Foreword

The clinical articles on Urological subjects in this issue of the Bulletin have been contributed by the members of the Department of Urology, Dalhousie University, and include experiences with Urological cases in the Victoria General Hospital, Camp Hill Hospital, the Children's Hospital and the Halifax Infirmary.

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Pediatric Urology

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UROLOGY, generally speaking, is in much of its practice applied to the geriatric patient. Anomalies and diseases of the urogenital tract do occur, however, in infants and children, and may be a very important factor in the health and development of the child.

A survey of hospital admissions which could be classified as primarily pediatric urological cases indicated that these fall within four main groups in the following order of frequency:

1. Anomalies
2. Infections
3. Trauma
4. Neoplasms

The two last classifications represent a small percentage of the total. There is another group of miscellaneous entries, such as redundant prepuce, enuresis, investigation only, etc.

The total number of urological cases compared to the total admissions is relatively small, so that in general practice, one would not expect to encounter many of these problems. Nevertheless, the more the family physician is aware of their possible existence, the earlier and more accurately diagnosis is likely to be made, appropriate advice given, and treatment initiated.

Congenital anomalies were found to be of most frequent occurrence. The most obvious of these are abnormalities of the external genitalia, and these are of great concern to the parents, particularly the mother, so that any reassurance that can be given does much for their peace of mind.

The abnormal location of the external urethral meatus on the ventral or under surface of the penis, constituting the condition of hypospadias, is not uncommon. There are varying degrees of this deformity, fortunately the more minor ones being of greater incidence. The most common location of the meatus in this condition is at the coronal junction just proximal to the glans. However, the opening may occur anywhere along the shaft of the penis or in the perineum. In the latter case, the scrotum is bifid, that is, has failed to fuse in the midline and at first glance may appear as labial folds, leading to some difficulty in determining sex In all cases, the prepuce has a hooded effect, and if the opening is along the shaft of the penis or farther back, the penis is curved under by a fibrous band, the effect being described as a chordee.

In regard to treatment, first of all, these cases should never be circumcised. The redundant prepuce is a valuable piece of skin, which can be used to good advantage in plastic procedures if correction is necessary. The mild first degree hypospadias requires no correction, as it produces no disability in voiding or sexual intercourse and no loss of fertility. Those of more severe degree, where the deformity necessitates sitting down to urinate, interferes with normal sex relations in later life, and tends to engender a feeling of inferiority in the child, require correction. When should this be done? Preferably between the ages of four and five years, before beginning school, not because repair is more difficult later (actually it may be easier, "the more the cloth the better the cut"), but because of the psychological factors involved.

As to the method of repair, multiple surgical techniques have been described, which, of course, serves to indicate that no one technique has been entirely satisfactory. These are all stage procedures, the first stage being correction of the chordee, followed by the formation of a urethra at a second operation. A most satisfactory technique has been described by Dennis-Brown for the construction of the urethra, which in its simplicity and satisfactory results has received wide approval. Diversion of the urine, either by suprapubic cystostomy or perineal urethrostomy, is requisite for post-operative healing.

The condition of epispadias, where the anomalous urethral orifice is on the dorsum or upper surface of the penis, is of rarer occurrence than hypospadias. In the majority of these cases the defect is complete; that is, the urethra opens directly into the bladder under the pubis. These children are often incontinent, depending on whether or not the bladder neck and posterior urethra have been formed. Fortunately, they respond well to surgery—again a plastic, multiple stage procedure, consisting of first of all forming a bladder neck to permit voluntary retention of urine within the bladder, and then a subsequent operation burying a tube of epithelium in the penis to form a urethra. Developmental arrest at an earlier stage than this results in no anterior wall of the bladder, the trigone and posterior wall lying exposed. There is no reservoir for urine; hence the child is always wet, and the odor of infected urine prevails, rendering him objectionable to others as well as himself. This distressing condition is known as ectopic bladder, and the best we have had to offer these children in the past has been diversion of the urine to the bowel by implantation of the ureters in the rectosigmoid. This can be done, provided that anal sphincter control is present. The bladder is then removed, and the resulting defect in the anterior abdominal wall closed and, in the case of the male, the epispadias repaired so that the urethra will conduct the seminal secretions. Although operative mortality is low, morbidity in the post-operative stage and later is of relatively high incidence, due to ascending infection, with the development of chronic pyelonephritis and associated electrolyte imbalance. Antibiotics and a better recognition and understanding of electrolyte balance have done much to minimize these complications, but they still exist as a potential threat to the well-being of the patient.

Utilization of an isolated segment of ileum, with the ureters implanted in one end and the other end opening on the abdominal surface, similar to an ileostomy or colostomy, has been developed in an attempt to avoid ascending infection and its sequelae. This still means, however, that the child has to wear some appliance for collection of the urine and its disposal at a convenient time.

Ideally, in these cases of vesical ectopia, treatment would be directed towards developing what bladder the child has into a reservoir of adequate capacity to permit intermittent emptying. In the past, this has been unsuccessful, but now, with greater control over infection and improved techniques, it has become possible in some cases. In any event, attempt at construction of a bladder is now the first approach, and if the result is a complete failure, urinary diversion by one of the other methods remains available. The earlier these cases are brought to surgery the better, and sometime during the second year of life would seem to be the most satisfactory time, as children withstand surgery very well at this age, and infection has not damaged the upper urinary tract.

