Section of Urology

President—R. H. O. B. ROBINSON, F.R.C.S.

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Some Problems of Renal Lithiasis

PRESIDENT'S ADDRESS

By R. H. O. B. ROBINSON, F.R.C.S.

MANY excellent papers on the subject of calculus in the upper urinary tract have been read before this Section. Lett (1936) described a thirty-year survey at the London Hospital which included 2,100 cases of renal and ureteric stone. He mentions the higher incidence in the male sex and the very high incidence of associated infection. In a group of 546 cases of stone in the upper urinary tract, sterile urine was found in only 12%.

Winsbury-White (1938) reported a series of cases also stressing the higher incidence in males and pointing out the preponderance of stone in the upper urinary tract as compared with the lower. He confirmed this (1946) in a further detailed statistical report.

I propose to discuss some particular problems bearing on the ætiology, diagnosis and treatment of stone in the upper urinary tract, drawing attention to several points which are still unsolved and some difficulties which I have encountered personally. The following is an illustrative case.

A young woman aged 22 in 1936 developed backache, considered to be due to spondylitis and for which treatment with heat and massage was initiated. Right-sided abdominal pain developed and two years later the appendix was removed without in any way influencing the symptoms. On interrogation she stated that all her life she had had frequency of micturition. In due course, in 1943, she was called up for service. In 1944 an attack of dysuria developed, but later in the year she proceeded to the Middle East, where a fresh attack of cystitis with hæmaturia occurred. Renal investigation, then carried out for the first time, showed bilateral renal calculi and a renal function of 48%. Retrograde pyelograms showed definite dilatation of the calices on both sides. Twelve months later the urine still contained pus but was sterile. The general condition was fair with a blood urea of 40 mg.%, but backache was persistent. X-rays at this stage showed a definite increase in the opacities, particularly in the right upper calix, and an operation was carried out to remove this upper fragment, which was discovered by preliminary needling of the kidney followed by splitting the upper pole. This was presumably done for the dilatation of the upper calix above the stone, although there was some increase in the dilatation of all the calices on both sides. Convalescence was uneventful and the post-operative X-rays showed the upper fragment removed but a definite increase in the remaining stones, while the intravenous pyelogram still showed gross dilatation particularly of the upper calix. One year later, that is to say eight years after the onset of the original symptoms, I was consulted to advise for relief of the persistent backache. The

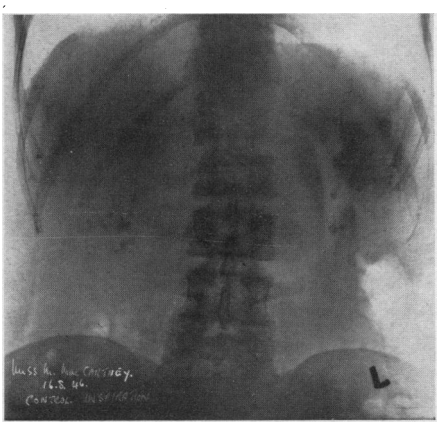

FIG. 1.—Multiple stones, both kidneys.

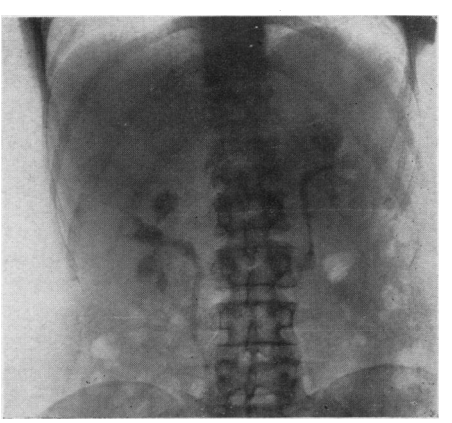

FIG. 2.—Associated dilatation of calices.

present position is that the pain is now more prominent on the left side and there is some nocturnal frequency. No obvious physical signs are present, but X-rays of the spine show bone changes of infective type. The urine is sterile but contains pus cells in fair quantity with an occasional red cell. X-rays of the kidney (fig. 1) show once more a definite shadow in the right upper calix in addition to the opacities in the lower and middle calices, and an intravenous pyelogram (fig. 2) shows the same degree of dilatation in the calices.

In this case: (1) Are we dealing with mobile renal calculi or fixed calcifications in the kidney substance? (2) Is the condition in any way associated with immobilization, the patient having been kept in bed for treatment of the spondylitis? (3) Has the kidney condition any connexion with infection, as the patient had acute cystitis, although at the time this occurred the calcification was well marked? (4) Immediately before the development of cystitis the patient was in the Middle East and therefore the possible effect of a hot climate has to be considered. In addition, she had received considerable heat treatment for her backache. (5) Lastly, can we find any indications in her case of a disordered metabolism? Under this heading are included hyperparathyroidism, hypervitaminosis D, hypovitaminosis A, toxins and maladjusted diet.

I will now discuss these various possibilities in more detail.

When I use the term mobile renal calculus, I refer to a stone which can migrate within the drainage system of the calices and renal pelvis, being of variable size but usually of such a size as to be capable of temporarily obstructing the ureter in its attempt to reach the bladder, and thereby predisposing to dilatation and stasis behind it, with an ever-present invitation to coincident infection. By fixed calcification I mean a condition which is well illustrated by the following case, which I reported to this Section in November 1934.

A woman aged 44 attended St. Thomas's Hospital in January 1934 with a three months' history of dragging pain in the left lumbar region. X-rays showed an irregular group of shadows in the lower pole. The urine was normal and cystoscopy revealed no abnormality. Pyelography showed the shadows to be in the lower pole of the kidney, outside the pelvis and without dilatation of the calices. Nine months later the patient was readmitted still complaining of pain, and it was decided to explore the kidney. There was well-marked perinephritis over the area involved, at which point the cortex was slightly depressed, yellowish in colour and gritty on section. The rest of the kidney, pelvis and ureter appeared normal. I removed the kidney on the supposition that it was tuberculous and I did not feel justified in carrying out a partial nephrectomy. Macroscopically there was a wedge-shaped area containing small calculi imbedded in the renal tissue and firmly adherent. Microscopic sections showed degeneration and chronic fibrosis of the kidney with dilatation of the tubules in the region of the calculi. Prior to operation the patient had been taking a well-balanced diet and had not had any excess of alkali. There was no evidence of disorder of the parathyroid glands, the plasma phosphate being 2·4 mg.% and the serum calcium 10·2 mg.%. Two years later, the patient was perfectly fit; but the pain was unrelieved. It is obvious that it would have been wiser to have treated the patient conservatively or performed a partial nephrectomy of the ischæmic area to avoid the potential development of renal hypertension.

Further, this type of calcification is different from that usually seen in tuberculosis, in which there is always evidence of destruction of one or more calices on pyelography and a less well-defined calcification, points discussed in detail by Sandrey before this Section in 1938. Nevertheless, Randall has recorded one very interesting case in which this type of intratubular calcification occurred outside the tuberculous area and was presumably due to the effect of the toxin on the surrounding tubules. Intratubular calcification is brought about by an excessive excretion of calcium phosphate and actual deposition in the walls of the tubules by degenerative changes in them associated with defective blood supply or toxic irritants. Experimental ligation of the renal artery and injection of mercuric chloride produce this change, as do hypovitaminosis A, hyperparathyroidism and specific toxins. The exact relationship of such calcification, Randall's Type II, to the interstitial papillary calcium plaque, Randall's Type I, is not perfectly clear, although it is conceivable that they are manifestations of the same phenomenon, that is to say, the response

of the kidney to toxins, the lesion depending on the intensity, frequency and length of time the organ is subjected to their influence.

