STONE DISEASE IN PARAPLEGIA*

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STONE disease has been known for as long as the human race has existed. An abundance of literature has been accumulated but, despite all our endeavours, the causes of the disease are not yet entirely clear.

In the case of the paraplegic patient, stone disease was regarded for a long time as an almost inevitable corollary to the loss of neurological function. If present nowadays, it still is regarded in the long run as a main killer of the paraplegic patient. The serious prognosis of this disease and our most encouraging experiences in avoiding its onset prompted me to report on our observations at Southport over a period of fifteen years.

It would go beyond the scope of this communication to report on all the past and current theories of stone disease. I shall limit myself, therefore, to mentioning briefly the basic principles underlying various types of stone formation.

TABLE I

Principles of Stone Formation

1. *Nucleus*

(*a*) Intrarenal: Randall's plaque.
Carr's pouch.
Keratinised epithelium in hypo-vitaminosis A.

(*b*) Extrarenal: Lack of calcium solvents, colloid-crystalloid imbalance.
Products of infection.
Foreign bodies.

2. *Growth of Stone*

Excessive calcium in urine and alkalinity from:

(*a*) Dehydration. (*b*) Recumbency. (*c*) Peptic ulcer. (*d*) Hypervitaminosis D. (*e*) Hyperparathyroidism. (*f*) Renal acidosis. (*g*) Idiopathic hypercalciuria. (*h*) Infection with urea-splitters.

3. *Impaired Elimination*

(*a*) Oliguria.

(*b*) Stasis: Recumbency.
Congenital and acquired obstructive anomalies.

As can be seen from Table I, all theories of stone formation have three points in common:

1. There must be a centre of growth, a so-called nucleus, around which the mineral matter precipitates.
2. There must be an excessive concentration of salts in urine and a suitable reaction to make the stone grow.
3. There must be a tendency to retention.

* Read before the International Medical Society of Paraplegia on 27 July 1962.

1. Location of Stone. The nucleus is formed either inside the renal tissue, or in the collecting apparatus.

Randall (1937) was the originator of the first conception. He has shown that the initial calcification, the so-called Randall's plaque, is formed in the basement membrane of the renal tubule, or beneath the epithelium of the renal papilla. Later the separating lining becomes eroded and the stone finds its way into the renal calyx. Carr (1954) believes that there is a normal mechanism of precipitation and back-absorption of salts in the kidney which breaks down in the event of blocked lymphatic vessels. Hypervitaminosis A produces keratinising metaplasia and desquamation of epithelium which may serve as a nucleus.

Other authors believe that the primary nucleus forms in the urinary passageways. Lack of 'urinary solvents' like citrate radicle, organic acids or protective colloids may be the cause of precipitation of salts from a super-saturated solution. Infection may produce nuclei in the form of clumps of organisms, necrotic epithelium, clots, etc. Lastly, foreign bodies in the urinary system are known to become encrusted with salts in a very short time.

2. Growth of Stone. Crystallisation of salts around the nucleus is speeded up by excessive concentration of calcium in urine and by its alkaline reaction. Concentration may arise from easily understandable causes such as dehydration due to a hot and dry environment, pyrexia or vomiting, or from more complicated causes such as metabolic disturbances. Recumbency is known to cause decalcification of bones and hypercalciuria. Hypercalciuria may also be produced by hyperchlorhydria and intake of absorbable alkali or excessive consumption of milk in peptic ulcer. Hypercalciuria is known to occur in hypervitaminosis D due to mobilisation of calcium from the bone, for instance when lupus is treated with excessive dose of Calciferol.

As to the endocrine causes, hyper-parathyroidism is not uncommon. It brings about continuous mobilisation of bone salts and increased excretion of calcium and phosphorus in urine. Renal acidosis is extremely rare; the renal tubules are unable to form ammonia or excrete acid urine, leading to hyperchloraemic acidosis on one hand and hypercalciuria and alkaline urine on the other. There remains also a group of patients with excessive calcium excretion of unknown origin and, by exclusion, this group is labelled idiopathic hypercalciuria.

