PROSTATECTOMY

BRITISH MEDICAL JOURNAL 547

cases where a major operation was accepted as a calculated risk and a fatal outcome was not unexpected by patient, doctor, or relatives. However, perusal of the case histories provides no evidence of any serious previous disease necessitating treatment in hospital or by their own doctor in 10 cases. Although most of

TABLE VII.—Previous History of Those Who Died After Prostatectomy

	Total	Transferred from or Previously Treated in Medical Ward
Severe cardiac lesions Hemiplegia Severe bronchitis Nothing of note	93	5
	4 10	2

them had some degree of arteriosclerosis they were apparently fit men who were "killed by the operation." As stated above, four of these deaths resulted from coronary thrombosis, but it is interesting to note that, of the other six, four died of renal failure and two from "causes unknown." Furthermore, although 44% of all the cases admitted with retention had been catheterized before admission, 17 (81%) of the 21 deaths after prostatectomy for retention had previously been catheterized (often repeatedly), including this particular group of six.

## **Summary and Conclusions**

A review is presented of 197 cases of prostatic disease admitted to the surgical wards of a small provincial hospital over the five-year period 1955-9, with, in particular, a consideration of the fatalities.

Prostatectomy for prostatic disease without retention has a low mortality, and earlier operation for milder degrees of prostatism, before the serious consequences of the disease are manifest, would seem to be the only way to reduce materially the overall mortality.

Too many cases of prostatic disease to-day present with retention, and although a two-stage operation is often the best treatment for these cases this method carries a higher mortality and morbidity rate than does the one-stage prostatectomy. More cases would be fit for one-stage prostatectomy if referred to hospital sooner than is the custom now.

Any operative procedure in the older patient must entail risk, and if, as a general principle, surgical treatment aims at a return to normal physiological function as opposed to merely preserving life, then additional risk and a higher mortality must be accepted-not only by the patient (who is usually only too willing to accept the risk) but also by the surgeon. Having accepted this calculated risk any method of reducing it must be searched for, and study of the cases in this series suggests two possible approaches. Firstly, the education of the public and the general practitioner regarding the dangers of the use of a catheter except in a hospital theatre. Secondly, the organization of the hospital services so that these cases can be dealt with as emergencies with bacteriologist, biochemist, etc., available as easily as is surgeon, physician, and anaesthetist.

In essence, this paper is a plea for the establishment of a specialized urological service. Although it is realized that general surgeons, such as myself, will miss the joy of having the survivor telling him that "I feel 20 years younger," there would be more survivors if these cases were treated in the equivalent of the teaching hospital of Lee et al. (1957).

It is a pleasure to thank Dr. E. L. Wilson, my medical colleague, for his invaluable help in many of these cases; also Dr. J. E. Morison for his guidance in the pathological reports; and Sister Martin and her nurses, whose care has often meant the difference between success and failure. I am indebted to a succession of house-surgeons (now scattered all over the world) on whom so much of the postoperative care devolved, especially the enthusiastic young lady house-surgeon who counted the 97 stones in the bladder. My registrar during this period, Dr. R. Stinson, has been a most helpful assistant and has had most of the responsibility for the day-to-day treatment. Finally, I wish to thank my secretary, Miss M. Acheson, for her assistance in collecting the records necessary for the production of this review.

REFERENCES Badenoch, A. W. (1953). In Modern Trends in Urology, edited by E. W. Riches, p. 264. Butterworth, London. Bomford, W. B. N. (1959). Lancet, 2, 262. Caine, M. (1954). Brit. J. Urol., 26, 205. Ellis, H., and Leatherdale, R. A. L. (1958). Lancet, 2, 1189. Hey, W. H. (1945). Brit. J. Surg., 33, 41 Johnston, J. H. (1953). Brit. J. Urol., 25, 155. Lee, J. A. H., Morrison, S. L., and Morris, J. N. (1957). Lancet, 2, 785. McEachern. A. C. (1959). Am. P. C. W. G.

785.
 McEachern, A. C. (1958). Ann. Roy. Coll. Surg. Engl., 22, 151.
 Millin, T. (1953). In Modern Trends in Urology, edited by E. W. Riches, p. 285. Butterworth, London.
 Riches, E. W. (1950). In British Surgical Practice, 7, 165. Butterworth, London.
 Ross, J. C., and Tinckler, L. F. (1959). Brit. med J., 2, 663.
 Wells, C. (1953). In Modern Trends in Urology, edited by E. W. Riches, p. 301. Butterworth, London.
 White, E. P., and Berry, N. E. (1950). Canad. med. Ass. J., 62, 556.
 Young H. H. (1945). J. Urol. (Baltimore) 53, 188.

