STONE IN THE URINARY TRACT

SECOND EDITION

WINSBURY WHITE

BUTTERWORTHS MEDICAL PUBLICATIONS

STONE

IN THE

URINARY TRACT

SECOND EDITION

H. P. WINSBURY-WHITE

M.B., Ch.B. Ed., F.R.C.S. Ed., F.R.C.S. Eng.

HUNTERIAN PROFESSOR 1925 AND 1933, ROYAL COLLEGE OF SURGEONS OF ENGLAND; SENIOR SURGEON AT ST. PETER'S HOSPITAL AND ST. PAUL'S HOSPITAL FOR UROLOGICAL DISEASES, LONDON; UROLOGIST TO THE LEWISHAM GROUP OF HOSPITALS, ST. CHARLES HOSPITAL, THE ITALIAN HOSPITAL AND QUEEN ELIZABETH HOSPITAL FOR CHILDREN; CO-FOUNDER, FORMERLY EDITOR (TWENTY YEARS), NOW CONSULTING EDITOR OF THE *BRITISH JOURNAL OF UROLOGY*; EX-PRESIDENT, SECTION OF UROLOGY, ROYAL SOCIETY OF MEDICINE; GUITERAS LECTURER, AMERICAN UROLOGICAL ASSOCIATION, 1936; HONORARY MEMBER OF THE AMERICAN UROLOGICAL ASSOCIATION.

LONDON
BUTTERWORTH & CO. (PUBLISHERS) LTD.
1954

STONE
IN THE
URINARY TRACT

AFRICA: BUTTERWORTH & CO. (AFRICA) LTD.
DURBAN: GOODRICKE'S BUILDINGS, MASONIC GROVE

AUSTRALIA: BUTTERWORTH & CO. (AUSTRALIA) LTD.
SYDNEY: 8 O'CONNELL STREET
MELBOURNE: 430 BOURKE STREET
BRISBANE: 240 QUEEN STREET

CANADA: BUTTERWORTH & CO. (CANADA) LTD.
TORONTO: 137 DANFORTH AVENUE

NEW ZEALAND: BUTTERWORTH & CO. (AUSTRALIA) LTD.
WELLINGTON: 49/51 BALLANCE STREET
AUCKLAND: 35 HIGH STREET

PRINTED AND BOUND IN GREAT BRITAIN BY
LOVE AND MALCOMSON LTD., REDHILL, SURREY

PREFACE TO SECOND EDITION

THIS second edition has long been overdue, if for no other reason than the author's dissatisfaction from the beginning with the first edition. Many events have conspired to postpone the re-presentation in a more acceptable form of the original work, which has been now entirely re-written. The passage of so much time involved in the fulfilment of this task has offered at least the advantages of intervening experiences, and the author has tried to reflect these in this edition.

Eight hundred and sixty-six personal cases of urinary calculus are the foundation upon which this volume is built. Aetiological factors continue to be a fascinating study, for they are numerous and appear to vary according to changing conditions of life; this is suggested by the increased age incidence in upper urinary tract lithiasis.

All these points are discussed, but more light is needed for a full understanding.

It is the hope of the author that some readers may find at least a modicum of enlightenment in perusing data which have given him so much instruction in compilation.

H. P. WINSBURY-WHITE

London
December, 1953

PREFACE TO FIRST EDITION

THERE is no disease affecting the urinary tract which offers a more varied succession of interest than lithiasis. A subject of these wide dimensions can never be dealt with adequately within such space as is usually allotted to it as a part of a work on general surgery. Even in its place in a urological text-book, the requirements which necessitate that an orderly proportion of the whole should be maintained, make brevity essential where more detail would be an advantage. In dealing with the topic as it is now presented there are no such restrictions, with the consequence that the subject-matter occupies considerably more space than is customarily allotted to it. The author feels this is done with every justification, and hopes that his endeavours fulfil a useful purpose.

As the work proceeded it became apparent that if a fairly clear limit was not defined, a much more extensive work would be evolved than was originally intended. In reaching its ultimate proportions, a middle course was set between brevity and prolixity, in the hope of producing a volume which would give detail attractively presented in a concise form.

Those who seek more information than is given in the text, are referred to the extensive bibliography on urinary calculi at the conclusion of the volume. This covers a period of eighteen years, and includes all the important literature on the subject from 1910 to 1928.

The work is profusely illustrated because this was felt to be the simplest and best method of emphasis. The specimens represented are mostly to be found in the Museum of the Royal College of Surgeons of England or the Museum of St. Paul's Hospital. The great majority of the latter were removed at operation by the author.