Infection with urea splitters such as *B. proteus* contributes considerably to the fast growth of stone.

3. Impaired Elimination. No comments are needed to explain that oliguria renders washing away of concretions difficult.

Stasis may be due either to recumbency in the supine position or obstruction and dilatation resulting from congenital or acquired anomalies. As to the former, the main feature is that stones develop in the dorsally and cranially directed calyces. It is obvious that nature intended us to be upright for two-thirds of the twenty-four hours. Figures 1, 2 and 3 demonstrate a typical example of recumbency stones in a patient who was confined to bed for many years.

Special Features of Paraplegia. In paraplegia, many of these causative factors are present such as:

(*a*) Concentration of urine.
(*b*) Stasis.
(*c*) Infection.
(*d*) ? Specific factor(s), metabolic or of neurogenic origin. This has not yet been established conclusively.

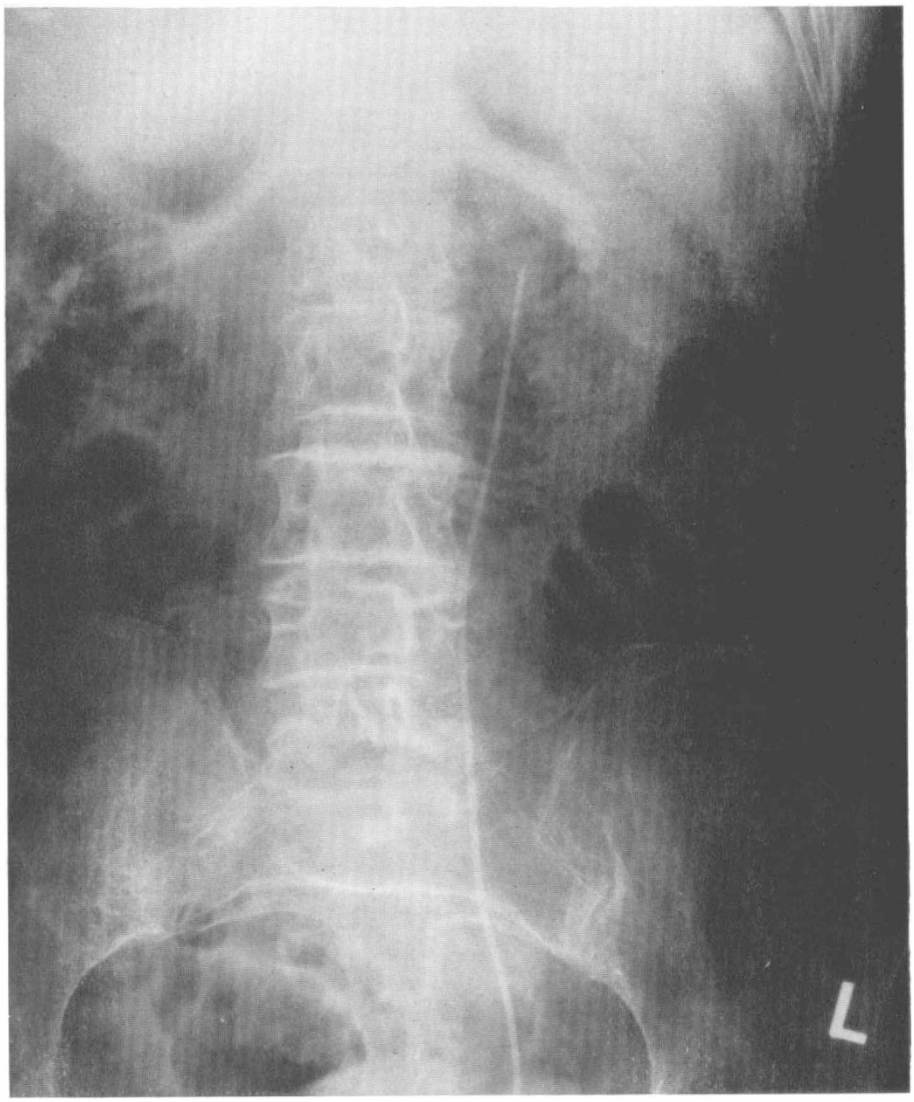

FIG. 1

This figure shows a film preliminary to retrograde pyelography. One can see a large shadow probably in the upper calyx of the left kidney and also a tiny one in the lower calyx. The right kidney is obscured by gas; it is suspected that the patient has also a stone in the upper calyx of the right kidney.

