malignancy (Myers, 1958). However, a fall in serum calcium with cortisone is very good evidence against a diagnosis of hyperparathyroidism. Correspondingly, if a raised serum calcium does not fall with cortisone it should not be attributed to sarcoidosis even if that disease is known to be present (Snapper *et al.*, 1958).

In our patient the initial change in serum calcium on bed rest alone precluded the diagnostic use of cortisone but was of great diagnostic value in itself. The patient's symptoms of anorexia, loss of weight, and constipation, which were probably due to hypercalcaemia, started while he was working in the open air, exposed to the sun. The fall in serum calcium, which was accompanied by symptomatic improvement, occurred after removal from this exposure. This relationship to sunlight has been observed before (Mather, 1957), and Dent (1958) has shown that ultra-violet irradiation may exacerbate both hypercalcaemia and hypercalciuria in sarcoidosis.

Summary

The case is presented of a patient with sarcoidosis and hypercalcaemia in whom decreased tubular reabsorption of phosphate was demonstrated, producing a biochemical resemblance to primary hyperparathyroidism.

The differential diagnosis between the two conditions is discussed, and it is concluded that tests of phosphate excretion may be misleading, but that the response to cortisone gives valuable information.

We thank Dr. A. D. Everett for referring the patient to us, Dr. A. Gordon Signy for his help with the histological and biochemical investigations, Dr. F. Harwood Stevenson for his criticism of the manuscript, and Professor C. E. Dent for his continued help and advice in the management of the patient.

REFERENCES

- Albright, F., and Reifenstein, E. C. (1948). *The Parathyroid Glands and Metabolic Bone Disease*. Williams and Wilkins, Baltimore.
- Anderson, J., Harper, C., Dent, C. E., and Philpot, G. R. (1954). *Lancet*, **2**, 720.
- Chambers, E. L., Gordan, G. S., Goldman, L., and Reifenstein, E. L. (1956). *J. clin. Endocr.*, **16**, 1507.
- Citron, K. M. (1955). *Postgrad. med. J.*, **31**, 516.
- Davidson, C. N., Dennis, J. M., McNinch, E. R., Willson, J. K. V., and Brown, W. H. (1954). *Radiology*, **62**, 203.
- Dent, C. E. (1956). *Brit. med. J.*, **1**, 230.
- (1958). *Proc. VIII Middle East Medical Assembly*, p. 162.
- Goldman, R., and Bassett, S. H. (1954). *J. clin. Invest.*, **33**, 1623.
- Harrell, G. T., and Fisher, S. (1939). *Ibid.*, **18**, 687.
- Hauch, E. W., and Fisher, R. L. (1957). *Calif. Med.*, **86**, 333.
- Henneman, P. H., Dempsey, E. F., Carroll, E. L., and Albright, F. (1956). *J. clin. Invest.*, **35**, 1229.
- Herbert, F. K. (1951). In *Recent Advances in Clinical Pathology*, 2nd ed., edited by S. C. Dyke. Churchill, London.
- Howard, J. E., Carey, R. A., Rubin, P. S., and Levine, M. D. (1949). *Trans. Ass. Amer. Physns*, **62**, 264.
- Jacobs, E., and Verbanck, M. (1953). *Acta med. scand.*, **145**, 143.
- Kabakow, B., Mines, M. F., and King, F. H. (1957). *New Engl. J. Med.*, **256**, 59.
- Klatskin, G., and Gordon, M. (1953). *Amer. J. Med.*, **15**, 484.
- Longcope, W. T., and Freiman, D. G. (1952). *Medicine (Baltimore)*, **31**, 1.
- Mather, G. (1957). *Brit. med. J.*, **1**, 248.
- Myers, W. P. L. (1958). *Cancer*, **11**, 83.
- Nordin, B. E. C., and Fraser, R. (1956). In *Ciba Foundation Symposium on Bone Structure and Metabolism*, p. 222. Churchill, London.
- Schatten, W. E., Ship, A. G., Pieper, W. J., and Bartter, F. C. (1958). *Ann. Surg.*, **148**, 890.
- Scholz, D. A., and Keating, F. R. (1956). *Amer. J. Med.*, **21**, 75.
- Snapper, I., Yarvis, J. J., Freund, H. R., and Goldberg, A. F. (1958). *Metabolism*, **7**, 671.
- Thomas, W. C., Connor, T. B., and Morgan, H. G. (1958). *J. Lab. clin. Med.*, **52**, 11.
- Westra, S. A., and Visser, J. F. (1949). *Ned. T. Geneesk.*, **93**, 18.