A chapter has been devoted to children, first of all to accentuate the fact that the disease does occur in early life, and secondly because it is well to bring forward those features which are peculiar to childhood.

Much more could have been profitably said with regard to pregnancy. For the present sufficient data have been brought forward to show the frequent association of calculous disease and this condition. It must be left to a future occasion, however, to do full justice to this important relationship.

Grateful acknowledgement is expressed to the Royal College of Surgeons of England for the opportunity offered of recording many valuable specimens, most of which are of exceptional interest. The radiograms are the work of Dr. Mather Cordiner, Mr. Critchell McDuff, and in a few instances, Dr. Ulysses Williams. The author wishes to record his indebtedness to these gentlemen.

All the drawings are by Mr. Thornton Shiells, the high standard of whose workmanship is apparent.

H. P. WINSBURY-WHITE

London

CONTENTS

CHAPTER 1

AETIOLOGY OF URINARY CALCULI

WHEN we consider urinary calculus as it occurs amongst us at the present time we are struck with the different form which this disease takes compared with the incidence of it up to the end of the last century. For until that time this complaint was mostly found in the form of vesical calculus in children of the working classes. Our realization, that this type of case is practically non-existent in Great Britain to-day, gives us some interesting material upon which to reflect.

Anyone not conversant with the facts might wonder whether this benefit is the result of an organized campaign against vesical calculus. I need hardly say that this is not the case. The change has been a slow one and has merely gone hand-in-hand with the improving standard of living in the lower strata of the population.

DIETARY AND OTHER FACTORS

It has fallen to the lot of the research workers in the past 30 years gradually to fit together the successive pieces of evidence which have made it abundantly clear that the fault was dietetic. One may say that diet has always been a matter for great consideration in connexion with attempts to reduce the incidence of urinary calculus. The old conception, however, that the presence of stone depended largely on the ingestion of the chemical substances of which the stone is composed, has now faded into insignificance.

Let us consider two of the commonest components of urinary calculus, namely calcium oxalate and uric acid. The discovery, that oxalate and calcium occurred in numerous food-stuffs which composed our daily diet, seemed to suggest that control of the tendency to the formation of oxalate stone was only a matter of excluding these substances from the daily food. In like manner the fact that uric acid was an end-product of protein metabolism pointed to the conclusion that this form of lithiasis would be equally easy to eliminate by diminishing the ingestion of protein. But because a strict control of diet seemed to have no appreciable effect on stone-formation, the fact that both oxalate and uric acid resulted also from chemical processes within the body began to claim attention. Indeed, in time it came to be realized that uric acid stones were found quite commonly amongst people whose diet never included any protein, but was always of a simple and monotonous carbohydrate nature.

It can be seen how strongly these facts called for a serious revision of the views formerly held concerning the relationship of diet and urinary calculus. Support was no doubt given to these erroneous views by the misinterpretation of certain experimental data. For instance, it was found that, as a result of the extensive feeding of animals with oxamide (diamido-oxalic acid) over a prolonged period, oxalic stone was produced. These experiments were carried out many years ago (Ebstein & Nicolaier, 1891), and the conception of the production of oxalate stone in this manner has been revived and demonstrated experimentally from time to

S. in U.T.—1

time since then, more recently by Keyser in 1932. There is a fundamental weakness, however, in this theory. It is that it still has to be demonstrated that the high concentration of oxalic food, which was necessary to produce lithiasis in these animal experiments, can be taken and maintained for sufficiently long periods to give rise to stone in human beings. Moreover, oxalate stones can be produced by feeding animals on an oxalate-free diet. On the other hand, the presence of oxalate in the food does result in the formation of oxalate stone when there is a deficiency of either calcium or magnesium in the diet. These facts were demonstrated by Greta Hammarsten (1937).

It is now generally held that, when the incidence of stone has a relationship to diet, it is to do with deficiency, rather than excess, of food constituents. Deficiency of vitamin A of animal origin and a deficiency of absorbable calcium in the diet, independently of the vitamin lack, have been amply proved to be important faults; another factor has been shown to be some constituent of the whole-meal of cereals when taken under certain conditions (see page 4). Lack of other vitamins is a less important contributory cause. It is from the work of McCarrison (1931) in India that so much evidence on these matters has been forthcoming.