Young, H. H. (1945). J. Urol. (Baltimore), 53, 188.

# URINARY INFECTION IN CYSTOSCOPY

#### BY

## BERNARD LYTTON,\* M.B., F.R.C.S.

Formerly Senior Surgical Registrar, London Hospital

There is a good deal of evidence that one of the major factors in the aetiology of infections of the urinary tract is urethral instrumentation (Beeson, 1955-6). Gillespie et al. (1960) have shown the part played by catheterization and instrumentation in the production of urinary infections after prostatectomy. They have clearly indicated that by the proper sterilization of cystoscopes and the adoption of a closed system of catheter drainage the incidence of post-prostatectomy infections fell from 80% to 6%. Marple (1941) and Slade and Linton (1960) have investigated the role of catheterization in female patients in producing urinary infections; and Slade and Linton advocate that specimens of urine from female patients should be obtained by collecting a mid-stream specimen and not by catheterization.

It was the purpose of this investigation to determine to what extent routine cystoscopic examination, which is so often performed as an out-patient procedure, resulted in urinary infections.

### Methods

The study was performed on a consecutive series of male patients who underwent a cystoscopic examination in the urological out-patient department of the London The majority of them had been treated Hospital. previously for a papilloma of the bladder and attended for follow-up examinations. Their ages varied from 33 to 75.

\*Now at Department of Surgery, King's College Hospital Medical School, London.

A specimen of bladder urine was collected through the cystoscope and examination of the centrifuged deposit for leucocytes and organisms was performed. The deposit was also cultured for bacteria. A midstream specimen of urine was collected four days later and was examined in the same way.

The procedure of cystoscopy and the method of sterilization of cystoscopes employed in the urological department of the London Hospital are described, as these vary greatly in different clinics. The cystoscopes are sterilized by boiling in distilled water for five minutes. The telescopes are not boiled, but are sterilized by immersing them vertically in a solution of equal parts of 1:2,500 chlorhexidine (" hibitane ") and 1:1,000 oxycyanide of mercury (Miller et al., 1958). The irrigating solution consists of 1:5,000 chlorhexidine. The patients are examined in the lithotomy position in a cystoscopy chair and no sterile towels are used. The glans penis is swabbed with saline and the urethra is anaesthetized by two instillations of 2% "xylocaine gel" at an interval of five minutes. A penile clamp is applied after each instillation to retain the anaesthetic agent in the urethra. The surgeons wear a clean waterproof apron and wash their hands so that they may be clean but not sterile. No gloves are worn. Care is taken that only the proximal end of the instrument is handled and that the tip of the cystoscope is introduced directly into the urethra.

#### Results

During the period of investigation 110 cystoscopic examinations were performed on 84 patients (23 were examined twice and a few on three occasions). On 96 occasions (88%) the urine was found to be uninfected both before and four days after cystoscopy (see Table). In two instances the urine was found to have become infected after cystoscopy. In each case a moderate number of leucocytes and a moderate growth of organisms on culture were found in the second specimen of urine. In the first case the infection was due to Streptococcus faecalis; this cleared spontaneously during the next two weeks without any treatment. In the second case the infection was due to Escherichia coli and Proteus vulgaris. The patient had a papilloma at the time of the examination, and his infection cleared up after fulguration of the papilloma and the administration of alkalis and did not recur. These infections were both asymptomatic. It must be mentioned, however, that the presence of a papilloma does not appear to predispose to an infection, as 18 other patients in this group had papillomata present at the time of cystoscopy and they did not develop urinary infections.

#### Table of Results

	No.	No. of Patients
Cystoscopic examinations	110 (100%)	84
Urine sterile before and after cystoscopy	96 ( 88%)	73
,, infected after cystoscopy	2 (<2%)	2
,, found infected before cystoscopy	12 (* 11%)	9

On 12 occasions (nine patients) an existing urinary infection was present at the time of the original examination, which persisted. It was thus interesting to find that about 11% of patients undergoing follow-up cystoscopy in this clinic were suffering from an asymptomatic urinary infection.