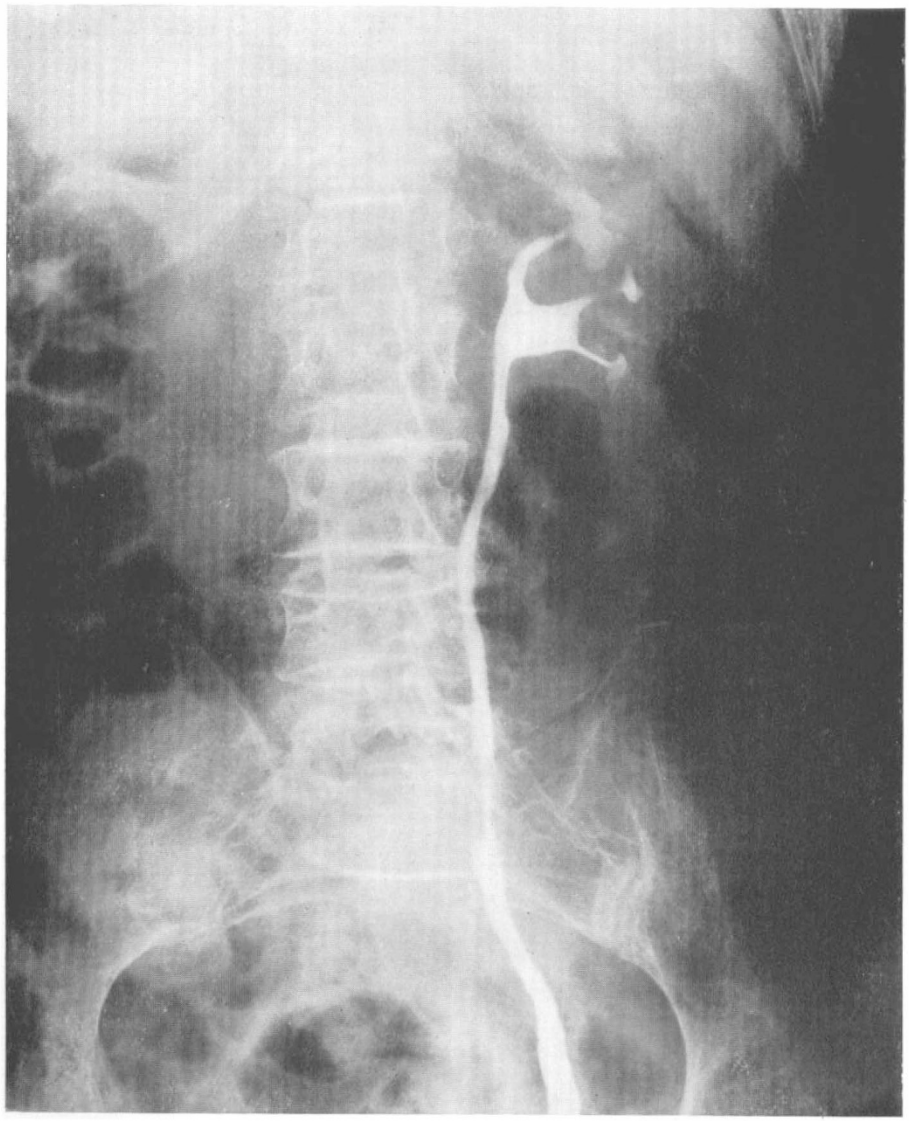

FIG. 2

This figure shows a left retrograde pyelogram to establish the exact location of the stones. There is no doubt that the large primary stone fills the upper calyx.

