retention are already suffering from a severe cardiovascular or respiratory disease.

We shall always be faced with the problem of selection of cases for prostatectomy from among ageing men whose health is seriously impaired. If after three or four weeks of the best medical treatment improvement has occurred and morale is high, operation should be safe and is worth undertaking even though the patient's expectation of life is very limited.

Between our first and second series of cases we abandoned the permanent suprapubic cystostomy in the treatment of urinary retention and we have not regretted it, for it is a token of the surgeon's despair. An indwelling Riches suprapubic or Gibbons urethral catheter, if carefully managed, can be retained for many weeks, and it leaves the way clear for a safe prostatectomy.

Summary

Two series of 200 cases each of urinary retention due to prostatic obstruction have been analysed. About 64% of each of these came to prostatectomy. Two-thirds of the remainder in each series died without being fit for operation.

After an eight-year interval the mortality of those selected for prostatectomy fell from 15.5% to 6.2%.

Infection and haemorrhage, major causes of death in those who underwent operation in the first series, played a much less significant part in the second series.

Cardiovascular, respiratory, and cerebrovascular disorders, which often precipitate urinary retention, have become the most frequent causes of death after prostatectomy. They also account for the majority of deaths of those with urinary retention who never become fit for surgical treatment.

We would like to acknowledge the work of three ward sisters—Miss M. Armstrong, Miss E. M. Brown, and Miss E. S. Stewart—without whose kindness and skill so many old men would never have regained their health. We also thank Mr. F. D. Breslin for his help in preparing this paper, and Mr. H. G. Hanley, consultant urologist, under whose general supervision all patients were admitted.

REFERENCES

- Dobson, J. F. (1921). *Brit. med. J.*, 1, 289.
 Ellis, H., and Leatherdale, R. A. L. (1958). *Lancet*, 2, 1189.
 Freyer, P. J. (1912). *Brit. med. J.*, 2, 868.
 Groves, E. W. H. (1908). *Ibid.*, 2, 1008.
Lancet, 1956, 2, 343.
 Lloyd-Davies, J. A. (1954). *Brit. med. J.*, 1, 796.
 Miller, A., Gillespie, W. A., Linton, K. B., Slade, N., and Mitchell, J. P. (1958). *Lancet*, 2, 608.
 Rees, W. S. (1947). *Brit. J. Urol.*, 19, 83.
 Riches, E. W. (1954). *Ann. roy. Coll. Surg. Engl.*, 15, 120.
 Salvaris, M. (1960). *Med. J. Aust.*, 47, 370.
 Tenney, B., and Chase, H. M. (1906). *J. Amer. med. Ass.*, 46, 1429.
 Thomson-Walker, J. W. (1921). *Brit. med. J.*, 1, 71.
 — (1930). *Lancet*, 1, 1273.
 Wade, H. (1921). *Brit. med. J.*, 2, 845.

A new British Standard for antral trocars and cannulae (B.S. 3348:1961) has been prepared. They are specified in sets of sizes 6, 7, 8, and 9 designated by the Charrière gauge. Basic requirements for design are laid down and the small Luer conical fitting has been made the standard fitting for connexion of the tubing mount to the cannula. Copies may be obtained from the British Standards Institution, Sales Branch, 2 Park Street, London W.1, or from Coronation House, Market Street, Manchester 1 (price 3s. each, postage extra to non-subscribers).

C

EFFECT OF LOW-CALCIUM DIET ON URINARY CALCIUM IN OSTEOPOROSIS*

BY

S. D. BHANDARKAR, M.D., F.R.F.P.S.
M.R.C.P.Ed.

AND

B. E. C. NORDIN, M.D., Ph.D., M.R.C.P.

University Department of Medicine, Gardiner Institute,
Western Infirmary, Glasgow

We have suggested elsewhere (Nordin, 1961) that osteoporosis could be due to prolonged negative calcium balance arising from failure of the individuals concerned to adapt to their calcium intake. The mechanism of adaptation to low calcium intakes is not fully understood but appears to consist of two components—an improvement in calcium absorption and a reduction in calcium excretion (Malm, 1958). So far as urinary calcium is concerned, we have previously reported (Nordin, 1961) that patients with osteoporosis appear to be less able than normal subjects to reduce their urinary calcium on a low-calcium diet and have suggested that this might be a contributory cause to rather than the result of their disease.