POTASSIUM DEFICIENCY IN ANOREXIA NERVOSA, WITH REFERENCE TO RENAL TUBULAR VACUOLATION*

BY

R. D. WIGLEY, M.B., M.R.C.P., M.R.A.C.P.

Physician, Palmerston North Public Hospital, New Zealand

Electrolyte deficiency in anorexia nervosa may create a therapeutic problem, not mentioned in several recent papers, in the treatment of this disorder (Williams, 1958; Wall, 1959). Diagnostic difficulties may arise, especially when secondary renal involvement occurs, leading to serious consideration of the alternative diagnoses listed in Table I. Investigation of a case presenting typical features of anorexia nervosa together with hypokalaemic alkalosis, and evidence of renal failure with chronic pyelonephritis, led to a search of the relevant literature. Sunderman and Rose (1948)

TABLE I.—*Differential Diagnosis*

1. Hypopituitarism and Addison's disease
2. Malabsorption syndrome
3. Habitual purgation
4. Potassium- and sodium-losing nephritis
5. Primary aldosteronism
6. Chronic nephritis and chronic pyelonephritis
7. Pineal tumours and hypothalamic lesions
8. Familial periodic paralysis

TABLE II.—*Summary of Cases Reported to Date (Serum Levels in mEq/l.)*

Authors	Age	Na	K	Cl	HCO ₃	Evidence of Renal Involvement	Notes	
Sunderman and Rose (1948)	26	—	3.0	58	62	—	Enemas and vomiting	
Elkinton <i>et al.</i> (1951)		127	2.8	74	44.5	Yes	No vomiting; laxatives	
Beck and Brochner-Mortensen (1954; Case 26)	28	137	2.6	96	—	„	Vomiting. Blood urea 51 mg.; 26 mg. after treatment	
Luft and Sjögren (1954)	1	26	139	2.2	45	49	„	Vomiting and tetany.
	2	34	139	1.5	98	21	„	Albuminuria. No vomiting. No albuminuria
Klotz and Lumbroso (1955)	29	141	2.0	—	55	—	—	Vomiting
Rossier <i>et al.</i> (1955)	St. H.	28	140	2.0	74	45	—	—
	F. E.	24	—	2.9	—	29	Yes	Pyelonephritis
	K. M.	24	146	2.6	97.4	28	—	Diarrhoea and vomiting
	M. M.	23	142	3.1	78	37	—	Vomiting
Siebenmann (1955)	T. E.	17	148	3.6	105	29	—	—
	H. A.	28	143	3.4	—	24	—	—
Forssman (1955)	27	133	1.8	109	20	Yes	Yes	Took laxatives
Ljunggren <i>et al.</i> (1957)	17	143	3.0	50	—	„	„	Said to have nephritis
Elkinton and Huth (1959)		128	2.9	85	40	—	—	—
	A. B.	29	120	1.7	53	52	Yes	Magnesia
	M. B.	43	126	2.4	69	44	—	Vomiting and magnesia

described the first case with hypokalaemic alkalosis. Seventeen cases have now been reported (Table II). Elkinton and Huth (1959) have reviewed the electrolyte disorders that may occur. Most of the cases showed alkalosis, some hyponatraemia, and eight showed evidence of renal dysfunction.