At this stage it might be well to emphasize some of Randall's findings. In investigating 1,154 autopsies he discovered subepithelial papillary calcium plaques in 19·6% and in 6·5% true renal calculi originating from plaques, that is Type I calculi. Further, in a series of 265 primary calculi voided naturally, facets of origin from a papilla were obvious in 40%. Calcium deposition occurred in a relatively avascular and devitalized area in the interstitial tissue of the papilla beneath the epithelium, sometimes being deposited in rings round the basement membrane of the collecting tubules also. No evidence of infection was present and the calcium was constantly combined in the form of calcium nucleinate and not in any other form. Subsequently the covering epithelium necrosed, exposing the plaque to urine in the calix, and a secondary deposition of calcium phosphate, calcium oxalate, or uric acid occurred. He found that his Type II intratubular calcification occurred in 1·9%, or with only a tenth of the frequency of Type I. The condition usually affected a wide area of the kidney and multiple papillæ, and in 4 of the total 23 cases true renal calculi were originating from the papillary tubular matter. In 19 cases calcium phosphate was the precipitated salt and in 4 uric acid. All grades of involvement were found and in some cases isolated intrarenal calculi. Similar examples are the uratic inspissation of infants, the calcium infarction of Henle and the nephrocalcinosis of Albright. The condition had been suspected by Huggins (1933) to be a precursor of stone and Lubarsch has pointed out that, while the calcium is usually intratubular, it may also be extratubular in the cells and especially the collagenic membrane. Infection is not a primary feature and Randall suggests that the term calcium inspissation of the collecting tubules is a better one.

Type II calculi are definitely associated with the hyperexcretory state in which the colloid crystallized balance of the urine is upset. Underlying causes are (1) supersaturation with crystalloids such as oxamide or sulphonamide, (2) hypovitaminosis A and hyperparathyroidism, and (3) urea-splitting organisms in the urine.

Decubitus calculi, recumbency stones or stones associated with immobilization develop at a rapid rate in patients who are immobilized in the supine position, and are due to stasis alone. Therefore the importance of postural drainage of the kidneys has been stressed by many writers including Ogier Ward and Boyd. Rotation of the patient on to the face for some hours during the course of the day—particularly if associated with active diuresis—drains the calices and should be carried out unless it is impossible owing to retention splints. Pulvertaft (1939) recording 60 cases, mainly of bone and joint tubercle, pointed out that this difficulty can be overcome with an anterior plaster shell. The tendency to precipitation is very much increased when calcium metabolism is upset, partly by the inevitable disuse atrophy of bone and by bone disease or bone trauma. Often an unsuitable diet with an alkaline ash base has been given and there has been insufficient stress on fluid intake. Recumbency stones are composed primarily of calcium phosphate, soft and coralline in structure and situated in the most dependent calices. They tend to disperse fairly rapidly if the kidney can be flushed by getting the patient out of bed. This type of stone is a definite problem in the traumatic surgery of war, when recumbency with bone injury is commonplace. An important feature of such calculi is that they may be extremely silent in their early stages during which they are most easily dispersed. In Boyd's paper, 3 of the 4 cases he records were associated with anterior poliomyelitis, and I have such a case under my care at the present time. Here the stasis in the kidney due to deficient respiratory movements is probably a factor. A small boy of 8 with infantile paralysis developed hæmaturia and X-ray showed multiple coralline stones in the left kidney. An I.V.P. showed these to be in the calices rather than in the cortex. Flocks (1945) pointed out the necessity for repeated X-ray investigation in recumbent cases. Examination should be carried out at intervals of one, two, three and six months after the onset of the primary condition. Higgins (1940) emphasized the need for an acid ash diet fortified if necessary by ammonium salts, pointing out that the normally acid urine becomes rapidly alkaline on immobilization. He also pointed out the undesirability of prescribing mineral oil aperients as these lead to deficient

absorption of vitamin A. Injudicious and faulty instrumentation of the urethra may at any time introduce infection, and if urea-splitting organisms gain a foothold disastrous results may supervene. The failure to detect decubitus stones in their early stages has impressed me in cases of this type with which I have had to deal.

The relationship of infection to stone formation appears at first sight to resemble the fable of the hen and the egg. To simplify the problem, it is well, following Randall, to discard cases where calcification is found accompanying grossly diseased states of the kidney such as calculous pyonephrosis, chronic pyelonephritis with alkaline incrustations and simple calculi of long duration. The view is generally held to-day that at various times the kidney excretes living bacteria, that these produce changes in the renal tissue which may resolve completely, and that in the absence of obstruction chronic infection of the renal pelvis is most unlikely. The close relationship between infection and stone is brought about by the following facts: (1) Infection upsets the colloid crystalloid balance (Keyser, 1923); (2) it produces epithelial clumps which form nuclei for crystallization (Eisenstaedt, 1931); (3) it may produce papillitis (Aschoff, 1913); (4) living bacteria have been found in and cultured from the centre of calculi (Hryntschak, 1935; Hellstrom, 1936); (5) urea-splitting organisms produce calcium phosphate precipitation, as urea has definite solvent properties for this salt (Hellstrom, 1929). There is also the work of Rosenow and Meisser (1922) on specific streptococci cultured from the urine of patients with calculus, which some investigators, however, have been unable to substantiate.

Randall has carried out some extremely interesting work bearing on this point. He investigated 75 cases in which obstructing calculi were removed by open ureterolithotomy, cultures being made from the urine in the obstructed renal pelvis by preliminary aspiration. He found that in 48% the urine was sterile although ideal conditions prevailed for the growth of any organism present, while in the 52% in which the urine was infected 15 different varieties of organisms were isolated and in only 38% were staphylococci present. This conflicts with Hellstrom's postulate of at least 75% of urea-splitting staphylococci. Randall failed to find evidence of infection in his histological investigation of kidneys showing calcium plaques or intratubular inspissation. He did, however, demonstrate that the kidney is susceptible to bacterial toxins, excreting them and concentrating them in so doing. If a definite strength were reached, then the renal tissue suffered damage, the damage being most marked in the collecting tubules.

An interesting and well-substantiated observation is the fact that the organisms in the kidney urine may change from time to time; on one occasion, for example, proving to be *B. coli com.*, on another *B. lactis aerogenes* and later *B. coli* again. This suggests that the kidney urine varies considerably and that organisms absorbed from the bowel and excreted may or may not find conditions favourable to their growth, similar conditions having an opposite effect on other organisms.

The onset of tuberculosis in a kidney following preliminary obstruction by a calculus appears to be very rare, but I think this occurred in the following case:

A girl aged 5 developed tuberculous caries of the cervical spine in August 1931, and was treated by immobilization until February 1934. In May 1934 the disease became active again and further immobilization was instituted. In July 1934 transitory paraplegia developed and in November leftsided lumbar pain was complained of for the first time, but there were no urinary symptoms. The left kidney was palpable but there were no abnormal constituents in the urine. No tubercle bacilli could be found in the urine, sputum or stools. X-ray investigation showed old disease in both lungs and upper dorsal caries. The I.V.P. showed a well-developed calculus at the left ureteropelvic junction with an enlarged functionless kidney above it, whilst on the right side the kidney appeared normal in shape and function. At operation I found a phosphatic stone which I submit was of the decubitus type blocking the top of the ureter with ulcero-cavernous tuberculosis of the whole kidney, necessitating nephrectomy.

The influence of prolonged exposure to heat and sun in hot climates on the incidence of renal calculus brings out some interesting points. White races appear to have a higher susceptibility than black. Vermooten (1941), reporting on one million South African negroes admitted to hospital, found only one case of renal calculus, and the American negro, while not immune, shows a much lower incidence than the white. Pierlo and Bloom (1945) found that under desert conditions in the American Army among a group of men with adequate diet, but drinking hard water, and without pathological lesions in the genito-urinary tract, the ratio of white to coloured stone cases was 10 to 1. This upsets a dietetic causation which might operate in the African negro whose diet is deficient in calcium in spite of which the teeth and bones are perfect. Quinland (1945) investigated 16,000 hospital cases among American negroes, and found only 17 instances of stone in the upper urinary tract.

Turning now to the most important or metabolic factor in the causation of stone, we have first to consider the effect of disordered parathyroid activity. Calculi associated with hyperparathyroidism are a well-recognized group at the present time, but a point which is not so well appreciated is that they can occur without any lesions of the bony skeleton. This fact has been emphasized particularly by Albright and his associates. In a ten-year survey covering 67 cases of hyperparathyroidism they found that the typical picture of osteitis fibrosa occurred in only one-third, while in another third only mild skeletal changes were present, and in the last third no bony changes at all could be found. Albright insists that the renal changes are much more important than the osseous. Cook and Keating (1945) reported on 18 cases for the Mayo Clinic during an eighteen-month period in 1943-1944. They found that in 13 cases renal calculi were the prominent features and in the majority bone disease was minimal or absent. During the same period hyperparathyroidism was proven in 2% of all cases of renal calculus at the Clinic, but as not all calculus cases were fully investigated from this angle they consider the figure is probably higher. In many cases the stones were predominantly composed of oxalate. Foulds (1945) recorded an interesting case of bilateral renal calculi in which typical bone changes and hypophosphatæmia did not develop for eight years, although hypercalcæmia had been present for some time. In this case calcium oxalate was the predominant salt. On the experimental side Randall has produced calculi in dogs receiving parathormone and showing hypercalcæmia, finding that these calculi were of both Types I and II and that calcium deposition apart from calcium inspissation was preceded by necrotic changes in the renal tissue.