The previously mentioned conditions are obvious and visible anomalies whose sufferers will seek advice on their own initiative; but there remain the

less obvious ones, which frequently are the more important, because when they come to investigation it may be too late for successful therapy. As was listed above, the next most common diagnoses are infections, and it is as true in infants and children as in adults that the majority of these are due to some obstructive pathology in the urinary tract. The obstructive lesion, predisposing to stasis and infection, may occur anywhere from the external urethral meatus to the junction of ureter and renal pelvis, or even in the infundibulum draining a single calyx.

If the obstruction is in the urethra or at the bladder neck, the child may exhibit frequency, enuresis, crying and obvious straining on voiding, and a urinary stream of inadequate size and force. The bladder may be visibly, palpably, or percussably distended. Urinalysis may reveal pus and red blood cells. Inspection may disclose a pin-point urethral meatus or one that is scarred or crusted from chronic infection or ulceration. An adequate meatotomy and follow-up dilation will cure this particular condition. Calibration of the urethra with a catheter will reveal any congenital urethral stricture, and often catheter dilation at repeated intervals will be all that is necessary. Catheterization will also disclose the presence of residual urine, indicative of bladder dysfunction of either obstructive or neurogenic origin. Congenital valves of the posterior urethra may cause marked obstructive pathology in early infancy, and should be suspected in all cases of difficult voiding in the early months. Urethroscopic examination in suspected cases and destruction of the valves by transurethral electrosurgery, or retropubic exposure and resection of the valves, are the treatments of choice in such cases.

Congenital contraction of the bladder neck, comparable to the median bar or bladder neck sclerosis encountered in the elderly male, likewise can be suspected, investigated, and if present, treated by either transurethral or suprapubic resection.

Neurogenic dysfunction, due to faulty innervation of the bladder itself or to a disturbance of the nerve supply to the bladder, as, for example, in a spina bifida with meningomyelocele, may be a cause of disturbed urination and infection.

Not infrequently one finds lesser degrees of bladder dysfunction and predisposition to infection, due to either minor obstructive lesions or neurogenic causes, where no specific surgical intervention is indicated. These cases usually respond well to long term therapy of the infection and prophylaxis against recurrence. Such cases should be periodically reassessed by urinalysis, urine culture, determination of residual urine, etc., to ascertain that no progressive damage is occurring in the urinary tract.

As in the adult, the intravenous pyelogram is a most useful method of surveying the urinary tract, and can be done in the very young infant with safety and satisfaction.

It is not intended to give the impression that all urinary infections occurring in the infant or child are due to anomalies or other serious pathology; because certainly many acute infections, such as pyelonephritis and cystitis, occur without any apparent predisposing condition, respond very quickly to treatment, and that is the end of them. In those cases, however, which do not respond to an adequate course of therapy, which recur frequently, or become chronic, the physician should become suspicious that there is something interfering with the normal dynamics of the urinary flow, and investigate accordingly.

Any of the lesions described above, while affecting primarily the urethra or bladder, may if left untreated, result in changes in the ureters and kidneys eventually ending in renal failure. In addition, there may be interference with normal drainage in the ureter itself, due to anomalies such as ureteroceles, megalo-ureters, aberrant blood vessels and uretero-pelvic strictures, which if unilateral and unsuspected, may result in tremendous dilation of the ureter hydronephrosis, and if bilateral and untreated, may result in renal function being reduced to a degree incompatible with life.

Neoplasms

As a group, malignant tumors of the urogenital tract are least frequent. The most important of these tumors is the Wilms' tumor, or embryoma of the kidney. Testicular tumors are very rare in infancy and childhood. The prostate may undergo sarcomatous growth, and in the female, sarcoma botryoides may involve the bladder. These last two are highly malignant, and treatment usually purely palliative.

The results of treatment in Wilms' tumors have not been encouraging, but the outlook would appear to be no different from that in other malignancies, where it has been well demonstrated that the earlier the diagnosis and treatment, the better the results. Unfortunately, by the time many of these cases come to surgery, the tumor has spread locally or distantly beyond any possibility of total excision. The tumor has an insidious onset, and urinary symptoms or signs rarely occur in the early stages of growth. An abdominal mass, unilateral, is generally the first indication of such a tumor. This is usually noticed by the mother or nurse when bathing the child, or by the family physician on routine periodic examination of the child. Localizing pain is late in appearing, and hematuria, also because of the manner of growth and late invasion of the renal calyces or pelvis. Constitutional symptoms, such as anemia, malaise, fever, loss of weight, etc., are likewise rather late symptoms. In differential diagnosis of such an abdominal mass, one has first to consider the Wilms' tumor as being the most common retroperitoneal tumor occurring in children, and such things as neuroblastoma, hydronephrosis, solitary cyst of the kidney, polycystic disease, retroperitoneal sarcoma, etc., as being less likely. Investigation of such a mass by intravenous pyelography is usually sufficient to substantiate the diagnosis, and any delay in proceeding with further investigation and exploration of such a case should be avoided. Treatment consists of surgery and irradiation in various combinations. Some urological surgeons prefer pre-operative irradiation, with a further post-operative course. Others favor nephrectomy as soon as the diagnosis is suspected, followed by irradiation; while some rely on pre-operative irradiation plus nephrectomy. Irradiation alone is not suitable therapy except in those cases deemed inoperable. Of the possible combinations, the majority of urologists would seem to favor nephrectomy plus post-operative irradiation.

The prognosis in these cases is always very serious, and despite the radiosensitiveness of this tumor, it would seem that the only hope for improvement in results would be earlier diagnosis and treatment. In a recent statistical study of cases reported in response to enquiry of a large number of urologists, Abeshouse concluded that "there was a low operative mortality rate of 2.7% and a very high percentage of patients dying in the first post-operative year (54.3%) and a relatively low number of patients surviving two or more years."