The present paper reports the effect of a low-calcium diet on urinary calcium excretion in 13 normal subjects and 24 cases of osteoporosis.

Methods

Urinary calcium excretion was first measured for two to four days on the ward diet. The subjects were then given a low-calcium diet containing not more than 200 mg. of calcium for 10 days, and the urinary calcium was re-measured in the final two or three days of this 10-day period.

The normal subjects were ward patients without evidence of metabolic bone disease, in the majority of whom x-ray films had shown a normal skeletal status. Their ages ranged from 47 to 72 years and they consisted of seven males and six females.

The cases of osteoporosis were diagnosed by the radiological method of Barnett and Nordin (1960), which implies that they were all relatively severe cases. Seventeen were cases of spinal or "mixed" osteoporosis and seven were cases of peripheral osteoporosis. Their ages ranged from 49 to 79 years and they consisted of 4 males and 20 females.

Calcium was measured in the urine by titration with ethylenediamine tetra-acetic acid in an EEL titrimeter, using ammonium purpurate as the indicator after removal of phosphate in an ion-exchange column. Creatinine was estimated by the method of King and Wootton (1956).

Results

The 24-hour calcium excretion in the normal and osteoporotic subjects on the ward and low-calcium diets is shown in Fig. 1. The mean values are given in Table I. The mean calcium excretion of the normal subjects on the ward diet was 231 mg./24 hours and on the low-calcium diet 167 mg./24 hours. The difference

*This work was carried out during the tenure of a grant from the Medical Research Council.

is highly significant. In the osteoporotic cases the mean urinary calcium excretion on the ward diet was 203 mg./24 hours and on the low-calcium diet 197 mg./24 hours. This difference is not significant.

FIG. 1.—Urinary calcium excretion in 13 normal subjects and 24 cases of osteoporosis on ward diet and low-calcium diet. The horizontal bars represent the mean values $\pm$ two standard errors.

TABLE I.—Mean Urinary Calcium (mg./24 hours)

Group	No.	Ward Diet	Low-calcium Diet	P
Normal	13	230.6 (14.0)	167.0 (13.8)	<0.01
Osteoporosis	24	202.6 (13.8)	197.4 (12.3)	—

Values in parenthesis represent one standard error of the mean.

In 11 normal subjects and 17 cases of osteoporosis the creatinine was also estimated in the urine, and the results in these cases can therefore be expressed as the calcium/creatinine ratio (Ca/Cr) (Nordin, 1959), as shown in Fig. 2 and Table II. The mean fall in Ca/Cr in the normal subjects was 33% and in the osteoporotic cases only 16%. The difference is highly significant. In only 3 of 11 normal subjects did the Ca/Cr fall less than 25%, whereas in only 4 of 21 osteoporotic cases did it fall more than 25%.

The percentage fall in urinary calcium has also been tested in relation to the age and sex of the subjects. No relation was found between age or sex and the ability to reduce urinary calcium excretion on a low-calcium diet.

FIG. 2.—Percentage change in Ca/Cr in 11 normal subjects and 17 cases of osteoporosis. The horizontal bars represent the mean values $\pm$ two standard errors. (Note: In calculating the mean change, a rise in Ca/Cr has been regarded as equivalent to no change.)