Wilder and Howell (1936) believe that there is a relationship between vitamin-D deficiency and hyperparathyroidism, vitamin D aiding in the absorption of calcium from the intestinal tract while the parathyroids mobilize it from osseous tissue.

Deficiency of vitamin A disturbs the calcium-phosphate ratio and produces changes in the epithelium of the urinary tract. Stones found under such circumstances are of Randall's Type II, starting as tubular inspissation of calcium phosphate but in prolonged hypovitaminosis severe tubular changes occur. This is probably not a very important factor at the present time in the majority of cases of stone.

The relationship of calculus in patients suffering from peptic ulcer appears well established and is readily attributable to their high intake of milk, alkalis and calcium carbonate with a resultant alkaline urine. There are, however, few statistics bearing on this particular point. Keyser (1943) stressed this fact.

Oxamide and sulphonamide calculi are obvious instances of crystallization due to the hyperexcretory state in which the colloid can no longer hold the excess crystalloid in solution.

Oxalates we have already seen are of importance as producing secondary deposition on pre-existing calcium plaques, especially if hyperoxalæmia and hyperoxaluria are present. They may occur from exogenous sources such as rhubarb or spinach, or from endogenous errors in metabolism. Oxaluria may be due to a disturbed calcium-phosphorus ratio or by incomplete oxidation of carbohydrates.

Calcium phosphorus metabolism is probably the most important factor in stone formation, as calcium figures in all the types of stone under discussion constituting the vast majority. Normal serum calcium is 9·5 to 10·5 mg.%. It is absent from the blood cells and exists as a large non-diffusible fraction combined in the serum and a small diffusible fraction in the ionized state, and varies directly with the pH of the blood, being more ionized in acid. The calcium level is influenced by parathormone, vitamin D, serum protein and the serum phosphorus, with which last it varies inversely. Hypercalcinuria is present in hyperparathyroidism, hypervitaminosis D, uræmia and many diseases of bone, the principal endogenous source of calcium: 70 to 90% is excreted in the fæces and 10 to 30% in the urine. Phosphorus normally ranges from 3 to 4 mg.% in the serum and is increased in hypoparathyroidism, hypervitaminosis D, retention nephritis and fractures. 30% of phosphorus is excreted in the fæces and 70% in the urine. Flocks investigated the relationship of stone to excessive urinary calcium excretion and found that 66% of calculus patients showed hypercalcinuria on a neutral ash diet. Normal calcium excretion is 90 to 150 mg. daily, while stone patients excreted 200 to 450 mg. on similar diet. Vitamin D and acid ash diet increase calcium elimination. An excessive chloride acidosis increases the urinary excretion of calcium and is most marked in the case of ammonium chloride, a practical point as excessive acidosis may damage the renal tubules with resultant calcification.

The metabolism of citric acid has definite relationship to the formation of renal calculi. Important contributions to this subject have been made by Scott, Huggins and Selman (1943) who pointed out that urinary citric acid runs a cyclical course with the reproductive period and that with a normal blood citrate the urinary citrate of patients with calculi was lower than normal, indicating an excessive oxidation in the kidney of citric acid in such subjects. Shorr (1945) made very interesting suggestions for the use of œstrogens and aluminium hydroxide gels in the treatment of renal stone.

Œstrogens increase the citrate excretion in the urine and lower that of the calcium. There is a definite citric acid cycle in metabolism and it is readily convertible into glycogen and glucose, participating in the oxidation of carbohydrate and fatty acids to carbon dioxide and water. Citric acid is present in large quantities in bone, where it is interrelated with calcium. The amount of urinary citric acid is regulated by, first, the urinary hydrogen-ion concentration rising in alkaline and falling in acid urine; secondly, the urinary output of calcium running *pari passu* with it; and thirdly, the sex hormones varying with the menstrual cycle, output being highest at the mid-menstrual period and lowest at menstruation. Œstrogens augment the output of citrate and androgens reduce it. The presence of the citrate-ion influences the ionization of calcium tending to replace the calcium-ion participating in the precipitation of calcium phosphate by the readily ionized soluble calcium-citrate complex, the extent of the effect depending on the relative concentration of calcium nitrate and hydrogen-ions. Below a hydrogen-ion concentration of 7 the reaction is progressively reduced, but even in acid ranges the effect of the citrate complex in removing calcium-ions is still considerable. Addition of citrate would prevent precipitation of calcium phosphate from solution, and this effect is maximal in alkaline solution in which calcium phosphate is least soluble. The effect is less in acid solution in which calcium phosphate is more soluble and requires less citric acid. This is obviously an ideal mechanism for the kidney to prevent precipitation

of calcium phosphate, and it is significant that two conditions favouring precipitation, namely hypercalcinuria and an alkaline urine, are accompanied by increased excretion of citric acid. That this is a purposeful function is supported by investigating patients suffering from renal calculus. It has been shown that in many cases of recurrent renal calculi without infection subnormal amounts of citric acid and excessive amounts of calcium are present in the urine. Further, in such cases smaller amounts of orally administered citric acid are excreted than in normal controls. If infection is superadded two further unfavourable factors may arise, namely a reduction of citric acid present in the urine by the organisms which use it as a metabolite and an increased alkalinity of the urine from the organisms breaking down urea to ammonia. It is obvious that an increase in urinary citric acid excretion might prevent stone formation or check the increase of pre-existing stones. Unfortunately, feeding citric acid is useless, since however much is given by the mouth only very small increases result in the urine. If sodium citrate is given, any increase in urinary citric acid is due to increased alkalinity from the sodium-ion and is offset by the resultant reduced solubility of calcium phosphate in the alkaline urine. Œstrogens, however, increase urinary citric acid without affecting the hydrogen-ion concentration and they also lower the output of calcium. It is possible that the definitely lower incidence of renal calculus in women as opposed to men is due to this fact, the range for citric acid excretion in males lying between 0·4 and 0·7 gramme per twenty-four hours as opposed to 1·5 grammes during the mid-menstrual phase.

A further step to prevent the precipitation of calcium phosphate would be the reduction of the phosphoric-ions, and this can be brought about by administration of aluminium hydroxide gel. This forms the highly insoluble aluminium phosphate in the gut and much reduces the absorption of phosphorus. Thus the plasma phosphorus is low and with it the urinary excretion. In other words, phosphorus excretion is diverted from the kidney to the intestine. It is possible by administering aluminium hydroxide gel in amounts of 120 c.c. during twenty-four hours to reduce the urinary phosphate excretion by 90%, while still maintaining perfect calcium, phosphorus and nitrogen balance. This is especially beneficial when urea-splitting organisms are present producing highly alkaline urine with resultant diminished solubility of calcium phosphate. Acid ash diets and acidifying regimes fail when urea-splitting organisms are present, as the urine in the calices is always alkaline even if acid in the tubules. Further, renal damage is often present, which makes highly acid states dangerous. Even without infection a urinary hydrogen-ion concentration of 4·8 would be continuously necessary to dissolve a stone. Acidifying agents lower the urinary excretion of citrate and increase that of calcium, thus keeping the urine saturated and only capable of dissolving the endogenous calcium phosphate without being able to dissolve any existing calcium phosphate deposits, in other words, to inhibit further stone growth but not to remove stones already present. This helps to explain some of the disappointments in the use of stone-dissolving solutions. To summarize: Shorr suggests that it would seem probable that both œstrogens and amphojel may prove helpful in guarding against recurrent post-operative stones or the development of decubitus calculi.