Trauma

Among the traumatic cases admitted to the Urological Service have been minor ones of penile and scrotal laceration, readily dealt with by cleansing, debridement, and suturing where necessary. Urethral trauma frequently accompanies straddle injuries caused by bicycle crossbars, fences, tree limbs, etc., and varies from contusion to complete transection. Bleeding from the external urethral meatus, difficulty in voiding, and retention of urine are indications of urethral trauma. The possibility of subcutaneous extravasation of urine when the urethra has been partially or completely sectioned must be kept in mind. Catheterization is necessary in those cases unable to void and, once successfully passed, the catheter should be retained as a urethral splint if it is suspected the urethra has suffered actual laceration. If unable to catheterize, one has to assume a more severe degree of urethral injury, and exploration at the site of obstruction, with anastomosis of the urethra over a catheter, is necessary.

Minor degrees of renal contusion, with resulting hematuria, not infrequently accompany abdominal injuries associated with falls, automobile accidents, etc. Rarely is the renal injury of sufficient seriousness to warrant surgical exploration, with attempted repair or nephrectomy. The majority of these cases show diminishing hematuria over several days, and bed rest, with prophylactic antibiotic or chemotherapy, is all that is necessary. An intravenous pyelogram as early as possible following the injury is very valuable in indicating the degree of renal injury and in demonstrating a normal contralateral kidney if removal of the damaged kidney should subsequently become necessary. Following renal injuries, the development of sequelae such as fibrosis, renal contraction, hypertension, and calculus formation has to be considered and watched for during future years.

Torsion of the Testicle

Infarction of the testicle due to torsion of the spermatic cord is best considered under trauma, because it frequently follows sudden physical exertion, although it may occur spontaneously with no definite precipitating factor in the history. The sudden onset of acute testicular pain, often associated with nausea and vomiting, is the usual history. Characteristically there is no fever or leukocytosis, and the urinalysis is negative for pus and red blood cells. The affected testicle is usually high in the scrotum, enlarged, tender to palpation, and elevation of the scrotum does not alleviate the pain. After a few hours, the pain subsides, but the testicle continues to increase in size, and during the next few days the overlying scrotal skin becomes edematous or adherent. Fever and leukocytosis then develop. If the condition is recognized and surgical reduction effected within the first three to four hours, the testicle may not suffer too greatly. Occasionally, spontaneous reduction will occur, and some cases are seen which give a history suggestive of recurrent episodes of torsion with spontaneous reduction. If untreated, the testicular reaction may subside over a two to three-week period, and the testicle undergo progressive atrophy during the next few weeks.

All too frequently these cases are misdiagnosed acute epididymitis or orchitis. True orchitis in the infant or child is a very rare occurrence per se or as a complication of mumps or any other infectious disease, whereas postpubertal orchitis as a complication in mumps is not uncommon. Likewise,

epididymitis in children would be most unusual, since most children have not have not had the experience of a urethritis and prostatitis. Therefore, one can say that acute testicular pain of sudden onset in the infant or child, associated with an enlarged, indurated, tender testicle, is a torsion until proved otherwise by surgical exploration.

Hydrocoeles

A congenital hydrocoele, in which the fluid in the tunica vaginalis of the testis increases in amount when the infant strains or cries, is often apparent shortly after birth. This is due to the tunica vaginalis of the testis communicating directly with the peritoneal cavity by a patent vaginal process. In the majority of these cases, obliteration of the peritoneal process occurs within a few weeks or months, and the hydrocoele is cured. In some cases, however, it persists, and surgical correction is necessary, the technique being essentially that of repair of a congenital hernia, but in addition, the tunica vaginalis of the testis should be incised and everted. A true hydrocoele of the tunica vaginalis of the testis alone may also occur, causing a tense transilluminable swelling, gradually increasing in size. In this case, the surgical approach is through the scrotum as in an adult. Periodic aspiration should play no part in treatment of either type of hydrocoele in an infant or child. If not attended to within a reasonable time, the increased pressure on the testis may result in atrophy and impaired development of the germinal epithelium.

Circumcision

Since the last war, the practice of routine circumcision in infancy has become very widespread. Parents frequently request it, even though the doctor may perhaps find no real indication for performing the operation. One often quoted advantage is that the individual circumcised in infancy will not develop carcinoma of the penis. There are no very valid arguments against routine circumcision, but one fairly common and troublesome complication encountered following the operation in infancy is that of meatal ulceration and stenosis. If at the time of circumcision, notice is taken of the calibre of the meatus, and meatotomy done if this is judged smaller than normal, a meatitis will be less likely to develop. Robbed of its protective covering, exposed to the continual irritations of ammoniacal diapers, even the normal meatus may become inflamed and ulcerated. Crusting, fibrosis, and stenosis follow, with obstruction and difficulty in voiding. The best cure for such meatal ulceration and stenosis is an adequate meatotomy, but this must be followed by dilations periodically over at least a two-week period in order to prevent reformation of the stricture and a recurrence of symptoms.