These nine patients have been carefully investigated and in six of them an adequate cause for the persistent infection was found. In the remaining three the infection is unexplained and quantitative bacteriology has shown it to be significant, there being more than 3,000 organisms per ml. (Gillespie *et al.*, 1960). These infections have persisted despite various courses of treatment. The patients, however, are perfectly well, and it is difficult to persuade them to attend the clinic, as they have no complaints. At least two of the patients in this group have had a persistent asymptomatic urinary infection for four and six years without any apparent ill-effects, and they both have a normal blood urea and intravenous pyelogram.

#### Discussion

Opinions vary greatly over the dangers of instrumentation and catheterization in producing urinary infections. Beeson (1955–6), in a comprehensive survey of factors concerned in the pathogenesis of pyelonephritis, felt that the frequency with which catheterization and urethral instrumentation are performed indicates that many clinicians believe the danger to be small. There is in fact a lack of factual information on the frequency with which these procedures give rise to urinary infections.

In studies by Kass (1956) and Kaitz and Williams (1960) a significant number of male patients, selected at random and attending a general hospital for a variety of reasons, were found to have an asymptomatic bacteriuria. Their figures respectively were 4% and 10% of such patients. They found that nearly all these patients had undergone previous urethral instrumentation or catheterization. These findings correspond with the 11% of cases with existing urinary infection at the time of cystoscopy in this study. Two-thirds of these patients, however, had some abnormality of the urinary tract to account for this.

This investigation indicates that cystoscopy appears to carry little risk of producing a serious urinary infection. In both cases which developed an infection subsequent to cystoscopy the infection cleared fairly quickly, one spontaneously within two weeks, the other after treatment of his papilloma. Although in 12 examinations (nine patients) there was an existing urinary infection, some of which may have been the result of previous instrumentation, no fresh case of persistent urinary infection occurred in the remaining 98 cystoscopic examinations (75 patients). The persistence of the infection in these nine patients despite medical treatment is in accordance with the findings of Garrod et al. (1954), who showed that patients who had urinary infections associated with abnormalities of the urinary tract responded poorly to treatment with chemotherapy.

Ørskov (1952) felt that cross-infections occurring in the urinary tract were largely due to inadequate sterilization of instruments. Gillespie *et al.* (1960), as has already been mentioned, stress the overriding importance of effective sterilization of instruments. In the case of cystoscopes, sterilization by boiling is felt by many to be the ideal method (Winsbury-White, 1959). This is the procedure employed in this clinic, together with that advocated by Miller *et al.* (1958) for the telescopes, and it appears to be satisfactory. This study confirms that the sterilization of the instrument is the most important factor in preventing urinary infection, and that if this is properly carried out the precautions of "scrubbing up" and the draping of the patient with sterile towels appears to be unnecessary.

I think it is fair to say that cystoscopy performed with an adequately sterilized instrument does not appear to carry a serious risk of infection.

### Summary

A study has been made of the incidence of urinary infection following a routine cystoscopy on 110 occasions in the out-patient department. In 11% of the patients an established asymptomatic urinary infection was found before cystoscopy. This is discussed.

The risk of cystoscopy producing a serious urinary infection appears to be slight, less than 2%, provided adequate precautions are taken for sterilization of the instruments.

I thank Miss P. Bretton, sister in charge of urological out-patients, for her invaluable assistance in this investigation; also Mr. G. C. Tresidder and Professor V. W. Dix for their helpful advice and criticisms. I am grateful to Dr. H. B. May and the staff of the clinical laboratory for carrying out the examinations of the urine specimens.

#### REFERENCES

Beeson, P. B. (1955-6). Yale J. Biol. Med., 28, 81.

Beeson, P. B. (1955-6). Yale J. Biol. Med., 28, 81.
Garrod, L. R., Shorter, R. A., and Curwen, M. P. (1954). Brit. med. J., 2, 1003.
Gillespic, W. A., Linton, K. B., Miller, A., and Slade, N. (1960). J. clin. Path., 13, 187.
Kaitz, A. L., and Williams, E. J. (1960). New Engl. J. Med., 262, 425.
Kass, E. H. (1956). Trans. Ass. Amer. Phycns, 69, 56.
Marple, C. D. (1941). Ann. intern. Med., 14, 2220.
Miller, A., Gillespic, W. A., Linton, K. B., Slade, N., and Mitchell, J. P. (1958). Lancet, 2, 608.
Ørskov, I. (1952). Acta path. microbiol. scand. Suppl. 93, p. 259.
Slade, N., and Linton, K. B. (1960). Brit. J. Urol., 32, 416.
Winsbury-White, H. P. (1959). Proc. roy. Soc. Med., 52, 999.