Treatment

We can appreciate how the facts that have so far been considered have a bearing on the control of the calculus patient, particularly in the field of post-operative care. Further, that preventive treatment has to be initiated at an early stage to have any chance of success. In the case of definite renal calculi, the indications for active surgical intervention are obstruction of the drainage mechanism of the kidney either in the ureter, or renal pelvis or calices, as evidenced by dilatation

shown on intravenous pyelography, with the proviso that there is no likelihood of the stone being passed naturally. Any evidence of failing function in the obstructed part or, more important, suggestion of the onset of infection, necessitates active intervention.

Non-operative treatment is indicated under the following conditions: (1) Limitation of the calculi to the cortical part of the kidney where they have no macroscopic connexion with a calix; (2) mobile stones not causing obstruction and of such a size as to make passage *per vias naturales* possible; (3) stones composed of calcium phosphate or mixed phosphatic stones; (4) bilateral stones associated with gross disease and limited function in both kidneys, rendering any operation hazardous.

Much can be done by forced fluids, by correction of faulty diet and by attempts to control superadded infection. Postural drainage may be very helpful in dislodging a stone. In the case of decubitus calculi or stones consisting wholly or largely of phosphates, attempts may be made to dissolve the calculi by the use of Suby's Solution G, and it is most important to bring this about before secondary infection supervenes, particularly with urea-splitting organisms which render the stones relatively insoluble. The continuous use of Solution G presents practical difficulties, as it necessitates the passage of a ureteric catheter over a long period of time, possibly up to two to three months, any one catheter being changed at intervals not greater than ten to fourteen days. 20 c.c. of Solution G should be injected at three-hourly intervals or some continuous irrigation apparatus used, utilizing up to 3,000 c.c. in twenty-four hours, and the desirability of using two catheters, one for inlet and one for outlet, has to be borne in mind. Sometimes dissolution will proceed satisfactorily and then be arrested owing to the stone having a part composed of some insoluble salt such as oxalate, but nevertheless it may have brought about such a reduction in size as to render the natural passage of the stone possible. Hamer (1944) recorded such a case. Another case of possible difficulty has been pointed out by Keyser. This is a slowing down or cessation of dissolution in Solution G due to the deposition of a mucoid gelatinous covering on the surface of the stone. If a 0·5% solution of urease be substituted for a few hours, this envelope is dissolved and Solution G may again prove effective.

In many cases of gross bilateral calculus disease, it is surprising how little disability the patient experiences. A patient, aged 45, had stones in both kidneys in 1933 and was operated on elsewhere. Now, in spite of gross changes he appears quite fit, has little pain and the blood urea is 43 mg.%. Obviously any operation would be extremely hazardous and could hardly improve the man's condition, while it would almost certainly render it worse.

When we return to a consideration of operative treatment, the first point that arises is the approach to the kidney, and for some fourteen years past I have favoured an incision in the line of the twelfth rib with subperiosteal resection of the rib at the posterior end of the incision. There is nothing novel in this, and I was converted to its merits by Victor Dix following the practice of Von Lichtenberg. However, I do not think its merits have been sufficiently appreciated by the majority of surgeons. Kenelm Digby recently (1941) called attention to this fact. The advantages are as follows: The incision is straight and comparatively short; it is remarkably avascular, it being quite often unnecessary to tie any vessels at all in the parietes; it is atraumatic, as it lies between the eleventh and twelfth nerves and it is as a rule unnecessary to carry the incision far forwards into the flank muscles; it gives a perfect exposure of the upper pole of the kidney, which can be explored without any undue trauma, and if necessary the suprarenal and sympathetic can also be dealt with. The pleura is far less likely to be damaged in a difficult case, and in the event of nephrectomy proving necessary it is the ideal incision. The only

objection is that a second exposure of the kidney may be rendered difficult by regeneration of bone in the site of the rib.

Calculi should always be removed by pyelolithotomy if possible, and it is surprising how, with a proper exposure and suitable curved forceps to enter the calices from the pelvic incision, what a large proportion of stones can be removed in this manner. I prefer an incision in the long axis of the ureter and choose to suture the pelvic incision provided hæmostasis is complete and suture is easy, splinting the suture line with perinephric fat. On the other hand, if the pelvis is thickened and friable it is difficult and unnecessary to suture it, and I have never seen any ill-results accrue from not doing so.

Nephrolithotomy has always seemed to me an undesirable operation, and I must admit to a dread of incisions into the renal substance in order to reach an incarcerated stone. The bloodless line is a figure of speech and the suture of the kidney, even with such aids as ribbon gut and muscle or fat grafts, is not always easy. It is tempting to make the incision into the kidney with the diathermy cutting current, but this is undesirable from the long-term point of view, as it predisposes to further calcium deposition in the incised area. Even if the primary hæmorrhage is satisfactorily controlled, there is always the nightmare of secondary hæmorrhage, and it is particularly in cases where the kidney is grossly infected that nephrolithotomy is undesirable. Many of us must have had the mortifying experience of having to carry out a secondary nephrectomy in a case where a comparatively small incision has been made in the kidney substance. I have always felt that some unnecessary damage must be done to the secreting part of the kidney which is avoided in pyelolithotomy, although Mimpriss (1934) investigated this problem and could find no evidence of renal impairment after splitting the kidney. As opposed to this, Gray (1936) in experimental work on dogs found serious damage in 5 out of 12 cases.

The following case illustrates some of these points:

A woman aged 57 in 1934 had pain in the right flank, and an examination of the urine showed pus cells and *B. proteus*. A pelvic operation had been carried out many years before, and apart from a history of dental sepsis nothing was discovered in the past history. X-rays showed bilateral cortical renal calculi with a stone apparently in the upper right calix. Renal function was excellent and the blood calcium normal. Attempts were made to control the infection in the urinary tract without effect, and in 1935 (fig. 3) the small stone in the right upper calix had grown and filled the right

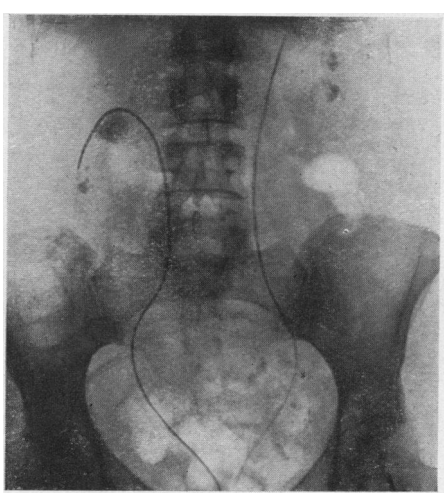

FIG. 3.—Cortical stones with large secondary stone in pelvis.

pelvis, urine from which grew *B. proteus* on culture. I therefore removed this stone by pyelolithotomy, without any difficulty. Inadvisedly I removed the cluster of stones near the lowest calix by

splitting the lower pole to ascertain their nature, and because I was not certain they were all fixed in the renal tissue. All seemed to be proceeding very well until the twelfth day, when she suddenly had profuse hæmorrhage into the wound and into the bladder with high fever. A transfusion and secondary nephrectomy were carried out, the kidney being offensive and necrotic in the area of the incision. Three months later the urine was reported sterile. Four years later, however, she reported with pain in the left loin and a coralline stone in the renal pelvis of the remaining kidney. Pyelolithotomy with avoidance of the cortical stones would have been wisest.

An interesting problem is whether or not to put in a nephrostomy tube after removing a renal stone, and I think on this point there is some difference between American and British practice. I consider that, in cases where dilatation has resulted from the presence of a stone, a nephrostomy tube can do no harm and may do good, and is extremely useful if lavage of the pelvis should be necessary. It is essential that drainage should be from the dependent point of the lowest calix, preferably by Cabot's method. More information is needed on the effects of nephrostomy, and I have been disappointed in the lack of recovery in the kidney which is often seen after the removal of a stone. Probably this is largely a question of the degree of infection and damage to the kidney prior to operation.

The following case is that of a man aged 27 in 1942 who had right renal colic, and in 1943 a stone was removed from the right kidney, following which operation he developed a lumbar abscess which had to be drained and he was considered unfit for further military service. He reported persistent turbidity of the urine with no other symptom. Investigation in February 1943 showed a large stone in the left kidney, with fair function and good function on the right (fig. 4). Urine was sterile but contained pus cells. Renal function was good and there was no leucocytosis. For domestic reasons operation was postponed until September 1943, during which interval he had no symptoms whatever. Operation revealed a calculous pyonephrosis and a branched stone which I removed by pyelolithotomy followed by nephrostomy and closure of the pyelolithotomy wound. Twenty-eight days later, urine from the left kidney was sterile on ureteric catheterization. Six weeks later I removed the nephrostomy tube. Seven months later he was fit, with sterile urine, but little function in the left kidney (fig. 5).