Prophylaxis. With these principles in mind, we have applied with success preventive treatment as follows:

1. Early mobilisation and exercises to avoid decalcification of the skeleton, hypercalciuria and stasis.
2. Urinary acidifiers and high-protein diet to avoid alkalinity.
3. High fluid intake to produce polyuria and wash away nuclei.

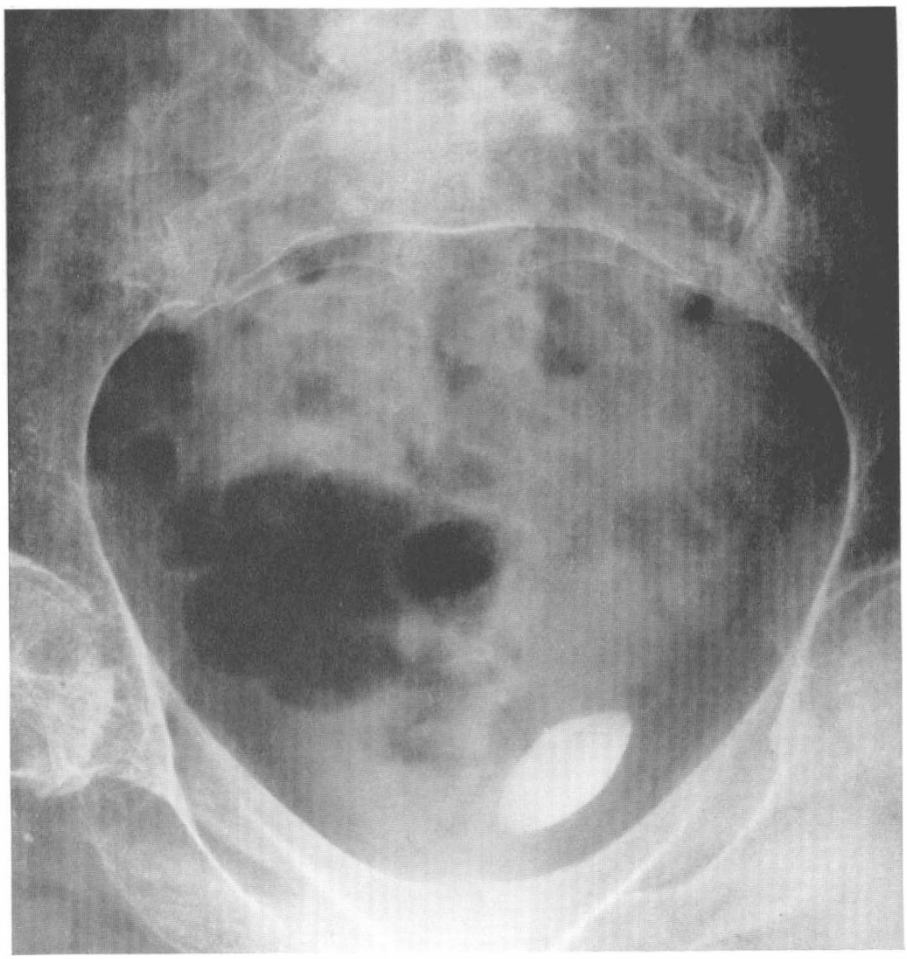

FIG. 3

This figure shows the urinary bladder of the same patient. Note that there was also a large stone in the bladder.

4. Efficient catheter drainage at first and elimination of residual urine and catheter in the later stages to do away with stasis and infection.

Incidence. The Southport Centre commenced in January 1947 when the first batch of patients were admitted from general hospitals. Figure 4 shows the number of patients admitted, the white column, and of those affected by stone, the black column.

In 1947, we had 17 admissions and 8 patients affected by stone, the incidence being 47 per cent. In 1948 the incidence was 28 per cent., in 1949 it fell to 20 per cent. and in 1950 it rose slightly to 25 per cent.