*Based on a paper read at a meeting of the Royal Australasian College of Physicians at Auckland on September 17, 1959.

It is now generally accepted that potassium depletion can cause renal dysfunction, resulting in thirst, polyuria, isosthenuria with tubular vacuolation, and often pyelonephritic changes (Relman and Schwartz, 1958; Milne *et al.*, 1957). The case of anorexia nervosa described by Siebenmann (1955) is of particular interest in that post-mortem examination showed vacuolation of the renal tubules and fragmentation of heart muscle. Diarrhoea from aperients must have contributed to the severe potassium depletion in this case. Three cases of anorexia nervosa in which there was no history of habitual purgation are reported. One of these showed tubular vacuolation in a renal biopsy and at necropsy.

Case 1

The patient was a married woman aged 38. She was admitted to the Palmerston North Hospital in July, 1957, with loss of weight, amenorrhoea, and sensitivity to cold for one year and lack of energy for six months. Her appetite was variable. Usually she claimed that it was good, but her husband said that she ate very little at the onset of illness and vomited frequently. It appears that what she regarded as a good appetite was small by ordinary standards. Her skin was dry, with a yellowish tinge, and a growth of fine downy hair was noticeable on the face. Blood-pressure was 108/70. She had a normochromic anaemia (haemoglobin 9.5 g./100 ml.). The serum cholesterol was 480 mg./100 ml.; and potassium 3.2, sodium 135, chloride 108, and bicarbonate 25 mEq/l. The urine contained albumin with numerous granular casts. The specific-gravity range was 1009–1026. *Escherichia coli* was cultured from the urine. 25% of a 1,500-ml. water load was excreted before and 90% after 50 mg. of cortisone. Urine 17-ketosteroids excretion was 4.5 mg./day and 17-ketogenic steroids 3.8 mg./day. An oral glucose-tolerance curve rose from a fasting level of 85 to 100 mg./100 ml. at three-quarters of an hour, falling to 50 mg. at one and a half hours. B.M.R. +1%. No excess of fat globules was found in the faeces. Barium-meal examination and follow-through showed nothing abnormal.

The psychiatric findings (Dr. S. Mirams) were consistent with a diagnosis of anorexia nervosa. The patient was said to have changed mentally after having a stillborn child, 11 years before. A tubal ligation followed this. Her husband fell below her in standards of social behaviour and intelligence, and was an alcoholic. The home was unhappy and she was frigid.

Arrangements were made for fat- and electrolyte-balance studies, but the patient could not be persuaded to re-enter hospital. A trial of prednisone, 15 mg. daily, as an out-patient produced an improvement in appetite and she gained 7 lb. (3.2 kg.) in weight, but the serum potassium remained low (3.7 mEq/l.) and it was thought inadvisable to continue. In February, 1958, her weight fell to 5 st. 8 lb. (35.4 kg.), with weakness, marked anorexia, and hypotension (90/60). The serum potassium was 2.3 mEq/l. She again refused admission.

In June, 1958, she agreed to out-patient treatment. The serum potassium was lower at 2.2 mEq/l., total N.P.N. 59 mg./100 ml., and cholesterol 320 mg./100 ml. A renal biopsy showed marked tubular vacuolation indistinguishable from the post-mortem changes (see Fig.). Oral potassium chloride was prescribed, but it is very doubtful if she took much of it, as she was admitted eight days later with pneumonia and hypokalaemic paralysis which failed to respond to an intravenous infusion of potassium chloride and antibiotics. She died six hours later. The serum potassium, chloride, and sodium before treatment were 1.3, 103, and 135 mEq/l. respectively and the urine potassium 9.0, chloride 58, and sodium 61 mEq/l. (normal potassium level greater than 10 mEq/l.: Schwartz, 1955).