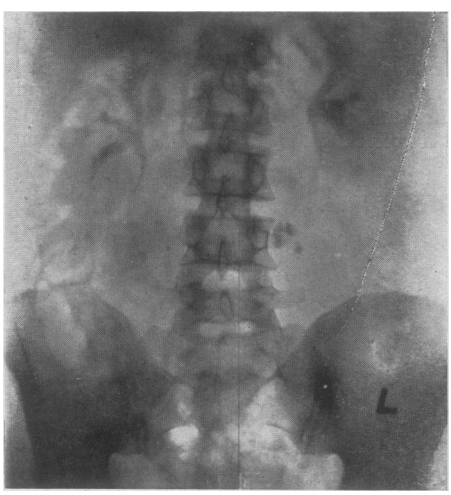

FIG. 4.—Large stone in left kidney. Good function.

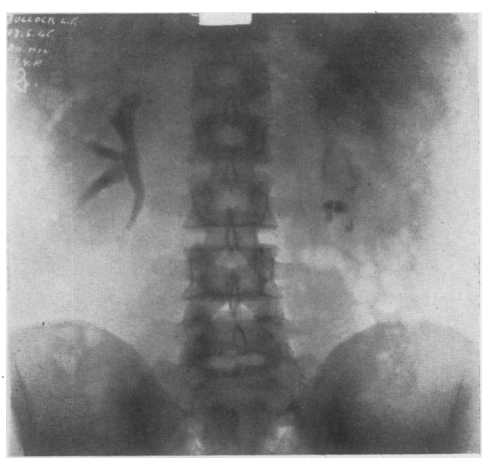

FIG. 5.—Poor function after removal of calculus.

I would emphasize again the value of a nephrostomy tube in enabling the renal pelvis to be irrigated by the two-way method with an indwelling catheter, and all evidence goes to show that this method should give the best results with stone-dissolving solutions.

I have no personal experience of the method advocated by Dees (1946) for avoiding the danger of overlooking small fragments of a branched calculus when per-

forming a pyelolithotomy, namely by utilizing an intrapelvic coagulum of thrombin and human fibrinogen for their extraction.

The following case illustrates a more practical point in pyelolithotomy, the patient being a woman of 40 with left renal colic and hæmaturia. X-ray showed a catheter had passed easily above the stone, which was therefore unlikely to be in the ureter or ureteropelvic junction. Lateral X-ray showed the bougie in front of the stone and a pyelogram indicated a horseshoe kidney, with the stone in the lowest left calix, which was confirmed by intravenous pyelography.

In horseshoe kidney the renal pelvis lies in front and is approached in front of the renal mass in contradistinction to the usual approach from behind. I have found the operation comparatively simple if this point is borne in mind.

Calicectomy or partial nephrectomy may be used, particularly when a ragged cavity remains in the lower pole of the kidney after removal of a stone, in which case recurrence is only too likely. In a clearly incised and accurately approximated area of renal tissue, post-operative hæmorrhage is less likely than in a ragged infected cavity round a calculus.

Cases of bilateral stone require considerable judgment in deciding whether to operate at all or which side to approach first. Most surgeons are opposed to simultaneous bilateral operation, although Hryntschak reported a series of cases in which he had done so without fatality.

Nephrectomy or even nephro-ureterectomy may prove the wisest course, as the infected calculous kidney seldom if ever returns to complete normality. The presence of a dilated pelvis with residual urine in it and fibrosis in and around its walls means the inevitability of urinary infection. When, however, the disease is bilateral, conservative surgery, even if it leaves a second-rate kidney in the end, may be the only possibility. The following case illustrates this point:

A woman aged 45 complained of lumbar pain in the left side of four years' duration. She appeared a poor surgical risk with myocardial degeneration. The renal efficiency was gravely impaired and both kidneys appeared to be equally affected. The urine showed pus and triple phosphates, with a growth of enterococci on culture.

The pre-operative X-ray and the pre-operative retrograde pyelogram showed multiple stones in a dilated renal pelvis. I performed a pyelolithotomy which passed off uneventfully, and the post-operative retrograde pyelogram, two months after, showed appreciable but incomplete recovery.

Ureteric Calculi

The indications for active surgical intervention in the case of ureteric calculus are the same as in the case of renal calculus, that is to say dilatation of the upper urinary tract above the stone, particularly if infection is superadded. If infection is present without dilatation, one should always be suspicious of further disease apart from the presenting stone in the ureter.

If neither dilatation nor infection is present, a conservative attitude can be adopted indefinitely, provided the patient is kept under close observation. Equally well, it is surprising the rate at which an obstructed kidney may become totally destroyed once infection gains a foothold. It is disturbing that these points are not better appreciated even in surgical circles, and many books of reference give an arbitrary time of six weeks for the natural passage of a ureteric calculus, which is utterly meaningless and a most dangerous doctrine.

Let me quote two cases illustrative of these points.

In March 1936 I saw a patient aged 68 who had had his right kidney removed nine years previously for calculous pyonephrosis. Three weeks prior to my seeing him he had had a brisk attack of left renal colic with some vesical irritation. He remained perfectly well with a good urinary output. X-rays showed a stone near the bladder, with good renal function. Six months later, as the stone was still present, I cystoscoped him with some misgiving, as he had a large prostate, and passed a catheter up to the kidney, alongside the stone, tying it in for forty-eight hours. There was no reaction, but it was not until six months later that the stone passed. During the whole of this time he remained perfectly fit and without any evidence of infection.

A man of 36 had a sharp attack of right renal colic, and X-ray investigation showed three stones in the lowest calix. Intravenous pyelography showed a generalized dilatation of the pelvis and calices,

suggesting that the obstructing stone must have dropped back into the lowest calix. Shortly afterwards a second attack of colic developed, and unfortunately, in spite of severe and persistent pain together with fever, he was not sent up to hospital for three weeks. X-ray then showed one of the three stones impacted in the upper ureter and a complete absence of function in an enlarged kidney. When I exposed the kidney, I found it pyonephrotic and disorganized, necessitating nephrectomy.

A point of the greatest practical importance is the site of impaction of ureteric calculi, and in my experience this occurs usually either in the abdominal segment between the second and third lumbar vertebræ or on the pelvic floor in the last two inches of the ureter. Of the two the pelvic position is considerably the commoner.

The prognosis and treatment are very different in the two instances. In the case of a stone impacted in the abdominal segment of the ureter, obstruction to the secretion of urine is usually well marked and for some reason further progress seldom occurs. It is true that most of the cases seen are instances of spiculated oxalate calculi tending to imbed themselves in the ureteric mucosa, but it would appear as though there were some anatomical factor which, having once arrested the stone, makes its further progress unlikely. Some interesting observations on the relative absence of nerves in this part of the ureter have recently been made by Emerson Smith and Strasberg (1942). Spontaneous retreat of a calculus back up to the kidney is also rare, unless the stone happens to have a smooth surface, and particularly in cases where the whole ureter is dilated. Moore (1946) records such a case.