Discussion

These observations indicate a significant difference between the ability of normal subjects and patients with osteoporosis to reduce their urinary calcium excretion. The absolute excretion did not differ in the two groups. The osteoporotic patients had a slightly lower urinary calcium excretion than the normal subjects on the ward diet

TABLE II.—Mean Calcium/Creatinine Ratio

Group	No.	Ward Diet	Low-calcium Diet	P
Normal	11	0.24	0.16	<0.05
Osteoporosis	17	0.25	0.22	>0.05

and a slightly higher excretion on the low-calcium diet, but in neither case was the difference significant. However, in the normal subjects the mean excretion fell significantly on the low-calcium diet, but in the osteoporotic cases it did not. In individual cases a fall of less than 25% was uncommon in the normal subjects and a fall of more than 25% was rare in the osteoporotics. This degree of fall in urine calcium on a low-calcium diet is similar to that reported by Hodgkinson and Pyrah (1958), but our normal subjects excreted rather more calcium on the low-calcium diet than those reported by McGeown (1957).

It is, of course, impossible to infer from these observations whether the difference between the normal and osteoporotic subjects is the cause or the result of osteoporosis, although for reasons which we have advanced elsewhere (Nordin, 1961) we think the former inherently more probable. On the other hand, if osteoporosis is in fact due to negative calcium balance, it might be thought that all these patients would have already reduced their urinary calcium as low as it was possible for them to do, and that no further reduction would ever be produced by low-calcium diet. This is not the case. Although the osteoporotic patients as a group failed to lower their urinary calcium, a significant reduction did occur in some of them. Thus in one patient the Ca/Cr fell from 0.41 to 0.28. It is true that even on a low-calcium diet her urinary calcium excretion was abnormal, but it seems none the less surprising that her unexplained hypercalcaemia should have been so easily modified by a reduction in dietary calcium intake if failure of adaptation was the cause of her osteoporosis.

We have reported elsewhere (Nordin, 1961) that there is no relation between dietary calcium and urinary calcium in osteoporosis, and the present observations amplify and confirm this statement. We have also found (and are reporting elsewhere) that at any given level of calcium intake the faecal calcium in osteoporotics does not differ from that of normal subjects, and that the negative calcium balance on low calcium intakes and positive calcium balance on high calcium intakes, which is a feature of osteoporosis, is due to the relative "fixity" of urinary calcium excretion. The implication of this seems to be that osteoporosis may be due to a disturbance in the renal handling of calcium, the cause of which at present is quite obscure, but might be connected with the alleged role of the oestrogenic hormones or might perhaps be connected with the high incidence of infection of the urinary tract in women. We are finding an apparently significant association between chronic pyelonephritis and osteoporosis, which recalls the alleged role of renal infection in the production of "idiopathic hypercalcaemia" (Albright *et al.*, 1953). Our data do not yet permit us to say whether failure to reduce urine calcium on a low-calcium diet is associated primarily with osteoporosis or with pyelonephritis, but it may be significant that two of our three "normal" subjects whose urine calcium fell less than 25% were subsequently found to be suffering from chronic infections of the urinary tract.

Summary

Urinary calcium excretion has been measured in 13 normal subjects and 24 cases of osteoporosis on a ward diet and after seven full days on a low-calcium diet.

The mean calcium excretion of the two groups did not differ significantly on either diet.

In normal subjects urinary calcium excretion fell significantly after a low-calcium diet; in the patients with osteoporosis the mean excretion did not change.

The data are compatible with the concept that patients with osteoporosis are less able than normal subjects to adapt to a low-calcium diet by reducing calcium excretion.

REFERENCES

- Albright, F., Henneman, P., Benedict, P. H., and Forbes, A. P. (1953). *Proc. roy. Soc. Med.*, **46**, 1077.
 Barnett, E., and Nordin, B. E. C. (1960). *Clin. Radiol.*, **11**, 166.
 Hodgkinson, A., and Pyrah, L. N. (1958). *Brit. J. Surg.*, **46**, 10.
 King, E. J., and Wootton, I. D. P. (1956). *Microanalysis in Medical Biochemistry*, 3rd ed. Churchill, London.
 McGeown, M. G. (1957). *Clin. Sci.*, **16**, 297.
 Malm, O. J. (1958). *Calcium Requirement and Adaptation in Adult Men*. Oslo University Press.
 Nordin, B. E. C. (1959). *Lancet*, **2**, 368.
 — (1961). *Ibid.*, **1**, 1011.