Post-mortem Examination (Dr. W. L. Kenealy).—The right kidney weighed 120 g. and the left 110 g. They were pale, with no reduction of cortical tissue. There was a small subcapsular haematoma at the biopsy site. Sections of the kidney confirmed the biopsy finding of marked vacuolation of the cells lining the proximal convoluted tubules and a minor degree of chronic pyelonephritis (see Fig.). The pituitary and endocrine glands were normal, A

Case 1. Photomicrograph showing vacuolation of cells lining the proximal convoluted tubules. (×264.)

penicillin-resistant *Staphylococcus aureus* was cultured from the bronchi, which were inflamed, and from the left lower lobe, which was consolidated.

Comment.—The patient was thought to be a case of anorexia nervosa with prolonged potassium deficiency resulting in tubular vacuolation and minor chronic pyelonephritic changes.

Case 2

The patient, a married woman aged 30, was admitted to the Green Lane Hospital, Auckland, in March, 1956, with intermittent paralysis of the legs. Swelling of the ankles, thirst, and nocturia had been present for about one year. Six years earlier, when her eldest child was born, her periods ceased. They had been irregular before this and she had been given injections in the hope of restoring a normal rhythm. It was about this time that family difficulties arose and she separated from her husband. There was no history of taking purgatives.

She weighed only 4 st. (25.4 kg.). Breast development was poor. There was fine downy hair all over the body, but hair distribution was otherwise normal. The skin was dry. Her blood-pressure was 100/84 and there was a generalized loss of muscle power. The serum potassium was 1.85, chloride 107, sodium 154, and bicarbonate 29 mEq/l. The total N.P.N. was 28 mg./100 ml., haemoglobin 11.3 g./100 ml., and serum cholesterol 190 mg./100 ml. Coliform bacilli were cultured from the urine, which contained large numbers of white cells. The urine potassium varied between 1 and 3 mEq/l., 17-ketosteroids 4.5 mg./day, and F.S.H. less than 48 units per day. Cardiography showed typical hypokalaemic changes. She refused medical advice for several years.

In 1958 her weight fell as low as 3 st. 7 lb. (22.2 kg.). In March, 1959, she was admitted to the Auckland Hospital with piles and a fistula-in-ano. She weighed 4 st. 2 lb. (26.3 kg.) at this time. A catheter urine specimen contained no casts, cells, or organisms. The serum albumin was 2.5 and globulin 3.6 g./100 ml. In September, 1959, her doctor reported that with his assistance she had improved considerably and was again living with her husband.

Comment.—An otherwise typical case of anorexia nervosa with hypokalaemic paralysis.

Case 3

A spinster aged 33 came under the care of Dr. C. P. Powles, of Wanganui, in 1952, with depression and anorexia, insomnia, loss of weight, and nausea. She had an obsessional personality and there were emotional problems concerning her home life and her work. She developed amenorrhoea in 1953 and has had infrequent scanty periods since. She complained of thirst and admitted to occasional vomiting. Cachexia was marked, and there was some growth of fine hair and muscular weakness. Blood-pressure was 95/70. A diagnosis of anorexia nervosa was made and she was treated accordingly.

Her symptoms varied in intensity until she was admitted to the Wanganui Hospital in March, 1956, with increasing tiredness, nausea, and vomiting. Her serum potassium was found to be 3.3 mEq/l. and the sodium 135 mEq/l. In a relapse in 1958, the serum potassium was 3.0 mEq/l. She weighed 5 st. 10 lb. (36.3 kg.). At this time her specific-gravity range was limited to 1008–1016 and urea clearance was 59% of normal. Later she developed tetany, which responded to intravenous calcium gluconate. The serum calcium was 5–6 mg./100 ml.; bicarbonate was 26 and chloride 99 mEq/l.

In September, 1958, she was reinvestigated in Dunedin. At this time she weighed only 5 st. (31.8 kg.) and her serum potassium was 3.5 mEq/l., sodium 139, chloride 108, but the urinary potassium was low at 7 mEq/l. The total N.P.N. was 28 mg./100 ml., the urine concentration 1030, and the urea concentration 3.5%.