An interesting case illustrating this and other phenomena was that of a soldier aged 25, blown up in a jeep in April 1945. He was unconscious for a week, having sustained a fractured base and in addition injuries necessitating a bilateral above-knee amputation and a suprapubic cystostomy. Both stumps suppurated and his condition was critical. Two months later the suprapubic wound was allowed to heal and, although micturition was free, cystitis persisted. On November 16 he experienced left renal colic with a temperature of 103°, a palpable kidney and *B. coli* in the urine. X-rays seven days later, on November 23 (fig. 6), showed a round stone between the transverse

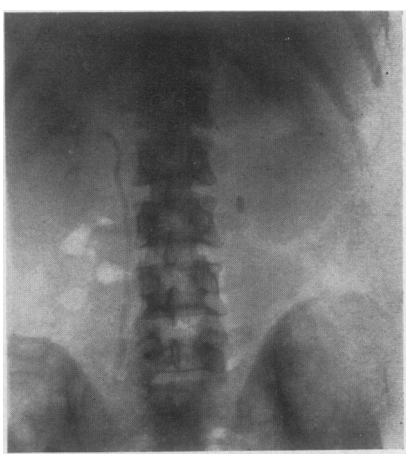

FIG. 6.—Stone in upper ureter.

processes of the second and third lumbar vertebræ, blocking the ureter, which is dilated above and collapsed below the stone, but demonstrates fair function in the kidney. The temperature subsided suddenly with relief of pain, and on November 26, three days later, X-ray (fig. 7) showed that the stone had retreated into the renal pelvis, appearing to have increased slightly in size. The urine now grew *B. lactis aerogenes* on culture. In view of the limb condition, no active surgical treatment was possible. On December 13 he was fit, but X-ray showed the round stone lying in the lowest calix with fair renal function but spasm of the ureter below the site of the original obstruction. On January 10, 1946, X-ray showed the same picture with improved renal function. He remained fit from the kidney point of view until March 8, when he had a further attack of left renal colic associated with fever lasting a week, the urine now growing *B. coli* again. An X-ray at this stage showed that a small oval secondary stone lay in the ureter opposite the transverse process of the

fourth lumbar vertebra and that the original stone had grown considerably, partially filling the renal pelvis. An X-ray on March 22 showed some progress of the ureteric stone and fair function in the kidney without obvious dilatation. On May 7 he passed the ureteric calculus spontaneously and he has remained fit since, but X-ray on July 2 showed a well-defined branched stone which will have to be removed as soon as his general condition permits. Renal function is good on intravenous pyelography (fig. 8), but there is definite dilatation which can also be observed in some degree on the other, sound side.

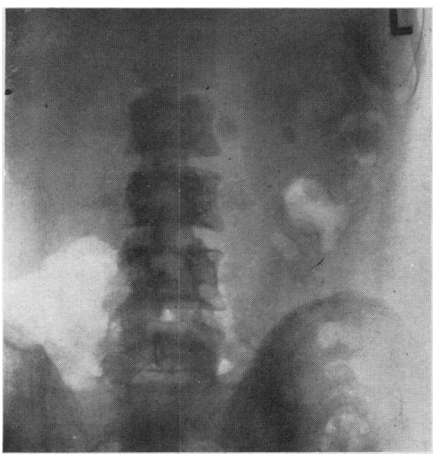

FIG. 7.—Stone passed back into renal pelvis.

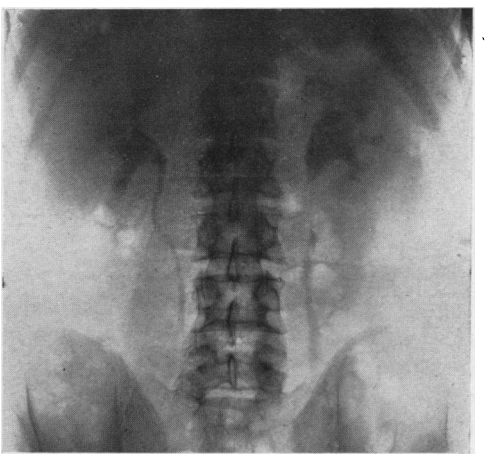

FIG. 8.—Good renal function. Large stone in left renal pelvis.

This case demonstrates first that a smooth ureteric stone can retreat into the renal pelvis, thereby relieving obstruction of the kidney; secondly, the rapid rate at which a renal calculus can grow in the presence of infection and obstruction, and the effects of continued infection on the kidney.

Retreat of a ureteric calculus may sometimes be brought about by preliminary investigations, and this fact emphasizes the importance of X-ray control immediately before or, preferably, if necessary during operation.

A woman aged 44 had left renal colic, and X-ray investigation revealed an oxalate stone blocking the top of the left ureter. A retrograde pyelogram confirmed this (fig. 9), showing a slight leak back above the stone. Some hours later, just prior to my operating upon her (fig. 10), the stone

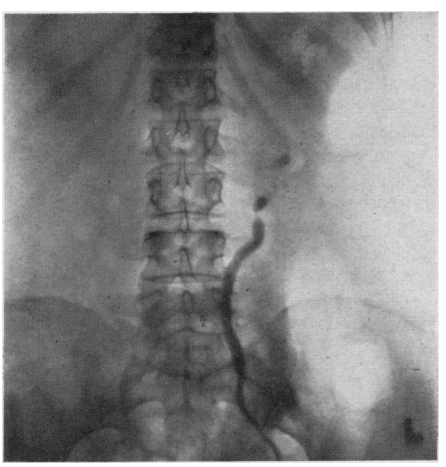

FIG. 9.—Calculus in upper ureter.

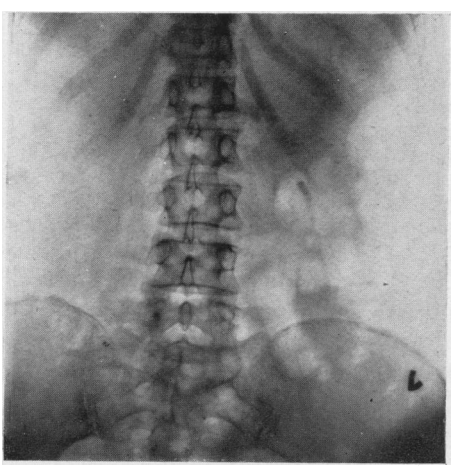

FIG. 10.—Same calculus in lowest calix.

was visible in the lowest calix, from which it was easily removed by pyelolithotomy, but it might have proved difficult to find without the preliminary guide of an X-ray film.

In the case of the pelvic portion of the ureter, spontaneous passage of calculi occurs in a very high proportion of cases—in my experience 90%—although some time may be taken in the process, as in the case I quoted earlier where twelve months elapsed. Retrograde movement may occur, but usually only when hydroureter co-exists, and if planning operation this fact should be borne in mind when tilting the patient on the operating table, as it is possible to roll the stones up and down the ureter by force of gravity. In my experience the necessity for operative interference is lower among the educated sections of the community, probably because they seek medical advice at an earlier stage.

A diagnostic point of interest to be remembered is that the lower end of the ureter may lie in or very near the middle line. The original report in one case was three stones in the bladder, but on cystoscopy only one was visible, which I removed. The patient returned some months later, and subsequently X-ray showed the two remaining shadows in the bladder and considerably increased in size. They were also crushed and removed.

When we consider the actual operative technique for the removal of ureteric calculi, it can be said that in the case of calculi in the abdominal segment any form of cystoscopic removal is inadvisable and dangerous, the problem being one of open surgery. As a rule a short muscle-splitting incision below the kidney will expose the peritoneum, which is displaced forwards revealing the ureter containing the impacted stone. The ureter should be controlled above and below to prevent the possibility of the stone slipping back, and an incision made in the long axis of the ureter over the stone, which is removed. If the incision can be closed with one or two catgut sutures which approximate the edges without transgressing the lumen, I consider this ideal, although it is not essential, as provided that there is no obstruction to the ureter below, the major part of the urine will pass down into the bladder. In my experience post-operative stricture will only occur if the mucosa has been destroyed by pressure ulceration due to the calculus, in which case the kidney will usually have been rendered functionless and require removal. Some surgeons are very much opposed to suture, as they consider free drainage of the kidney essential, and if there is any doubt about the state of the ureter it is probably best to drain it with a T tube round which regeneration may take place, thereby avoiding stricture.

Obviously some œdema of the ureteric wall must occur in the early post-operative period, which may interfere with renal function and it is interesting to note what does take place.

A boy aged 12 was admitted in February 1936 with a history of right renal colic and hæmaturia three weeks previously. The pre-operative X-ray showed a small stone impacted in the abdominal segment of the right ureter with an early hydronephrosis above it, the left side being normal. I removed the stone by lumbar extraperitoneal lithotomy, suturing the ureter. Ten days after the operation, the boy had a sharp attack of abdominal pain most marked on the opposite side. X-ray now showed a considerable increase in the hydronephrosis on the operated side and a definite hydronephrosis on the sound side. The boy had no further pain and he was discharged fit and well on the fifteenth post-operative day. X-rays taken six weeks later showed both kidneys to be normal. Wells (1935) commented on the length of time taken over recovery.