Enuresis

This presents a problem for the mother, the family physician or pediatrician, the urologist, the psychiatrist, and last but not least, in later years, the child himself. Copious literature abounds on this subject, but no one appears to have provided the answer to the majority of these cases. Generally speaking a very small percentage of them are considered to be due to any organic pathology or abnormality of the urogenital tract. In the remainder, the condition is usually attributed to emotional factors associated with the child's environ-

ment. If the enuresis has existed for some years, without day frequency, incontinence, or pyuria, and renal function is normal as indicated by adequate concentrating ability of the kidneys, it is unlikely that one will find any organic cause for the bedwetting. If, however, one suspects, from the symptoms or findings on office examination, that there may be something physically abnormal, further investigation is warranted. Intravenous pyelography and cystoscopy are the next steps. A surprising number of these enuretic children are better following urethral and vesical dilation at the time of cystocopy. The reason for this is not too clear, although it would suggest that there is some urethral contraction or diminished bladder capacity accounting for the involuntary voiding at night, or it may be that this particular investigative procedure interrupts a long established cycle. Various drugs have been tried successively as each new one comes along—those aimed at relaxing and inhibiting bladder contraction at night, those designed to prevent too deep a sleep and permit the child to be aware of the stimulus of the stretching urinary bladder. Ingeniously engineered devices whereby bells ring and lights flash at the first drop of urine on the bed sheet have appeared. One usually runs the gamut of these and other remedies, and perhaps occasionally finds one that seems to result in improvement, probably entirely coincidentally. Psychiatry may find the cause of this behaviour pattern, but treatment along these lines still seems non-specific and inadequate. Fortunately, one can usually assure the parents that the child "will grow out of it" eventually.

Cryptorchidism

The child with unilateral or bilateral cryptorchidism always presents somewhat of a problem, for although the condition is not frequently encountered, there seems to be a great variation in opinions regarding the proper handling of such cases. Each case seems to present itself as an individual problem in management, because, so far, there seem to have been no definite rules laid down which will apply to every case. In reading the literature, therapy and theory seem to vary from author to author and year to year. Some of the questions that arise in dealing with cryptorchidism are:

What is the optimum age for undertaking treatment?

What is the place of hormonal therapy in treatment of the undescended testicle?

What is the best surgical method to employ?

Is there any real evidence that malignancy occurs more frequently in the maldescended testis than in a normal one, and if so, does orchiopexy influence this tendency in any way?

What is the result of therapy, particularly in relation to fertility?

Normally, the testes pass through the inguinal canal into the scrotum at about the end of the seventh fetal month, but it is not uncommon to find them in the canal at birth, with final descent occurring in the early post-natal period. The mechanism of the descent is not well understood, although there are many theories to explain it. It is sufficient at the present time to say that the major factor in normal descent is hormonal, and conversely in maldescent, one has to postulate either some hormonal deficiency or a testis which fails to respond normally to hormone stimulation, although mechanical obstructive factors do occur.

The diagnosis of cryptorchidism cannot be made merely by noting the absence of a testis or testicles in the scrotum. True cryptorchidism may be

defined as an arrest of the testicle at some point along its normal path of descent, and it has to be differentiated from two other conditions. The first and most commonly encountered is the highly retractile testis which can be regarded as normal in infants, young boys, and indeed, some children up to the age of puberty. In these cases the testis and cord are normal, and the testis has been down in the scrotum, but the strong cremasteric muscle can pull the gonad up into the inguinal canal intermittently or for protracted periods. Thus the testis can completely disappear from view, and this frequently occurs when the child is being examined by the doctor. In such a case, however, the testis can usually be grasped and pushed down into the scrotum, or it will be noted by the mother, when bathing the child or during periods of relaxation, that the testes are present in the scrotum. The fact that these testes come down into the scrotum and remain there at about the time of puberty probably accounts for some of the figures published indicating that a very high percentage of cryptorchid testicles descend into the scrotum at puberty.

The other condition is the ectopic testicle, where the gonad had become directed into some abnormal channel. This may be in the superficial tissues of the thigh, in the perineum, or superficial to the external oblique fascia. The last is a fairly common finding. On examination, the testis appears to be in the inguinal canal, but at operation is found superficial to the external oblique outside the external ring. This type is very satisfactory to deal with surgically, because the spermatic cord is long, and the testis can be readily transplanted into the scrotal sac.

In a true crytorchid, if one cannot palpate the testicle at the external ring or somewhere along the inguinal canal, it will most likely be found just within the internal ring, although it may be anywhere within the abdomen or may be entirely absent. The majority of cases of cryptorchidism have a potential hernia, and some an actual congenital indirect inguinal hernia. The presence of the latter is an indication for treatment at any age, and at the same time as repair is made, an orchiopexy is done. Other reasons for performing orchiopexy are to remove a testicle from a site such as the inguinal canal, where it is very liable to trauma, or to prevent the child from developing feelings of inferiority due to his abnormality. The primary reason for placing the testis in its natural environment, the scrotum, is, of course, to promote normal development of the germinal epithelium and spermatogenic activity in adult life. It is known that the germinal epithelium becomes active at about the age of six years, and becomes increasingly more active at about the age of eleven or twelve, and has reached its full maturity by the age of sixteen years. Hence it would appear that if one is to save the testicle, the optimum age for operation is about the age of six years. Unfortunately, however, there are no satisfactory statistics to prove that operation at the age of six years produces a better testicle than at nine or ten years.

In regard to fertility following orchiopexy, there have been no very satisfactory follow-up studies made. Those that are available indicate that in cases of unilateral cryptorchidism where the other testicle is normal, fertility is normal. In one series of twenty-five cases of bilateral cryptorchidism, fourteen had complete aspermia, but in another series, thirty out of thirty-eight cases showed fertility acceptable as normal by evidence of seminal fluid analysis.