TABLE III.—Lowest Figures Recorded in mEq/l. Urine Levels in Parentheses. All Female Cases

Case	Weight	Age	N.P.N./ 100 ml.	Na	K	Cl	HCO ₃	Notes
1	5 st. 8 lb. (35.8 kg.)	38	86	135 (61)	1.3 (9.0)	103 (58)	22	Paralysis. Renal lesion
2	4 st. (25.4 kg.)	30	28	154	1.85 (1.0)	107	29	Paralysis. Urinary infection
3	5 st. (31.75 kg.)	33	28mg.	139	3.5, 3.0 (7.0)	108	N.	Transient renal involvement

Comment.—A long-standing case of anorexia nervosa with potassium depletion which was probably responsible for the thirst and transient impairment of renal function.

Discussion

Squires and Huth (1959), in an experimental potassium depletion in man, have shown that a negative balance occurs if intake is reduced from 14 to 1 mEq/day. Presumably the potassium depletion of anorexia nervosa is produced in the same way. Elkinton and Huth (1959), in a comprehensive review of this problem, emphasize that it is very difficult to be sure that these patients are not vomiting or taking purgatives. Either of these factors might aggravate the potassium deficiency. Though steatorrhoea was not seriously considered in the present cases it was not entirely excluded as a possible cause of potassium loss. These patients are rarely co-operative enough to conduct reliable fat-balance tests, and it is to be hoped that faecal potassium estimation, labelled fat- (Rowlands, 1959) and D-xylose (Butterworth *et al.*, 1959) absorption tests, and jejunal biopsy (Shiner, 1959) will enable more complete investigation of small-bowel function if further cases are encountered. Ljunggren *et al.* (1957) demonstrated low intracellular potassium levels in two of three cases of anorexia nervosa with serum levels below

3.7 mEq/l., indicating that the low serum and urine levels do reflect an intracellular potassium depletion. The low serum-chloride levels found can be partly explained by the extracellular alkalosis which occurs in experimental potassium depletion when sodium is not withheld (Fourman, 1954; Huth *et al.*, 1959). Conversely, the three cases without alkalosis may have been in salt depletion, though the serum sodium was depressed in only two of these. Chloride loss from vomiting may have aggravated the alkalosis to the point where the urine became alkaline, so that paradoxical aciduria did not always occur.

The possibility that hypersecretion of the adrenal cortex could explain the hypokalaemic alkalosis is discounted, as adrenal dysfunction has not been demonstrated in anorexia nervosa (Bliss and Migeon, 1957). The urine 17-ketosteroids were low in two of the present cases. Primary aldosteronism is excluded by the low urine levels of potassium. The increased water excretion in Case 1 after cortisone is difficult to explain.

Kleeman and Maxwell (1959) have cast some doubt on whether the thirst of potassium depletion is entirely of renal origin and offer evidence of a direct effect on the hypothalamus or posterior pituitary. Histochemical studies of the vacuolar lesion suggest that the translucencies are due to swelling of mitochondria (Pearce and MacPherson, 1958). This lesion is reversible so far as is known, but the associated pyelonephritic changes may be permanent (Relman and Schwartz, 1958). *E. coli* infection is a frequent complication, and Woods *et al.* (1959) have shown that potassium deficiency predisposes to experimental infection with this organism, but the possibility remains that prolonged deficiency may result in irreversible changes resembling those of pyelonephritis (Stanbury, 1958). Albuminuria, pyelonephritis, or nephritis was mentioned in 8 of the 17 cases in the literature (Table II), so it seems probable that tubular vacuolation was present in some of them.

The complex clinical picture produced by electrolyte and renal changes in anorexia nervosa may well have led to misdiagnosis in the past, accounting for the paucity of information available on this subject.