In 1951 we began to admit patients after a very short stay in a general hospital, at times almost direct from the site of the accident. This policy has proved a

great success in all respects. In 1951, 1952, 1953 and 1954 there were no stones. In 1955 our figure was spoiled by two patients: one of them was admitted with a stone 16 months after his spinal injury, the other after 6 months. In 1956 there were no stones. In 1957 we had two cases out of 19 admissions: one patient was admitted 5 months after injury and the other after 14 days, the latter with a severe urinary infection and frequent pyrexial bouts causing delay in early mobilisation. In 1959 there was one patient affected by stone. He was admitted to this Centre on the very day of his injury, but had numerous and severe concomitant injuries, fracture of the molar bone and of four ribs with haemothorax, injury to the brachial plexus and dislocation of a hip. It is obvious that the routine prophylactic measures could not be observed in his case. Although he was admitted to Southport almost immediately after his injury, he showed a stone in a calyx two months after his admission.

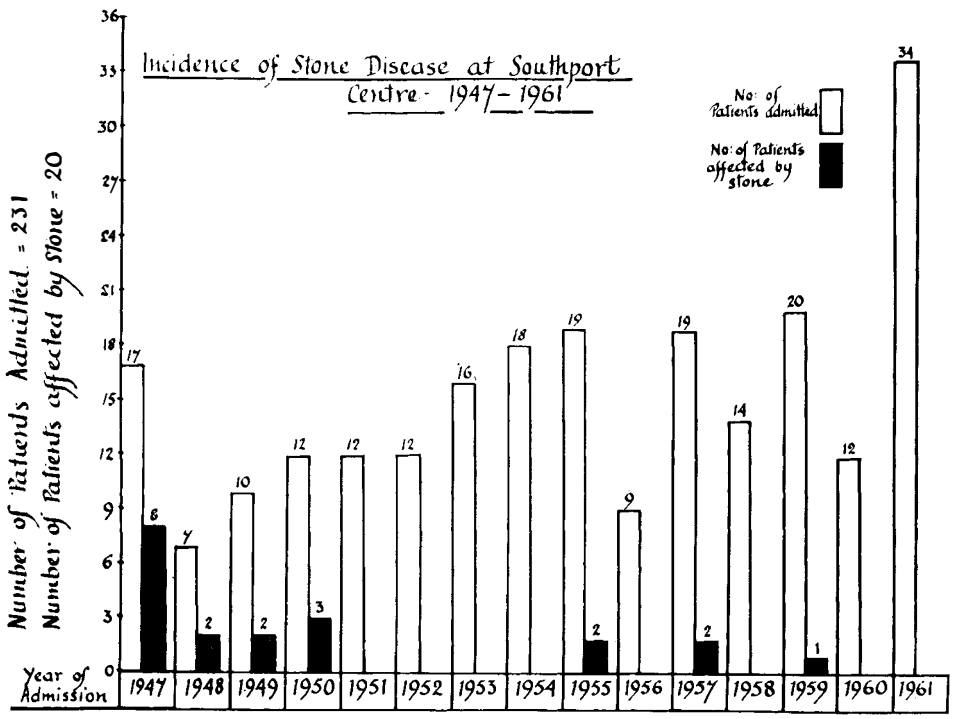

FIG. 4

There were no stone cases in 1960 or 1961. And in 1962, only recently, we admitted from her home an elderly female patient with multiple recumbency stones (figs. 1 to 3).

Onset of Stone Disease. In view of the fact that some of the patients had been admitted to the Centre late or very late after the spinal injury, it was not always possible to establish when they developed the stone. Nevertheless, it is my impression that the stone formation begins in the early stage of paraplegia.

Chemical Composition of Stone. All stones chemically examined consisted of calcium magnesium ammonium phosphate. They were often poorly calcified and formed a soft cast of the calyces and pelvis. This presents a difficulty in diagnosis as it may not show on X-ray.