TWO CASES OF ACCIDENTAL HYPOTHERMIA

BY

R. G. HARDWICK, M.B., M.R.C.P.
General Practitioner, Liphook, Hampshire

Accidental hypothermia is an uncommon medical emergency in England, although it is relatively common in North America (Britten, 1930). Cases reported have resulted mainly from exposure following intoxication from alcohol, the most celebrated being that of an American negress reported by Laufman (1951). Mountaineers and long-distance swimmers are at special risk, but most commonly it results from shipwreck. Below are reported two cases of different causation, treated differently, both occurring in the United Kingdom.

Case 1

A 72-year-old woman was admitted to the Royal South Hants Hospital on the evening of January 14, 1959. She was thought to be suffering from mild senile dementia when seen by her doctor three weeks previously. About one week before admission her neighbour, believing her to be unsafe with her gas fire, had switched off the main supply, and unfortunately this act coincided with the onset of cold weather, the mean air temperature in Southampton being 32.5° F. (0.28° C.) for the six days January 9 to 14. On January 14 she was found unconscious in bed, dressed in night attire.

On admission to hospital she was lying curled up, unconscious, emaciated, and dehydrated. Her face, arms, and legs were cold and blue. She responded only to painful stimuli; her muscles were generally stiff and the tendon reflexes absent. Her pulse was regular 60/minute, systolic blood-pressure 130 mm. Hg, and respiration 16/minute. Her abdomen was scaphoid, with boardlike rigidity. The bowel sounds were few but normal and the rectal temperature was 81° F. (27.2° C.). There was no evidence of frost-bite. Haematological and biochemical examinations revealed no gross abnormalities, but a haemoglobin of 108% and a C.S.F. pressure of 80 mm. of water supported signs of dehydration.

She was covered with blankets and given oral triiodothyronine 20 mg. eight-hourly and penicillin 500,000 units twice daily parenterally. External heat was avoided. Her temperature gradually rose to 98° F. (36.7° C.) after 56 hours (Fig. 1). She was then more deeply unconscious, her radial pulse being impalpable, carotid pulse weak, and blood-pressure unrecordable. On this day she was to be transferred to a neurosurgical unit to exclude a diagnosis

FIG. 1.—Rectal and axillary temperature in Case 1 from admission.

of subdural haematoma. Investigation was postponed for two days when her systolic pressure became stabilized at 95 mm. Hg. Bilateral posterior parietal burr-holes were made on January 19; the only abnormality found was a C.S.F. pressure of 30 mm. of water. This was confirmed by lumbar puncture, and after the injection of normal saline into the subarachnoid space, raising the pressure to 120 mm. of water, she regained consciousness. She was given water plentifully by mouth and added salt. There was no evidence of hypothyroidism, which was first considered as a possible factor leading to hypothermia, and she was eventually discharged on February 21 still slightly demented but probably no worse for her hypothermic interlude.

Case 2

This is the case of a long-distance swimmer, Florence Chadwick. Her first attempt to swim the Irish Channel, a distance of just under 21 miles (33.6 km.), was in 1957. She was then taken from the water after 11 hours and 47 minutes, some 2½ miles (4 km.) from the Scottish coast. She resisted rescue, but on reaching the deck of the rescue boat collapsed unconscious. Her temperature was not recorded, but she was pulseless, her blood-pressure was unrecordable, and her heart sounds were slow and faint. Her respiratory rate was also slow. Her body was described by Dr. W. A. Baird as being cold "like a mortuary specimen," and he expressed doubts concerning her recovery. She was wrapped in blankets but supplementary heat was avoided. After four hours her blood-pressure had risen to 90/60 mm. Hg and she had auricular fibrillation, which continued for at least 24 hours. After 48 hours she was fully recovered. Subsequent medical examinations, including E.C.G., have shown no abnormality.

In the autumn of 1960 she returned to England to make three further attempts. The first two were aborted by bad weather after two and six hours respectively. I was asked to accompany her on her third attempt, which eventually took place in good weather on October 15, 1960. Forty-