Summary and Conclusions

Seventeen reported cases of anorexia nervosa with hypokalaemia are summarized. Eight showed some evidence of renal disorder. Habitual purgation complicated the only case shown to have the tubular lesion of potassium depletion.

Three further cases with hypokalaemia are described, one showing tubular vacuolation.

It is thought that the potassium depletion is due to a deficient intake, aggravated in some cases by vomiting, and in some by purgation. Malabsorption may be more certainly excluded if further cases become available.

Fatal paralysis and possibly irreversible renal changes may occur if this complication is not recognized. Five of the 20 patients had died at the time of reporting.

Conventional treatment should correct the deficit, but oral or intravenous potassium chloride may prove necessary.

I am indebted to Dr. D. S. Dickson for providing details of Case 2; to Dr. C. P. Powles for allowing me to see

Case 3; to Dr. R. W. Medlicott and Professor J. E. Caughey for lending their records of Case 3; to Miss Anne Simmonds for the biochemical estimations in Case 1; and to Mr. Hamblyn for the photomicrograph. Dr. W. L. Kenealy and Dr. T. H. Pullar have given invaluable assistance in the preparation of this paper.

REFERENCES

- Beck, J. C., and Bröchner-Mortensen, K. (1954). *Acta med. Scand.*, **149**, 409.
- Bliss, E. L., and Migeon, C. J. (1957). *J. clin. Endocr.*, **17**, 766.
- Butterworth, C. E., Perez-Santiago, E., de Jesus, J. M., and Santini, R. (1959). *New Engl. J. Med.*, **261**, 157.
- Elkinton, J. R., and Huth, E. J. (1959). *Metabolism*, **8**, 376.
- , Squires, R. D., and Crosley, A. P. (1951). *J. clin. Invest.*, **30**, 369.
- Forssman, B. (1955). *Nord. med.*, **54**, 1373.
- Fourman, P. (1954). *Clin. Sci.*, **13**, 93.
- Huth, E. J., Squires, R. D., and Elkinton, J. R. (1959). *J. clin. Invest.*, **38**, 1149.
- Kleeman, C. R., and Maxwell, M. H. (1959). *New Engl. J. Med.*, **260**, 268.
- Klotz, H. P., and Lumbroso, P. (1955). *Sem. Hôp. Paris*, **31**, 440.
- Ljunggren, H., Ikkos, D., and Luft, R. (1957). *Acta endocr. (Kbh.)*, **25**, 209.
- Luft, R., and Sjögren, B. (1954). *Ibid.*, **17**, 264.
- Milne, M. D., Muehrcke, R. C., and Heard, B. F. (1957). *Brit. med. Bull.*, **13**, 15.
- Pearce, A. G. E., and MacPherson, C. R. (1958). *J. Path. Bact.*, **75**, 69.
- Relman, A. S., and Schwartz, W. B. (1958). *Amer. J. Med.*, **24**, 764.
- Rossier, P. H., Staehelin, D., Bühlmann, A., Labhart, A. (1955). *Schweiz. med. Wschr.*, **85**, 465.
- Rowlands, E. N. (1959). *Proc. roy. Soc. Med.*, **52**, 1.
- Schwartz, W. B. (1955). *New Engl. J. Med.*, **253**, 601.
- Shiner, M. (1959). *Proc. roy. Soc. Med.*, **52**, 10.
- Siebenmann, R. E. (1955). *Schweiz. med. Wschr.*, **85**, 468.
- Squires, R. D., and Huth, E. J. (1959). *J. clin. Invest.*, **38**, 1134.
- Stanbury, S. W. (1958). *Advanc. intern. Med.*, **9**, 271.
- Sunderman, F. W., and Rose, E. (1948). *J. clin. Endocr.*, **8**, 209.
- Wall, J. H. (1959). *Amer. J. Psychiat.*, **115**, 997.
- Williams, E. (1958). *Brit. med. J.*, **2**, 190.
- Woods, J. W., Welt, L. G., Hollander, W., and Newton, M. (1959). *J. clin. Invest.*, **38**, 1056.