The maximal incidence of this disease today in the upper urinary tract is in the beginning of the fifth decade of life (*see under* "Age", page 23). With the knowledge that the cases due to proved food faults occurred largely in children in the lower urinary tract, we seem to be in need of aetiological factors other than those already proved, to explain our present-day cases. The fact that stone resulting from food faults, when produced experimentally, occurs in the bladder many weeks before it is found in the kidneys, indicates a common cause for the clinical cases, in so far as they occur in children, as well as for the experimental cases.

The astonishing "stone wave" of central Europe, which apparently started about 1924 and was still continuing in 1938, most likely expressed a widespread change in the conditions of life in that part of the world, but what those essential factors are has yet to be discovered. That the fault is dietetic is highly probable, but that it is different from those errors which have already been identified is equally likely, because the form of the disease has altered to renal from vesical and to the fourth decade of life from childhood.

Hellström (1949) has recently commented on this phenomenon in relation to Sweden where, because of the existence of carefully composed vital statistics, and of the completeness of information provided in the annual reviews of hospitals, he has had special opportunities of observing this. He remarks that the increase in kidney and ureter stone, which set in after the First World War, made the incidence 10 times greater than that recorded in 1914. He is inclined to attribute the increase to changes in the nutritive condition of the people.

We must not lose sight of the fact that because of the obstructive conditions at the bladder neck which tend to develop with advancing years, vesical calculus occurring in men after middle life has always been, and continues to be, a feature of urinary lithiasis. But this form of disease has also somewhat diminished because of the large number of cases in which the obstruction is removed by surgical means.

In considering generally the causes of urinary lithiasis, full attention must be given to the fact that, either directly or indirectly, there are many factors which

may play a part in precipitating this disease. They may be conveniently grouped under the following headings: (1) Heredity; (2) race; (3) food faults; (4) geographical and climatic influences; (5) chronic renal disease associated with changes in the parathyroid glands; (6) inflammation; (7) bone lesions, recumbency and irradiation; (8) calcium metabolism; (9) situation, sex and age; (10) dilatation and other abnormalities such as injuries and foreign bodies in the urinary tract; (11) medication; (12) predisposing kidney lesions. (For Randall's theory *see* page 49 and for the influence of colloids, *see* page 32.)

Heredity

There are numerous instances quoted in the literature in which several generations of the same family have been afflicted. More interesting still are examples in which brothers and sisters, widely scattered in different countries, have all developed calculi.

Race

The Negro, in contrast with other dark-skinned races, is outstanding in his freedom from lithiasis. This applies to all parts of the world, even when he lives in the " stone areas ". It has not been shown that any other group of people enjoys such a degree of immunity, which, however, seems to be lost to some extent where his blood is mingled with that of Europeans. An investigation into the question of the immunity of the Negro in relation to urolithiasis at a hospital in Virginia, United States of America, by Reaser (1935), made it clear that urolithiasis is uncommon amongst American Negroes and that when it occurs it is at an advanced age. Vermooten (1941) calls attention to the absence of urinary calculus in the African Negro.

Food Faults

Without the conception that the aetiology of urinary calculus does not depend upon one factor alone, a proper view of this important subject is not possible. Such an outlook applies particularly to the roles of vitamin A and of absorbable calcium. For instance, there is a good deal of evidence in support of the view that a deficiency of these factors in the diet produces vesical calculus in children—the commonest form of urinary lithiasis at one time in Great Britain, although no longer so—while there is a lack of evidence to support the view that these food faults play any part in producing renal calculus in adults as it occurs amongst us today. There seems no doubt that a failure by some observers to recognize these two distinct pathological types has led to wrong conclusions in making investigations into the aetiology of urinary stone.

Deficiency of vitamin A

The first important proof of the influence of diet on the incidence of lithiasis was not put forward until 1917, when Osborne and Mendel found that rats fed on diets deficient in fat-soluble vitamins frequently developed stones. In 1927 McCarrison, as a result of his work in India with rats, corroborated the fact that deficiency of vitamin A was an important factor in stone-formation, but he showed that other factors also played a part. He fed rats on the diets of those peoples of

India amongst whom stone was common, and it was discovered that other factors were involved as well. He came to the conclusion that the cereal foods ranged themselves in the following descending order of stone-producing potency: whole wheat flour, oatmeal, Indian millet, white flour, rice, and South-Indian millet. By making certain additions to the stone-producing diets he found that lithiasis could be prevented (McCarrison, 1927). It was discovered that milk, butter and cod-liver oil prevented stone and that vegetable oils did not.