Laterality of Stone. Figure 5 shows the kidney affected by stone. In nine cases the right side was affected, in eight cases the left one, in three cases the disease was bilateral.

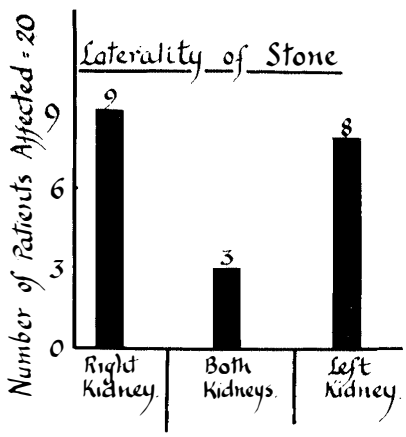

FIG. 5

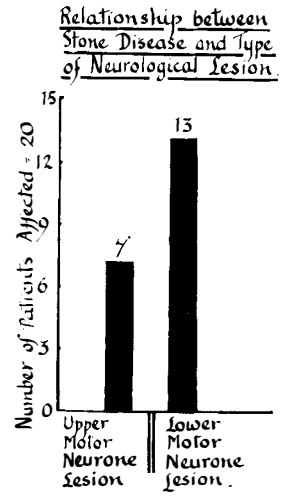

FIG. 6

Stone and Type of Neurological Lesion. Figure 6 shows the relationship between the stone disease and the type of neurological lesion.

Of the 20 affected patients, 7 were upper motor neurone lesions and 13 lower motor neurone lesions. This is more or less in line with the general distribution of lesions in our Centre. Eighteen patients were neurologically complete and 2 patients incomplete, the latter being cervical lesions.

Mortality. Out of the 20 affected patients, 12 are dead from uraemia. The time of their survival is set out in Figure 7. Of the 8 surviving patients, one had a nephrectomy 18 years ago: he still remains in good health and is full-time employed.

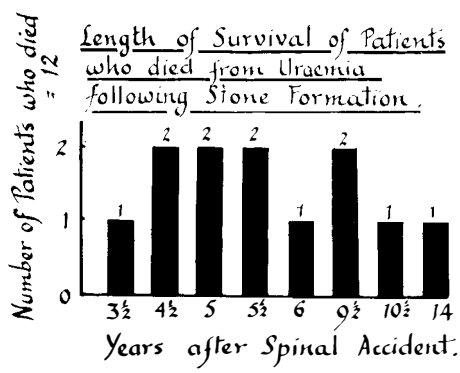

FIG. 7

Bladder Stones. All the above figures apply to the upper urinary tracts. As to the urinary bladder we had 26 patients with stones, many displaying them in the upper tracts as well. It is my strong impression that in many cases the stones formed around the débris which had fallen off the old-fashioned de Pezzer and Malecot catheters or the more modern Foleys. I have not seen a single bladder stone after use of a Gibbon catheter.

CONCLUSIONS

The report from our Centre tends to show that stone formation in paraplegia has fallen from a formidable proportion to a negligible figure. This happy result has been achieved by special measures designed to provide prophylaxis. I must, however, add a word of warning: when a stone has developed in the kidney in paraplegia, the long-term prognosis is not good.

RÉSUMÉ

Ce rapport de notre centre tent a demontrer que l'incidence de la formation de calculs chez les paraplegiques est tombee a un chiffre negligable.

Cet heureux resultat est du a des mesurer propylactiques speciales cependant, quand un calcul se forme au niveau du rein chez un paraplegique, le pronostic a long terme est assombrit.

ZUSAMMERFASSUNG

Der Bericht-lässt die Tendenz eines Absinkens der Steinbildung bei Paraplegie von einem hohen Prozensatz zu einer unbedeutenden Zahl erkennen. Dieses erfreuliche Resultat ist durch spezielle prophylaktische Massnahmen erzielt worden. Es muss allerdings eine Warnung angefuegt werden: Wenn es erst einmal zu Steinbildung bei der Paraplegie gekommen ist, ist die Spaetprognose nicht gut.

REFERENCES

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