When dealing with calculi in the pelvic segment of the ureter, open operation is very much a last resort. In many cases forced fluids alone suffice, and it is surprising how often cystoscopy is quickly followed by the passage of a stone. This happens so frequently that it cannot be mere coincidence, and it may be due to the dilatation of the posterior urethra leading to a reflex relaxation of the intramural part of the ureter. This occurs without the patient being aware of it, and very often the stone is voided unwittingly, which indicates how desirable it is for all urine to be passed under conditions where the expected calculus can be searched

for. It is surprising that it is not better appreciated that the pains associated with ureteric calculi are due to distension of an obstructed renal pelvis above and not to the onward passage of a stone, which is painless and can only occur when the ureter is relaxed. Of the instrumental methods, I have found the use of an indwelling ureteric catheter passed alongside the stone and kept in situ for forty-eight hours the most satisfactory method. If the Howard corkscrew or the Councill umbrella extractor is used, it is always wise to insert an indwelling ureteric catheter to counteract the effects of reactionary œdema. Personally I confess to being very apprehensive of their use, although I am well aware of the spectacular results that have been achieved. Wishard (1943) reported some unfortunate experiences with the basket extractor, and Dourmashkin (1945), reporting on a most exhaustive study of the cystoscopic treatment of 1,550 cases of ureteric calculus, advised against the use of forcible mechanical extraction. Councill himself (1945) recorded three instances of rupture of the ureter in a series of 504 cases. Actual meatotomy of the ureteric orifice is seldom necessary and is better avoided because of the risks of troublesome hæmorrhage and, more important, ureteric incompetence with vesico-renal reflux. If instrumentation fails, operation is called for and I have used both the mid-line and inguinal approaches. Of these the inguinal is simpler and quicker provided that the stone is not too low, in which case the mid-line approach is essential. It is accepted as axiomatic that the approach should always be extraperitoneal, but I would suggest that this is incorrect and that under certain circumstances transperitoneal approach may have great advantages, particularly in women.

On one occasion I set out to remove a stone from the lower end of the ureter by a mid-line extraperitoneal exposure, and found extreme difficulty in obtaining any adequate approach. It became obvious that a pelvic tumour was present, rendering the identification of the ureter impossible. I therefore opened the peritoneum and found a bilateral pyosalpinx, undoubtedly the underlying cause of the urinary condition, for which I carried out a bilateral salpingectomy. As a result, the ureter with the contained stone was well exposed, enabling me to open it and remove the calculus. Wishing to ensure free drainage from the kidney and to avoid extravasation of urine, I passed the fluted end of a ureteric catheter through the incision in the ureter up to the renal pelvis, subsequently passing the lower end down the ureter and through the ureteric orifice into the bladder in which the redundant part coiled up. I then sutured the ureter over the catheter and passed a stab extraperitoneal drain down to the suture line through a fresh incision in the flank. The peritoneum was then sutured over the ureter and the abdomen closed. Four days later I cystoscoped the patient and withdrew the catheter with duckbill forceps. I was so impressed with the smoothness of this patient's convalescence that I have repeated this procedure in several cases where it appeared desirable, one of which I reported in 1938.

In those cases of ureteric calculus where infection exists in the obstructed kidney behind the stone (and, according to Randall, that is in roughly 50% of cases) I have often observed a brisk flare in clinical cystitis after removal of the stone. Appreciating this fact, it is wise to culture the kidney urine and give a post-operative course of sulphonamide, if necessary.

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ABSTRACT.—There are many problems concerning the ætiology, diagnosis and treatment of stone which are still unsolved. The exact relationship between Randall's type I calculus formed as a papillary plaque and therefore liable to be passed naturally at an early stage of development and the type II formed as an intratubular deposition is obscure. It is possible that they are both due to the response of the kidney to toxins, the exact lesion depending on the intensity, frequency and length of time the organ is subjected to their influence. A variety of factors contribute to stone formation, such as the hyperexcretory state with an upset of the colloid crystalloid balance in the urine, recumbency with immobilization, infection, climatic conditions and disordered metabolism. Calcium and phosphorus metabolism and their disorders are vitally concerned in calculus formation. Recent work on citric acid metabolism and its alterations due to hormonic influence point the way to efficient prophylaxis. Œstrogens increase the excretion of citrate in the urine and lower that of calcium. Addition of citrate prevents precipitation of calcium phosphate from solution, an effect which is maximal in alkaline solution in which calcium phosphate is least soluble. This is an ideal mechanism for the kidney to prevent the precipitation of calcium phosphate. Reduction of phosphate ions would enhance this effect and such reduction may be produced by the administration of aluminium hydroxide gel which forms insoluble aluminium phosphate in the gut.

Treatment of calculi may be non-operative or surgical. Obstruction and infection call for prompt surgical relief. Non-operative treatment can be advised under the following conditions: (1) Limitation of calculi to the cortical part of the kidney. (2) Mobile stones of such a size as to render natural passage possible and not causing obstruction. (3) Stones composed of calcium phosphate or mixed phosphates and therefore capable of spontaneous or therapeutic solution. (4) Bilateral stones associated with limited function and disease affecting both kidneys, rendering operation hazardous.

Forced fluids, the correction of faulty diet and the control of infection may be of great assistance. Postural drainage should be tried and in addition in the case of phosphatic stones, Suby's solution G.

When operation becomes necessary, an exposure of the kidney through an incision in the line of the twelfth rib with resection of that structure may prove extremely helpful. Pyelolithotomy is the preferable method of removing stones and in most cases this method is practicable with adequate exposure. Incisions into the cortical part of the kidney should be avoided unless absolutely essential to effect removal of a calculus. Nephrostomy is worthy of more extended trial but in unilateral cases with gross infection nephrectomy or even nephro-ureterectomy may prove the best treatment.

Most ureteric calculi are eventually voided naturally although cystoscopic manipulation may be necessary if stones are retained in the pelvic portion of the ureter. Mechanical extractors should be used with considerable caution. Operative technique should be varied to suit the individual case and illustrative cases demonstrating these points are described.

In all cases post-operative follow-up and suitable prophylaxis are most important if improvement in recurrence rate is to be brought about.

RÉSUMÉ.—Beaucoup de problèmes concernant l'étiologie, le diagnostic et le traitement de la lithiase restent à résoudre. Les relations précises entre le calcul du type I de Randall, formé comme plaque papillaire, et tendant par conséquent à être expulsé par les voies naturelles à un stage précoce de son développement, et le type II, formé comme dépot intratubulaire, sont obscures. Tous deux sont peut-être dus à une réaction du rein à l'action de toxines, la forme précise de la lésion dépendant de l'intensité, de la fréquence et de la durée de leur influence sur l'organe. Plusieurs facteurs contribuent à la formation des calculs, par exemple l'état hyperexcrétoire, accompagné d'un dérangement de l'équilibre colloïde crystalloïde de l'urine, la position couchée avec immobilisation, les infections,

les conditions météorologiques et les troubles du métabolisme. Les métabolismes du calcium et du phosphore et leurs dérangements sont de toute première importance pour la formation des calculs. Des travaux récents sur le métabolisme de l'acide citrique et ses variations sous l'influence des hormones indiquent le chemin vers une prophylaxie effective. Les œstrogènes augmentent l'excrétion urinaire des citrates et diminuent celle du calcium. L'addition de citrates empèche la précipitation du phosphate de calcium d'une solution. Cet effet est au maximum en solution alcaline, où la solubilité du phosphate de calcium est au minimum. Ce mécanisme est idéal pour le rein pour empêcher la précipitation du phosphate de calcium. La réduction des ions phosphate augmenterait cet effet, et une telle réduction est possible par l'administration d'hydrate d'aluminium colloïde, qui produit du phosphate d'aluminium insoluble dans l'intestin.

Le traitement de la lithiase peut être opératoire ou non. L'obstruction et l'infection réclament l'intervention chirurgicale prompte. Un traitement non-opératoire peut être recommandé dans les conditions suivantes: 1° Calculs limités à la partie corticale du rein, 2° Calculs mobiles de grandeur adaptée à l'expulsion par les voies naturelles, et ne produisant pas d'obstruction. 3° Calculs composés de phosphate de calcium ou d'une mélange de phosphates, et par conséquent capables d'être dissous spontanément ou par un traitement médicamenteux. 4° Calculs bilatéraux avec fonction rénale réduite et maladie atteignant les deux reins, ce qui rend l'opération dangereuse.