Influence of hard water

In order to determine whether or not hard water had any influence on stone-formation, McCarrison added calcium to the diets deficient, amongst other things, in vitamin A. The result was not only an increase in the incidence of stone, but cystitis, hydronephrosis and pyonephrosis were increased as well, showing that stone is only one of the diseases which result from faulty nutrition. He also found that the lack of sufficient phosphates to combine with the calcium present, increased the incidence of stone. Grossmann (1933), from his own experiments, also came to the conclusion that a disproportion in diet between calcium and phosphorus predisposes to stone-formation.

McCarrison (1931) summed up his conclusions in the following words:

> "There appear therefore, to be two categories of dietetic factors concerning stone formation: (a) *Positive factors*, including excess of lime in the diet, and some unknown agent present in whole cereal grains; and (b) *negative factors*, including deficiency of vitamin A derived from animal sources and deficiency of phosphates relative to the amount of lime in the diet. I say 'Vitamin A derived from animal sources' because millions of people in India are dependent for their supply of this factor—whatever that factor implies—on vegetables; I am not yet certain that the latter source of vitamin A is, from the point of view of stone prevention, as good as the former. I do not think it is, and I notice that de Langen (1929) of the Dutch Indies is of a like opinion."

The full extent of the pathological processes which take place in the body as a result of a lack of vitamin A, and which lead ultimately to urinary lithiasis, has not as yet been properly defined, but it is clear in considering the reports of several reliable observers that stone is only one of a number of consequences of this deficiency. One result is degenerative change in the epithelial structures throughout the body; these involve the urinary tract to a marked degree, and there seems no doubt that desquamated epithelium often forms the nucleus of the stone. The question as to whether infection always precedes the stone-formation is a point which still has to be settled. It certainly is found together with the lithiasis in a large number of the experimental cases.

The work of Grossmann (1933) threw more light on the actual processes at work in the urinary tract which lead finally to stone in feeding rats on a diet free of vitamin A.

> These experiments involved the use of 30 series of rats, with 12 in each series. At the end of 3 weeks on the diet mentioned, the hydrogen ion concentration varied from 7·0 to 8·0 and rose even as high as 9·0. Accompanying this change was first the appearance, and then the steady increase in the number, of red blood cells in the urine, until a recognizable haematuria was established, while by this time a definite pyuria was also present. In nearly all the animals a urinary infection was established

at the end of 60–70 days. Most commonly this was due to *Staphylococcus albus*, less often to streptococci and diplococci, occasionally haemolytic streptococci and coliform bacilli, or a mixed infection of coliform bacilli and other organisms. On one occasion *Bacillus proteus* was present.

Changes in the urinary tract were noted first in the bladder itself after the first month and in the kidneys after the second month. These could be classified into three groups: (1) Hyperplasia and metaplasis of the lining epithelium; (2) inflammation; and (3) calculous deposits in the urinary passages. The rapidly proliferating epithelium soon became desquamated, many clumps of which became encrusted with lime salts. All stages of inflammation were found from a slight hyperaemia to a marked necrosis. Haemorrhagic cysto-pyelitis, cortical abscess in the kidney, and pyonephrosis were different forms of inflammation noted. These conditions were the common causes of death in animals which were not killed.

Macroscopic sand, which could be regarded as calculi, appeared in the bladder in the fifth week, but never before the eightieth day in the kidney. Very rarely were fully developed renal calculi found without vesical calculi. Close examination of the clumps of desquamated epithelium revealed deposits of calcium. When inflammation was already present, the calculous deposit was found on the debris of inflammation as well. Grossmann verified the conclusions of other experimenters, that rats fed on a normal diet do not develop stone. He found with his experiments that all the changes, which he described as occurring in the urinary tract, took place earlier if an excess of calcium was added to the diet.

Criticism has been directed against the experiments, which set out to show that a specific deficiency of diet may be responsible for urinary calculi, on the ground that the diets employed were deficient not only in vitamin A but also in other vitamins. The experiments of Bliss, Livermore and Prather (1933) are free from such defects and appear to substantiate the generally accepted conclusion that deficiency of vitamin A alone can induce the formation of urinary calculi in the rat.