The question of the undescended testicle having an increased incidence of malignant degeneration has been much discussed in recent years. Many statistics have been presented for and against, and most authors seem to criticize

the statistics and the method of obtaining them of their contemporary authors. It is, however, pretty generally accepted that there is a higher incidence of testicular tumor in cryptorchidism. Studies of cryptorchid testicles and biopsies of testicles taken at the time of orchiopexy indicate a fairly high incidence of faulty development of the testicular cells. This maldevelopment is in contrast to the faulty maturation which is described in any testicle which remains in the inguinal canal or outside of the scrotum at or about the time of puberty. Testicles which undergo embryonic tumor degeneration have also been shown to have a high incidence of this cellular maldevelopment, so that there is probably a definite association between this dysgenesis and the development of malignant testicular tumors of the embryonal type. It is likely, then, that there are at least two types of cryptorchid testicles; one which, due to a developmental defect, fails to descend and has a tendency to malignant degeneration, and which, even if placed in the scrotum before puberty, does not show normal spermatogenic activity; and the other which for some reason fails to descend, but if placed in the scrotum at the proper time, may develop quite normally and have no predisposition to neoplasia.

The optimum age for therapy is still a subject of controversy. The minority view advocates surgery within the first two years of life, and the majority, in the cryptorchid uncomplicated by hernia, tend to defer surgery until the ages of eight to twelve years. Various apparently valid reasons are given for the preference in the latter group. First, despite the fact that it is known that maturation of the germinal epithelium commences at about the age of five or six years, there is no proof that the testicle which is left until the age of ten or twelve suffers as a result of this. Secondly, many testicles which are undescended at the age of five or six years or younger have spontaneously descended at about the time of puberty. Thirdly, technically, the operation is more simple and satisfactory in the older child.

In regard to hormone therapy, varying results have been claimed, and a few facts have evolved. Hormone therapy, using a chorionic gonadtropin such as Antuitrin S, causes descent of the testicles only in those cases which would normally descend at puberty. It is therefore of value as a provocative test prior to the onset of puberty, because if descent occurs under the influence of therapy, nothing further need be done, and the patient is of an age where it is unlikely that the hormone will have any effect on his skeletal growth. If the testes do not descend, surgery can be proceeded with prior to the full pubertal changes taking place.

It is realized that this fails to answer definitely all the questions postulated at the beginning, and many others not mentioned, but the problems associated with cryptorchidism will probably only be solved through study and research in a large number of cases carefully followed by testicular biopsies and functional tests of treatment over a period of years.

An Evaluation of Urinary Frequency In The Female

W. Alan Ernst, M.D.

THE unfortunate female is pursued by the threat of urinary frequency from diaper life to old age. With each succeeding decade she must confront the common genito-urinary lesions of her age. So common is this complaint that few women reach middle age without at least a brief encounter with it. In many patients this encounter is both benign and transient, while in others it is both persistent and intractable, and in a few it may have a somewhat sinister significance. It may, of course, occur alone or with other urological symptoms or with symptoms suggesting disease in other systems. The etiological diagnosis is often simple, but at times it may tax the clinical acumen of the most experienced. Too often the approach to this symptom is one of defeat, in which a token reassurance is offered to the patient that she has a mild cystitis and that a few pills will eradicate her trouble. There is a certain similarity between the approach to urinary frequency and the approach to low back pain; as in the latter, if a careful history is taken and a thorough physical examination performed, then order is brought out of confusion, and often an etiological diagnosis can be made on the history and physical findings alone.

Enumeration of the underlying lesions that produce urinary frequency in women will give a better appreciation of the problems involved in establishing the causes of this complaint. These have been variously classified but, for the purpose of this discussion, they can be divided into three general groups; namely, (a) general or systemic, (b) lesions in the surrounding organs but extrinsic to the genito-urinary tract, and (c) lesions in the genito-urinary tract itself.

The general or systemic causes include anxiety, menstruation, diabetes insipidus, diabetes mellitus, and chronic renal disease.

Urinary frequency produced by mental anxiety has certain characteristic features. It is usually unassociated with other urological complaints, and the frequency is marked during the waking hours but may be completely absent during the night. If the urine is examined, it will usually be found free of pathological cells. In frequency associated with acute anxiety the diagnosis is usually obvious, but it should not be overlooked in less obvious cases, since urinary frequency is a common somatic expression of chronic anxiety.

Occasionally the menses are associated in adolescence with urinary frequency, but the cause is soon recognized by the periodic recurrence. As the patient develops into womanhood, the condition often disappears without treatment. Diabetes insipidus is a rare condition in which the diagnosis may be suspected by the low specific gravity of and the absence of pathological elements in the urine. Diabetes mellitus is a relatively easy diagnosis, as the urinalysis is usually positive for sugar. Recurrent attacks of lower tract in-

fection are extremely common in the diabetic woman, and while, in most cases, the infection is low-grade in nature, it may be sufficiently severe to require additional insulin. Chronic non-obstructive renal disease, when well advanced, is usually easily recognized, but a urine concentration test and blood biochemical tests may be required to detect its presence in the less well advanced lesions.

Urinary frequency from lesions in the surrounding organs but extrinsic to the genito-urinary tract is not surprising when one considers the intimate embryological vascular, lymphatic, and nervous relationship of the urinary tract to the genital tract and the terminal bowel. Tumors, cysts, and infections of the uterus, ovaries, fallopian tubes, colon, and rectum can readily involve the urinary tract to produce a variety of urological symptoms, including increased micturition. However, in most cases, the primary diagnosis has been made, and the onset of urinary frequency indicates involvement of the urinary tract.

The lesions in the urinary tract itself are, of course, the most important causes of frequency in the female and, since almost all lesions of the genito-urinary tract are capable of producing frequency, little is to be gained by a detailed enumeration of these lesions. If, however, the lesions are grouped according to the various regions of the urinary tract, they can be considered and evaluated more advantageously.

