria originates in the prostate or bladder it is said to be partial or vesical. The urine in the ureters and kidney pelves is perfectly clear. But when bacteriuria originates in the kidney the whole of the urine is bacterial; there is total bacteriuria.

The association of bacteriuria with incontinence of urine has been noticed by several authors. It is an open question whether the bacteriuria causes the incontinence or the incontinence the bacteriuria. When the incontinence is due to a spinal lesion the bacteriuria may very fairly be attributed to the weakened resistance of the urinary organs to infection.



**Symptoms.**—*The Urine.*—The urine of bacteriuria is hazy. It contains no gross particles or cloud of pus, but seems to be filled with the finest sort of a white powder. In a strong light the urine has a peculiar opalescence. Its reaction is acid in the great majority of cases. Its peculiar sickening odour has been compared to that of a mouse or a dead fish. No deposit occurs on standing, nor is the haze affected by heat or chemicals unless albumin is present. The centrifuge affects the cloudiness but little, and throws down only the merest trace of a deposit. A microbic sediment may be obtained by adding equal parts of absolute alcohol to the urine and then centrifuging (Hallé).

Examination under the microscope of a drop of urine obtained from the bottom of a centrifuge tube reveals innumerable bacteria, usually the squirming bacillus coli, intermingled with a few epithelia and leukocytes, with perhaps casts and blood-cells and crystals.

The urine may be albuminous. It is rarely phosphatic.

**Course of the Disease.**—The urinary signs just described are the only essential characteristics of bacteriuria. In many cases there are no subjective symptoms whatever. The symptoms, when there are any, are those of the underlying prostatitis, pyelo-nephritis, etc. Thus bacteriuria may appear under many different forms. It may be the initial or the terminal stage of pyelitis, cystitis, or urethritis. It may be the most striking symptom of prostatitis or vesiculitis. It may result from typhoidal or other infection. Yet amid these and other clinical types there are two forms of bacteriuria so prominent clinically as to overshadow all other types of the disease. These are the pyelo-nephritic type and the prostatic type.

*Pyelo-nephritic Type.*—The course of the disease is that of a pyelo-nephritis. It may occur during a typhoid fever, during pregnancy, as a result of a general infection, of a diarrhea, or of a chronic constipation. The urine is acid, albuminous, bacterial. The disease is, in fact, a catarrhal pyelo-nephritis (p. 567).

*Prostatic Type.*—There may or may not be prostatic hypertrophy. The disease is often the terminal stage of an acute gonorrhea. There may be fistula or stricture. The urine is not albuminous unless it contains blood. It may be acid, containing bacillus coli, or alkaline, containing staphylococci or streptococci.[1] The symptoms are those of prostatitis or vesiculitis, and the bacterium is found in the expressed secretions of the prostate and vesicles. In fact, the course of the disease is that of a prostatitis or a vesiculitis, modified only by

[1] In only one case (Pedenko) were staphylococci found to be the cause of pyelo-nephritic bacteriuria.

the fact that the bacteriuria is for a longer or shorter time the prominent symptom.

**Diagnosis.**—If the urine is uniformly hazy, and that haze is cleared neither by chemicals, nor by the centrifuge, nor by standing, and there is no purulent deposit, bacteriuria is present. Bacteriuria may be suspected by the urinary appearance and odour. It can be diagnosed only by the centrifuge (which fails to clear the urine) and the microscope (which shows what little deposit there is to be almost entirely bacterial).

The distinction between pyelo-nephritic and prostatic bacteriuria may not be easy in a given case. Indeed, the two doubtless often coexist. Yet an alkaline bacteriuria is almost invariably prostatic, a bacteriuria following gonorrhea or due to instrumentation or to stricture is probably prostatic. A bacteriuria occurring during the course of a prostatitis or of a prostatic hypertrophy is doubtless prostatic. Finally, the expressed prostatic secretion (after urethral and vesical irrigation) will be found to contain the incriminated bacteria in great numbers if the bacteriuria is prostatic.

On the other hand, if the bacteriuria is pyelo-nephritic, the urine is acid and contains casts and albumin, and the clinical picture of pyelo-nephritis may be discerned.

**Treatment.**—Bacteriuria may continue indefinitely if left untreated, but unless of long standing it is usually very amenable to treatment. The treatment is that of pyelo-nephritis or of prostatitis, or, if no underlying lesion can be determined, the treatment is by urotropin and diluents (p. 373). Typhoid bacteriuria, for example, almost always yields readily to this treatment.

# CHAPTER XXIV

## *THE TREATMENT OF URINARY INFECTIONS AND INFLAMMATIONS*

So closely connected and so often confused are bacteriuria, cystitis, and pyelo-nephritis, and so many points of treatment do they possess in common, that it is convenient to group here their general therapeutic features, and to refer back to them in the succeeding chapters in such a way as to impress upon the surgeon the necessity of taking a broad view of the whole field. Thus, without losing sight of the particular details proper to each case and to each disease, he may appreciate what might be termed the Principles of Urinary Therapeusis applicable alike to the prevention and the cure of inflammation of the upper urinary tract. The subject may be subdivided into Prophylaxis, Palliative Treatment, and Radical Treatment.

### Prophylaxis

Clinically speaking, the prevention of urinary infection presents itself under three aspects:

1. The prevention of spontaneous infection when some disease of the urinary organs (notably prostatic retention) renders them especially liable to become inflamed.
2. The prevention of infection from urethral instruments.
3. The prevention of infection during or after operations upon the urinary organs.

1. **The Prevention of Spontaneous Infection.**—Since spontaneous infection of the urinary organs does not occur unless these organs are made vulnerable by the action of some predisposing cause (p. 359), the ideal preventive is the removal of such a cause. Thus the removal of stone, stricture, or tumour safeguards the bladder and kidneys absolutely. But in many cases, notably in prostatic hypertrophy, such radical treatment may well seem more formidable than the disease itself. Then the patient must be forewarned of the constant danger of infection, and forearmed against it by instructing him in the rules of what we have elsewhere termed Prostatic Hy-