At once arises the question of how we are to apply our knowledge of vitamins in a practical way. It would be erroneous and misleading to suggest that whole-wheat flour and oatmeal, when they compose the major part of an unbalanced diet, are stone-producing and therefore should be displaced from the dietaries of mankind. They need never be regarded as anything less than desirable constituents of a mixed diet when present in moderate proportions. McCarrison (1931) says: " No single food is in itself a 'complete food', even milk is not; if it be not faulty in one regard it is faulty in another. . . . Milk, as an exclusive food, will cause anaemia in rats; onions, anaemia in dogs; cabbage, goitre in rabbits; and oatmeal or atta will cause stone in rats; yet what better food can there be for mankind than a judicious mixture of all four ? "

As for excess of lime and deficiency of phosphates, an ordinary mixed diet, as known to modern civilization, provides the proper balance of these constituents. The imbalance of these substances becomes a source of danger only in the presence of other stone-forming factors.

In the light of the conclusions of modern research, much becomes clear on the occurrence of lithiasis in the old world. Thus we see why it is that the decline in the incidence of stone, in western Europe and in certain parts of Great Britain, has gone hand-in-hand with the substitution of white flour for whole-wheat flour, which at one time formed the principal article of diet for the masses. We

also see that milk and butter, if used in sufficient quantity, afford complete protection against calculous disease in rats. These facts, indeed, throw a full flood of light on the incidence of vesical calculus in Great Britain in former days; for while it was common in certain parts, yet there were adjacent localities which were particularly free from the complaint. It was, in fact, in the counties where the best pasture land was found that stone was not prevalent; for in these districts milk and butter—both rich in vitamin A and absorbable calcium—were commonly available. South-Western England and Ireland were conspicuous in this way.

Knowing what a relative abundance of vitamin A there is in the dietary of all classes at the present time compared with the days when vesical calculus was so common, it is difficult to believe that the absence of this constituent is still the dominating cause of stone-formation.

Higgins (1936, *a* and *b*) and others believe that vitamin A deficiency is a cause of the renal lithiasis which we see clinically today. These workers have demonstrated, by means of the biphotometer test, that a deficiency of vitamin A is common in cases of renal calculus—a state which was found to be unalterable after a prolonged course of vitamin A therapy. These conclusions were broadly confirmed by Long and Pyrah (1939). A number of other observers, however, have examined many patients with lithiasis and have failed to find a lack of vitamin A in them. Moreover a study of the diets of calculous patients made it clear that there was no lack in this respect.

Further light has been thrown on this subject by Moore (1937), from material obtained from 950 cases at autopsy. He found that, when death followed disease, the vitamin A reserves were lower than in material obtained after death from injury, and that the lowest ranges of all were encountered in diseases of the urinary system.

One may sum up the position with regard to tests carried out on clinical cases to discover a relationship between a lack of vitamin A in the food and renal calculus by saying that they have failed to show that there is any connexion between the two. What we must not lose sight of, however, is that in many diseased states, including renal lithiasis, the patients are largely unable to maintain their vitamin A reserves, and that this state of affairs cannot be altered by merely increasing vitamin A in the diet. It is possible, in these circumstances, that this deficiency, when it occurs as a secondary event in a pathological process, does play some part in the aetiology of kidney stone.

GEOGRAPHICAL AND CLIMATIC INFLUENCES

These have been responsible for a variety of opinions usually unsupported by scientific data. Climates with dry or moist heat have each in their turn been held responsible for the prevalence of urinary lithiasis.

Climatic Influence

The difficulty in assuming that atmospheric conditions have a direct influence are seen from the following facts. In Ecuador and the valley of the Amazon the atmosphere is hot and humid and there is no lithiasis. In South Africa the climate is hot and dry and there is no lithiasis. In Peru the climate is hot and dry and there is plenty of stone.

Having considered food faults, it is easy to see how geography and climate can play their part in an indirect way in relation to these. In some parts of the East children develop vesical calculus while still at the breast. In the so-called dry belts of the world, such as Northern Africa, Arabia, Persia, Mesopotamia and northern India, stone abounds. Because vegetation is scarce, meat, milk and butter are scanty, but the animals which do exist there depend on green fodder for vitamin A. Moreover, the dry food upon which they so largely live is rich in calcium and phosphates. It must at once be clear how all of these factors are contributory to stone-formation, and small wonder is it that lithiasis is common, even amongst the animals themselves. So far as the inhabitants are concerned, it is obvious that they must depend mostly on cereals for their existence.

Geographical Location in Relation to Food Supplies and Drinking-Water

We know that, even in productive and cultivated lands, the amount of vitamin A present in certain foods has a definite variation according to the season of the year. For example, in milk in the winter, it is less than one-half of what it is in the summer, while in eggs, towards the end of the winter it is only one-half of the summer level (Higgins, 1936b). It is possible that these facts play their part in encouraging urinary lithiasis, even in fertile lands, and especially where drinking water is of an excessive hardness due to the high content of calcium.