It is sometimes forgotten that relatively far advanced disease can exist in the upper urinary tract without upper urinary tract symptoms. Infection in the kidneys and the kidney pelves can progress insidiously without inconvenience to the patient, and only when the infection eventually invades the sensitive trigone will it produce urinary frequency. The mechanism by which infection spreads from the upper urinary tract to the bladder and trigone is probably by direct extension along the tract itself. There are some who feel that the lymphatic and haemotogenous routes are the important ones, but discussion of this controversy is not relevant here. One of the best illustrations of this mechanism is seen in genito-urinary tuberculosis, in which increased urinary frequency is often the earliest symptoms of the disease. However, when genito-urinary tuberculosis is the cause of urinary frequency, in most cases the condition is progressive, and generally followed by dysuria, urgency, and haematuria as the disease becomes established in the lower urinary tract. Large renal calculi or hydronephrosis, secondary to uretero-pelvic obstructions of various types with secondary infection, are also examples of upper tract lesions which produce the lower tract symptom of frequency.

Primary lesions in the ureter are a rare cause of urinary frequency, but it is an accepted fact that irritation of the lower ureter may produce a *reflex* type of frequency.

Pathological conditions in the bladder and urethra are the most common causes of urinary frequency in either male or female, and of these, infection is the most important. Perhaps this explains why many consider frequency in the female and cystitis as synonymous. Cystitis, ranging from acute to subacute to chronic, rarely exists as an isolated clinical entity, and in dealing with it the problem resolves itself into finding and eradicating the precipitating factor, frequently to be found in bladder or urethra or occasionally in the vagina.

In dealing with urinary frequency in the female, certain specific types of "cystitis" must be considered; namely, proliferative cystitis, interstitial cystitis, and radiation cystitis.

Proliferative cystitis is an all-inclusive term which may be used to cover a group of closely allied inflammatory lesions of the bladder characterized by the

formation of granulations, nodules, and small cystic cavities. Of these the most common is cystitis cystica. The etiology of these lesions is generally considered to be a low-grade, non-specific infection that has been present for a prolonged period. As in acute cystitis, there is usually some precipitating lesion present. Here, however, the history and physical examination give little indication of the presence of the lesion, since the diagnosis must be made on cystoscopic examination.

Interstitial cystitis differs from proliferative cystitis in that it produces a more marked degree of urinary frequency and is usually associated with varying amounts of suprapubic pain. The endoscopic findings in interstitial cystitis, unlike those in proliferative cystitis, may be difficult to recognize until the disease is well advanced. More important on the positive side is the fact that interstitial cystitis can be easily diagnosed by measuring the bladder capacity.

Radiation cystitis produces a symptom complex similar to interstitial cystitis, but the history of radiation therapy, usually for carcinoma of the cervix, will make the diagnosis evident. However, it is not generally appreciaed that the symptoms of chronic radiation cystitis may not appear until one or two years after the time of treatment. Proliferative cystitis, interstitial cystitis, and radiation cystitis have one common feature in that all three may produce marked urinary frequency and in the presence of a relatively normal urinalysis.

Other vesical lesions that may cause urinary frequency in the female are early neurological lesions involving the nerve supply to the bladder. If such a lesion is a slowly progressive one, then urinary frequency is likely to be the earliest expression of bladder nerve supply involvement.

Bladder tumors, while they are often associated with frequency of micturition, do not usually present themselves with this symptom. Bladder calculi are relatively rare in the female as compared to the male, and consequently they are rare causes of frequency in that sex. Cystocoele in the female may cause stress incontinence, but this is usually not associated with urinary frequency unless secondary infection is superimposed.

The female urethra, although short in length, is subject to a long list of pathological lesions. Situated in the vestibule in a trough between the labia minora, it lies in an exposed position, and because of this, it is bathed by rectal, uterine, and vaginal discharges from infancy to senility. It is also exposed to venereal infection during coitus, and during labor and gynaeclogical operations it may suffer injury. The intrinsic anatomy of the urethra also enhances the possibility of infection. The female urethra was formerly considered to be a short, straight tube that served to empty the bladder, but more recent studies of sections through the urethra, both in autopsy and surgical specimens, have shown numerous glands of considerable length that form an ideal habitat for pathogenic organisms. Urethritis and urethrotrigonitis are common lesions responsible for increased micturition in women of all age groups, and when dealing with them the primary source of infection must be sought. This may be found in the vagina in patients with non-specific trigonomal or gonorrhoeal leucorrhoea, but more frequently the source of infection or the precipitating lesion is found in the urethra itself. Since the urethra is a superficial structure, many of the lesions that disturb its function can be either visualized or palpated if a vaginal examination is performed. Such lesions as a urethral caruncle, prolapse of the urethral mucosa, cyst, diverticulum, or tumor of the urethra and synechia vulva can be easily diagnosed if the patient is properly examined.

Stricture of the female urethra, because of its common occurrence and universal neglect, merits special mention. Normally the adult female urethra

will easily admit a No. 26 or 28 Fr. sound, and most urologists will diagnose a urethral stricture if the urethra is snug to a No. 20 Fr. sound. Most commonly the stricture is situated at the external meatus, but it may be located anywhere along the course of the urethra. Urethritis or urethrotrigonitis secondary to urethral stricture is the most common single cause of urinary frequency in the female. A catheter specimen is worth the trouble when considering this symptom, and if a No. 20 F. metal catheter is used for the purpose, then the urethra can be calibrated, and the information obtained will serve as a guide for subsequent dilation.

A much less common and more frequently neglected lesion than stricture is contracture of the vesical neck. This lesion may be extensive enough produce very definite symptoms without being detected by urethral calibration, and an endoscopic visualization by an experienced examiner is required to establish the diagnosis. Fortunately, mild cases of contracture of the vesical neck respond well to a generous dilation of the urethra, but the more advanced and recurrent ones will require a transurethral resection at the vesical neck to produce good results.