# **CARBOHYDRATE AND SODIUM METABOLISM IN PERIODIC** PARALYSIS

BY

#### P. B. DOAK, M.B., Ch.B.

Medical Registrar, Public Hospital, Auckland, New Zealand

AND

## K. E. D. EYRE, M.D., M.R.A.C.P.

Assistant Neurologist, Public Hospital, Auckland, New Zealand

It has long been recognized that the administration of glucose, with or without insulin, will usually precipitate weakness in a person with periodic paralysis. The finding of a flat oral glucose-tolerance curve in such people by Aitken et al. (1937) and by Tyler et al. (1951) also suggests that their carbohydrate metabolism may be The significance of these findings was abnormal. largely neglected until MacGregor and Shaper (1957) confirmed the presence of a flat oral glucose-tolerance curve and also demonstrated hypoglycaemia unresponsiveness in a patient with the sporadic form of periodic These authors believed that there was paralysis. increased peripheral uptake of glucose in cases with periodic paralysis and that abnormality in glucose storage was important in the reduction of serum potassium levels and in the production of paralysis. However, the evidence was slight and the possibility of delayed intestinal absorption as the cause of the flat oral glucose-tolerance curve was not excluded.

The discovery that sodium retention often precedes attacks of paralysis (Conn et al., 1956) suggested that an abnormality of sodium metabolism might be the primary disorder in this condition. However, sodium retention does not always precede attacks (Jones et al., 1959), and the protective effect of a low sodium intake cannot always be demonstrated (de Graeff and Brocker, 1958).

The present study of members of a family with familial periodic paralysis was undertaken to reinvestigate carbohydrate metabolism in this condition and to re-examine abnormalities of sodium and creatine metabolism which have previously been reported.

### **Clinical Details**

Four members of the family were studied.

The father, aged 45, had his first attack of paralysis at 6 years of age. Attacks reached a maximum frequency (three or four a week) when he was between 16 and 19 years and have since diminished to one or two monthly. The longest attack occurred at age 19, when he remained paralysed for six days.

A twin son (Antony) and daughter (Susan), aged 14, had their first attack on the same day when aged 3 years, after a long train journey. Antony is the more severely affected and suffers six or seven attacks weekly. Susan has three or four attacks weekly. The twins have always received potassium chloride (up to 24 g. daily) for their attacks. As a result the maximum duration of a single attack is only 48 hours.

The fourth member studied, the only other sibling, is a brother (Peter) aged 10 years, who has never suffered attacks.

All affected members display the typical features of attacks of periodic paralysis. They often wake with complete paralysis of the limbs and trunk. Some movement of the head and neck is always possible, and bulbar and sphincter disturbances have not occurred. Examination at these times shows flaccid paralysis of all limbs with absent tendon reflexes. Less severe attacks can occur at any time of the day, and in some attacks isolated muscle groups were affected alone. Even between attacks muscle strength is impaired. Paralysis is more severe in cold weather, and each affected member recognizes that carbohydrate ingestion is likely to precipitate paralysis.

#### Methods

#### **Glucose** Tolerance

All tests were performed in the early morning after an overnight fast. The subjects were on a normal diet containing 200-300 g. of carbohydrate daily except for the three final oral glucose-tolerance tests performed on Susan when she was on a balance diet containing about 125 g. of carbohydrate.

Oral glucose-tolerance tests were performed using 100 g. of glucose. Venous blood was taken just prior to the administration of glucose and half-hourly for two hours thereafter. On one occasion a Miller-Abbott tube was passed into the jejunum of Antony, its position being confirmed by x-ray examination, and 100 g. of glucose in 150 ml. of water delivered into the small bowel.

Intravenous glucose-tolerance tests were carried out by the method of Amatuzio et al. (1953). For each test a glucose-disappearance rate was calculated.

Insulin-sensitivity tests were made using 0.1 unit of soluble insulin per kilogram of body weight injected