The improved standard of living of the working classes throughout the country has undoubtedly wiped out the " stone areas " from Great Britain today (Eastern Counties, Westmorland, parts of Derbyshire and North Wales were the localities in question). The part played by water-supply in urinary calculus under bad dietetic conditions is well illustrated in the fact that, many years ago, the disease diminished very considerably in South Staffordshire, where a softer water than was formerly used was obtained by sinking wells to a deeper level than had been previously reached. McCarrison's experiments have made it clear that it is in the presence of vitamin deficiency that excess of lime in the water is a factor.

It is wise to bear this important combination in mind, for we have examples in which hard water has no deleterious effects with regard to lithiasis; for example, in Switzerland the water is hard but lithiasis is relatively rare. In Holland the water is soft but there is plenty of lithiasis.

It is of great interest to reflect that in Norfolk—which one might with justification describe as the chief " stone " county of these islands during the previous century—certain local medical practitioners knew perfectly well why stone was so common in their county. For example, in 1828 William England (quoted by Thomas, 1949) wrote a paper in which he commented on the following facts: (1) The rarity of stone in Ireland, where the people lived on vegetables, potatoes and milk; (2) the scarcity of stone in Scotland, where the diet consisted of oatmeal, milk cheese, vegetables and fish; and (3) its prevalence in Norfolk, where the diet consisted of bread, water, tea and very little milk—the last because pasture land was poor. This author rightly concluded that deficiencies in diet were the cause of stone.

In 1874 Cadge (quoted by Thomas, 1949) took the same line, and considered that the lack of milk was an important factor.

That all these valuable data, presented to the profession so long ago, should have lain buried in the literature, and practically unknown to our own and, perhaps, the preceding generation, is but another example of the way in which a precious discovery can sometimes be forgotten.

Thomas goes on to show how, as the agricultural work of the country changed to mixed farming, and later the children got their daily milk, as well as a good mid-day meal at school, so vesical calculus in children (which was formerly the prevalent form of the disease) entirely disappeared from the county.

Switzerland and Normandy are two other places where the inhabitants have access to plenty of milk and its by-products, and where lithiasis is rare (Justin-Besançon and Lamotte-Barbillon, 1948).

Urinary lithiasis and bilharziasis

In lower Egypt there is an additional aetiological factor, for here urinary lithiasis and Bilharzia infestation go hand in hand; but, as evidence that the parasitic disease is not the sole cause, we find it prevalent elsewhere, especially in other parts of Africa, where stone is not often found. The variation in the relationship of bilharziasis to urinary lithiasis, according to locality, is doubtless to be explained by the differences in the habits of the people concerned, chiefly in relation to diet. Several observers have made careful investigations of the nuclei of calculi which have occurred in association with Bilharzia, and as a rule ova have not been discovered, although debris, which is obviously the product of ulcerative inflammation in the urinary tract, have been readily identified. The parasite, after entering the host through the digestive tract, is found in numbers in the venous spaces of the liver, in the portal vein and its tributaries, and in the vesicoprostatic plexus. The ova pass through the vessel walls and cause ulceration of the bladder and lower portion of the ureter.

Increased geographical incidence of unknown origin

There has been a remarkable increase in the incidence of lithiasis in Central Europe from 1924 onwards. Germany, Austria, Hungary, Czecho-Slovakia and Southern Poland are the countries where this has occurred. Grossmann (1938) has communicated some striking facts with regard to this phenomenon. He says that the type of stone is usually oxalate, it forms in the upper urinary tract, it is generally passed spontaneously *per urethram*, it does not tend to recur, and in some localities the increase in the incidence has been as high as 1,000 per cent. While he is at a loss for an explanation of this extraordinary change, he is of the opinion that the incidence is too long after the First World War to be explained by a faulty diet during that period.

There is some reason to believe that the same tendency, to a lesser extent, has occurred in Great Britain as well. In support of the general impression that stone in the upper urinary tract is on the increase, the figures of Lett (1936), which cover a study of 2,781 cases of urinary calculus from the London Hospital over a period of 30 years (1905–1934), prove it to be actually the case. These show that in the 1925–1934 decade there was actually an increase of nearly 50 per cent over the preceding 10 years in the cases of lithiasis admitted (*see* Table I). This increase was predominantly in the upper urinary tract.