DIAGNOSIS AND TREATMENT OF TRICHOMONAL URETHRITIS IN MEN

BY

R. D. CATTERALL, M.R.C.P.Ed.

Department of Venereology, General Infirmary at Leeds;
Formerly Physician to the Medical Research Council
Investigation into Non-Gonococcal Urethritis,
Whitechapel Clinic, London Hospital

Trichomonas vaginalis was first reported to be present in the genital tracts of male patients by Marchand in 1894. Since then many other instances and series of cases have been published. However, until recently the recorded incidence of infection in men has been low. With improved techniques and increasing interest in non-gonococcal urethritis during recent years, results have indicated that infestation with the parasite is more common than had formerly been supposed. There seems little doubt that the organism is one of the causes of genital infection in the male, though its precise importance in the aetiology of non-gonococcal urethritis is not yet determined.

The results of investigation have varied greatly in different hands. For example, Ackermann (1935) failed to find *T. vaginalis* in the urethral secretions of 37 men with non-gonococcal urethritis, though the parasite was present in the vaginal secretions of six of their consorts. Allison (1943) found *T. vaginalis* in 15%

of a group of white patients with gonorrhoea or other genital infections and also in 200 negroes who had been rejected for military service because of venereal disease: 95% of the negro patients had urethral strictures. Feo (1944) found the parasite in 121 of 735 male negroes (16.4%), and in 23 of 191 male white patients (12%). Whittington (1957) found *T. vaginalis* in 15.3% of 326 men with non-specific urethritis. The highest recorded incidence is that of Coutts *et al.* (1955), who claimed that 68% of 2,482 male patients in Chile were infected with the parasite.

The object of this investigation was to examine in detail the clinical and bacteriological findings in a large series of male patients with trichomonal urethritis and to attempt to evaluate various forms of therapy.

Material and Methods

All the patients included in this series attended the Whitechapel Clinic of the London Hospital during the three years from January, 1956, to January, 1959. A detailed history was obtained from each patient and a thorough physical examination performed. Urethral specimens for microscopical examination were taken by the method advocated by Lanceley (1954) by gently stroking the urethral wall with a platinum loop. The slides were examined at once by dark-ground microscopy. In addition, smears of the urethral secretions were also stained with Gram's stain and examined microscopically under the 1/12-in. objective. Cultures for *T. vaginalis*, using the liquid liver medium of Feinberg and Whittington (1957), were performed in half of the cases. Stuart's transport medium was also inoculated with specimens from the urethra, and these were later transferred to MacLeod's chocolate agar. In a small number of cases the centrifuged deposit of urine was examined microscopically and cultures were performed. In all cases in which the organism was found attempts were made to persuade the consorts to attend for examination and, if necessary, treatment.

Diagnosis

T. vaginalis was found in 126 cases. During the same three-year period there were 2,300 infections described as non-gonococcal urethritis recorded at the clinic. Trichomonal urethritis, therefore, comprised 5.5% of the total infections diagnosed as non-gonococcal urethritis.

The average age of the patients was 33 years. There were 82 white patients and 44 of negro race.

Eighteen patients (14.3%) had no symptoms referable to the genito-urinary tract. Ninety patients (71.4%) complained of urethral discharge: the type of discharge was very variable, but in most cases was small in quantity, mucopurulent, and most obvious early in the morning before the first micturition; in a small proportion of cases, however, it was frankly purulent, and thick, and was present throughout the day. Itching inside the penis was complained of by 26 patients (20.6%), dysuria occurred in 15 (11.9%), frequency of micturition in 4 (3.2%), two had noticed blood-stained urethral discharge, and one complained of haematuria. Pain and swelling of an epididymis was the presenting manifestation in one case.

At the first examination 80 of the patients (63.5%) were found to have a urethral discharge. In the cases