Les liquides forcés, la correction d'un régime défectueux et les mesures anti-infectieuses peuvent beaucoup aider. Le drainage postural doit être essayé et, en plus, dans les cas de calculs phosphatiques, la solution G de Suby.

Quand l'opération devient nécessaire, la mise à nu du rein par une incision parallèle à la 12e côte, avec résection de cet os, peut être extrêmement utile. La pyélo-lithotomie, qui est possible dans la plupart des cas si la mise à jour est suffisante, est la meilleure opération pour l'extraction des calculs. Les incisions de la partie corticale doivent être évitées à moins qu'elles ne soient absolument nécessaires pour enlever un calcul. Il vaudrait la peine de continuer les expériences de la néphrostomie, mais pour les cas unilatéraux la néphrectomie ou même la néphro-urétérectomie peut être le meilleur traitement.

La plupart des calculs urétéraux finissent par être expulsés par les voies naturelles, mais une manœuvre cystoscopique peut devenir nécessaire si les calculs sont retenus dans l'uretère pelvien. L'emploi des extracteurs mécaniques doit être accompagné de grandes précautions. La technique opératoire doit être variée selon les cas. Cet avis est illustré par la description de quelques cas.

Dans tous les cas l'observation post-opératoire et un traitement prophylactique approprié sont essentiels pour réduire la proportion de récidives.

SUMARIO.—Aun existen para aclarar muchos problemas de la etiologia, el diagnóstico y el tratamiento de la litiasis. Es obscura la relación exacta entre el calculo tipo I de Randall, que se forma como una placa papilar y por consiguiente es sujeto a ser evacuado por la naturaleza tempranamente en su producción, y el tipo II que se forma como un depósito intratubular. Es posible que ambos resultan de la reaccion del riñon a toxinas, la lesión exacta dependiendo de la intensidad, la frequencia y la duración del tiempo que el órgano esta expuesto a su influencia. Una variedad de factores contribuyen a la formación del calculo, como el estado hiperexcretorio acompañado por un trastorno del equilibrio coloide cristaloide en la orina, la inmovilización en cama, una infección, condiciónes climáticas y desordenes metabolicos. El metabolismo del calcio y del fósforo y sus desordenes son de primera importancia en la formacion del calculo. Estudios de origen reciente sobre el metabolismo del acido cítrico y las alteraciónes que causan en este las hormonas indican el camino a una profilaxis eficaz. Los oestrogenes aumentan la excreción de citrato en la orina y disminuyen la de calcio. La añadidura de citrato impede la precipitacion de calcio fosforado de la solución, un efecto que es máximo en una solucion alcalina en laque el calcio fosforado es el mínimo soluble. Esto ofrece al riñon un mecanismo ideal para prevenir la precipitación del calcio fosforado. La reducción de los iones de fosforo aumentaria este efecto y tal reducción puede ser producida por la administración del hidróxido de aluminio gel que forma en el intestino el aluminio fosforado insoluble. El tratamiento de casos de calculo puede ser terapeútico o quirúrgico. Una obstrucción o una infección exige la intervención quirúrgica inmediata. El tratamiento terapeútico es aconsejado sobre las condiciónes siguientes: (1) Cuando los calculos son limitados a la parte cortical del riñon. (2) Cuando los calculos son movibles y de tal tamaño que es possible evacuarlos naturalmente y que no causan una obstrucción. (3) Calculos del fosforado de calcio o de fosforados mezclados por esto capables de solución espontánea o terapeútica. (4) Calculos bilaterales acompañados de una limitacion de función y una condición patológica en los dos riñones, que haria arriesgada una operación.

La receta de liquidos en gran cantidad, la rectificación de alimentos defectuosos y la restricción de infecciónes pueden ser de gran ayuda. Se debe probar el drenaje postural y tambien además la solucion G de Suby en los casos con calculos fosforados.

Cuando una operación es necessaria, es muy provechoso exponer el riñon por una incisión en la linea de la costilla duodécima con resección desta estructura. Pielolitotomía es el método preferible para sacar los calculos y en la mayoría de casos este método es practicable si se emplea una exposición adecuada. Se deben evitar las incisiones en la parte cortical del riñon si no son absolutamente

esencial para sacar el calculo. Es justo probar la nefrostomia en mas casos pero en casos en que un riñon solo esta afectado y la infección es densa la nefrectomía o mismo la nefro-ureterectomía puede salir el mejor tratamiento.

La mayoría de los calculos uretericos se evacúan naturalmente con el tiempo aunque una manipulacion cistoscopica puede ser necesaria si los calculos son retenidos en el uréter pélvico. Extractores mecánicos se deben emplear con una cautela considerable. Se debe variar la técnica de operación segun cada caso individuo. Se incluyen en este estudio casos ilustrativos.

Es de primer importancia en todos los casos examinarlos de vez en cuando despues del tratamiento quirúrgico estar completo y exigir una profilaxia apropiada si una mejoría en la proporcion de reincidencias va ser efectuada.

АВСТРАКТ.—Многие проблемы, которые касаются этнологии, диагноза и лечения камней почек до сих пор еще не разрешены. Точная зависимость между первым типом камней (по Рандаллю), которые образуются ввиде папиллярной пляки и потому могут быть выделены естественным путем в раннем периоде образования и вторым типом камней, которые образуются ввиде интратубулярного отложения, — вовсе не ясна. Возможно, что оба типа являются следствием влияния токсинов на почку и точное поражение почки зависит от тяжести, частоты и продолжения их действия на орган. Множество факторов предрасполагает к образованию камней, как например, гиперэкстреторное состояние с нарушением коллоидо-кристаллоидного равновесие в моче, лежачее положение с иммобилизацией, инфекция, климат или нарушенный метаболизм. Метаболизм кальция и фосфора и нарушение этого метаболизма являются весьма важным фактором в образовании камней. Самые недавние работы над метаболизмом лимонной кислоты и его изменением под влиянием гормонов, указывают на путь к успешной профилактике. Острогены увеличивают выделение цитратов в моче и уменьшают выделение кальция. Прибавление цитратов предотвращает осадок фосфата кальция из раствора, что происходит в высшей степени в щелочном растворе, в котором фосфат кальция хуже всего растворяется. Для предупреждения осадка фосфата кальция, это является идеальным механизмом для почки. Редукция фосфатных ионов увеличит этот эффект и такую редукцию можно произвести применением жела гидроокиси алюминия, который образует нерастворимый фосфат алюминия в кишках.

Лечение камней может быть нехургическим и хирургическим. Обструкция и инфекция требуют немедленного хирургического вмешательства. Нехургическое лечение необходимо при следующих условиях:—

(1) Присутствие камня в кортикальной части почки.
(2) Подвижные камни такого размера, что их естественный проход возможен и не произведет обструкции.
(3) Камни фосфата кальция или смешанных фосфатов с восможностью самопроизвольного или терапевтического растворения.
(4) Двухсторонние камни, связанные с ограниченной функцией и болезнь обоих почек, при которой операция слишком рискованна.

Большое количество жидкостей, исправление неправильной диэты и контроль инфекции — весьма важны. Постуральный дренаж должен быть испробован, а также раствор „G"Суби при фосфатных камнях.

Когда операция становится необходимой, обнажение почки через надрез по линии 12-го ребла с его резекцией — может быть весьма полезным. Пиэолитотомия — более предпочтительный метод удаления камней и в большинстве случаев он возможен при достаточном обнажении почки. Нужно избегать надрезов кортикальной части почки, за исключением тех случаев, когда это необходимо для удаления камней. Нефростомия должна быть испробована более часто, но при односторонних случаях с сильной инфекцией, нефректомия или даже нефро-уретеректомия, является лучшим способом.

Большинство камней мочеточника обыкновенно выделяются естественным путем, хотя цитоскопическая манипуляция может быть нужна в тех случаях, когда камни задержаны в тазовой части мочеточника. Механические экстракторы должны быть применяемы с большой осторожностью. Операционная техника должна быть приноровлена к индивидуальным случаям. Автор здесь приводит иллюстрации для указания таких случаев.

Все болные должны быть подвергнуты наблюдению после операции. Для избежания рецидивов весьма важна соответственная профилактика.