The causes of urinary frequency in the female are many, and the diagnosis is sometimes difficult, but they will be less of a diagnostic and therapeutic problem if the vagina and urethra are examined and if the urethra is calibrated and dilated when indicated.

Prostatism In General Practice

Clarence L. Gosse, M.D.

BY definition, prostatism is interpreted as bladder neck obstruction. There are three distinct types, etiologically, pathologically, and therapeutically. They are prostatic hyperplasia, bladder neck contracture, and prostatic cancer.

The first thought which comes to mind in discussing this subject, is how truly remarkable has been the advance in prostatic therapy in the past ten or fifteen years. For this the credit can be laid at the doorstep of practically all types of medical practitioners. Some of these advances include improvement in anaesthesia, better methods for the estimation and control of tissue chemistry, newest urinary antiseptics and antibiotics, a different conception and additional therapeutic agents by the cardiologist or internist, and early diagnosis and requests for therapy by the general practitioner. I might add, with reference to the last of these, the practitioner's realization that prostatic surgery has become a highly technical procedure which should be done by surgeons working in an ideal hospital set-up, where there is available capable, trained nursing personnel and adequate laboratory control. I have purposely left out the easy availability of blood because it has little place today in prostatic surgery, except as a preparatory build-up for the anaemic patient. In fact, I feel that the administration of blood transufsions without specific definite indication can account for more post-operative deaths, both directly and indirectly, than the operation itself. This applies with particular emphasis to patients under anaesthesia, where transfusion reactions are not manifest and, occurring more often than we realize, account for some of the post-operative complications for which we can find no apparent cause.

I have mentioned the role of the anaesthetist, the biochemist, the bacteriologist, the cardiologist, the general practitioner, and the nurse. To these I would like to add a tribute to a highly efficient interne-resident team as one of the most potent factors in successful prostatic surgery.

In view of all these advances along so many lines, it was only reasonable for the urologist to avail himself of all of them and, as it were, to take quick advantage of the achievements of others. This he has done. Indeed, it is not too much to say that the advance in prostatic surgery in the past decade has been unparalleled by any other surgical procedure.

It is my intention to confine this discussion to points which may be of interest to the practising physician, those which might be involved in questions which are often asked of the physician by patients, rather than to stress the technical details of the operative procedures, though I believe there is value in the practitioner knowing to what his patients are being subjected.

I am going to start with the function of the prostate and what might be expected following its removal. First of all, we know very little about the purpose and uses of the prostate gland. We know it is a sex gland, but it has a doubtful influence on sexual impulses. It produces a hormone of indefinite action and about which we know very little. We know that pre-puberty castration prevents the prostate from developing. After puberty, castration seems to have little effect upon it except when malignant change has occurred. We know that the prostatic secretion serves as a vehicle for spermatozoa and forms the bulk of the ejaculate.

What happens following removal of the prostate as regards sexual desire, potency, and sterility? There is no reason why sexual desire or potency should be affected. It is altered, however, in a small percentage of cases, but I think a good many such changes stem from the psychological reaction and a gradual loss of interest. This is particularly obvious in the older age group. Following enucleation of the prostate, the patient is, of course, sterile, because the ejaculatory ducts are severed as they enter the prostate. In some cases where bladder neck obstruction is removed by transurethral resection, fertility is not impaired. There is, of course, no ejaculation following enucleation of the prostate gland, even though satisfactory intercourse may be accomplished. The patient often states that "intercourse is satisfactory but I shoot blanks."

What makes a prostate enlarge? Hypertrophy of the prostate is really a misnomer. Enlargment of the prostate is the result of adenomatous growth, either single or multiple, and may be in any one or all of the lobes. This adenoma compresses the normal prostatic tissue towards the periphery, and this compressed tissue forms the false fibrous capsule. Occasionally this compressed tissue does not become completely fibrotic, and sometimes a new adenoma forms, or carcinoma develops in the false capsule of the prostate. While many theories have been advanced as to the etiology of these adenomata, no definite information at the moment is available.

What happens to the remainder of the urinary tract when the prostate enlarges? First of all, obstruction develops in an insidious manner. The main bladder muscle, the detrusor, and the trigonal muscles hypertrophy. This is in response to an increased effort to urinate. The bladder wall thickens, and by virtue of this thicker wall, the bladder capacity diminishes. This diminution in capacity automatically brings about urinary frequency. At this stage, the bladder is compensated, and is usually able to empty itself at each voiding, leaving no residual urine. Sometimes this takes considerable straining, as a result of which some of the muscle fibres give way, the mucosa pushes through these fibres, and saccules or diverticula develop. As the obstruction increases, this compensation eventually gives way to decompensation. The bladder wall then dilates and becomes atonic, residual urine occurs and increases, urination becomes more frequent and increasingly difficult, until a complete urinary retention ensues.

The ureters and renal pelves are affected in exactly the same way as the bladder. The increased tone and thickness of the bladder muscle increases the resistance at the lower end of the ureters. The thin musculature of the ureters and pelves responds as well as it can to the increased resistance by an attempt at hypertrophy. However, the muscle walls of the ureters and pelves are in no way able to compete with the hypertrophy of the bladder, and as there is no pelvi-ureteral valve to protect renal function, such as there is at the ureterovesical junction, the intraureteral pressure is immediately transmitted to the renal pelvis, calyces, and tubules, producing an immediate pressure effect on the renal parenchyma.

The symptoms, therefore, of developing urinary obstruction are obvious: frequency—particularly at night, smaller stream, and hesitancy in initiating the flow. Not infrequently, an attack of urinary retention heralds the diagnosis where few previous urinary symptoms existed. Occasionally, gross hematuria is the first symptom or sign. This usually results from varicosities, which have ruptured following straining.

How severe should symptoms become before treatment is advised? This is a difficult question to answer, as it depends on the age of the patient, the condition of the patient, the size of the gland, and one's clinical judgment. Young men in their fifties and sixties should have prostatectomy early, provided there is definite glandular adenomatous hyperplasia. Symptoms in the younger age group, however, are often due to prostatic infection or stricture, rather than actual prostatic hyperplasia. Cystoscopic visualization of the gland and bladder is usually necessary before a decision can honestly be made. In the seventy and eighty-year-old groups with minimal symptoms, one tends to temporize, unless such symptoms become increasingly troublesome. Urinary retention, dribbling, excessive nocturia, and large residual or infected urines are definite indications for treatment.

What methods of treatment are available? Any relief of prostatic obstruction must be by surgical means. Hormones are useless, and may even be harmful except in carcinoma. We advocate three types of operations; namely: transurethral resection, retropubic prostatectomy, and transvesical or suprapubic prostatectomy. The decision as to which operation is done depends on the findings on the intravenous pyelogram, at cystoscopy, and on rectal examination. The age and condition of the patient and the result of a medical consultation is a prime consideration. Sometimes it makes little difference which type of operation is done, and in these instances, it becomes a toss-up or may vary with the urologist concerned. A review of 1000 cases which underwent surgery at the Victoria General Hospital between 1952 and 1956 includes:

Transurethral resections	— 533
Retropubic prostatectomy	— 381
Suprapubic prostatectomy	— 66

In other words, 54% were done by transurethral resection, and 46% by open operation. The average age for this group was 69 years, and the average hospital post-operative stay was 15 days. The over-all mortality rate was 1.2%

What determines post-operative results? There would appear in our series to be two general factors:—

1. The decisions leading up to and surrounding the actual operative procedure—pre-operative assessment and care.
2. The post-operative regime, including nursing, interne and resident service.

As regards pre-operative care, several points might be mentioned:—

(a) We assume all patients in the prostatic age group to be poor risks, and certainly many are very bad indeed.

(b) Medical consultation is practically the rule. However, if the patient is able to walk and is not in cardiac failure, we usually end up by operating any way. Very few patients are sent home on catheter drainage or without surgery.

(c) Special attention is given to the patient's chemical balance and to his haemoglobin estimation. This latter is one indication for blood transfusion, and we do not hesitate to use it pre-operatively to build the patient to the highest possible peak of condition for surgery.

(d) Routine cystocopy to determine the size of the gland and type of the obstruction, in order to obtain the best indication as to which operation is advisable.

(e) Special attention is given to:

1. Obese people with protuberant abdomens, where we favor
   (a) For small or medium-size glands—transurethral resection.
   (b) For large glands—retropubic prostatectomy through transverse incision.
2. Size of penis. The calibre and length of urethra may preclude transurethral resection. A small calibre urethra is prone to post-operative stricture, and is best handled by open operation. Urethrae from at least one section of the province tend to be abnormally long, and in some instances it is not possible to reach the inner bladder surface of the prostate with the resectoscope. Open operation is then advised.
3. Anaesthesia. We feel that a spinal anaesthetic is easier on the patient, and allows the surgeon to do a more careful and more efficient operation.

As regards the post-operative program, much has been said already. I would stress that the type of post-operative care, especially during the first twenty-four hours, is vital to a favorable outcome. Here the nursing, interne-resident service which we instituted in 1951 has brought about a tremendous change. We feel that this post-operative vigilance with trained personnel is the greatest single factor in reduced morbidity and mortality figures. Practically all these patients give the surgeon one good chance. If this chance is muffed and a needless complication ensues, a fatality is very likely to occur. If we have laid a good foundation for a favorable result, we can expect a happy outcome and essentially no likelihood of mortality.

Complications and sequelae. Certain misfortunes are possible following any form of prostatectomy, and occur not infrequently. These are the bugbear of the prostatic surgeon and the cause of many sleepless nights. Such complications can, for simplicity, be divided into immediate and late groups.

Immediate complications, that is, those which occur while the patient remains in the hospital, comprise the following:

1. **Excessive immediate post-operative bleeding.** Fortunately, today this is a rarity. When it does occur such patients are returned to the operating room, where the bleeding point is fulgurated through the resectoscope, regardless of whether the original operation was open or closed. Occasionally these patients are given blood transfusions, but only if there is considerable blood loss.
2. **Thrombophlebitis, pulmonary embolus, and pulmonary atelectasis.** Early post-operative mobility has practically done away with these complications. Atelectasis is possibly the most common complication of these three. Occasionally we see some degree of thrombophlebitis, but rarely do we see pulmonary embolism.
3. **Post-operative wound infection.** This still is our most frequent trouble maker. Early and active mobility is hard on wound healing, and lights up any latent infection. In this we have been helped by various antibiotics, but there is still no panacea in this respect.
4. **Secondary hemmorhage.** This may occur during the second post-operative week, and is usually a result of infection in the prostatic bed. Such cases occasionally have to be returned to the cystoscopy room for fulguration.

Late complications;that is, after the patient is discharged from hospital. This type of complication is more frequently seen by the patient's personal physician, and may be listed as follows:

1. **Continued urinary infection.** Causes of this are:
   (a) Residual urine as a result of an atonic bladder.
   (b) Diverticula of the bladder which may have been missed at the time of cystoscopy.
   (c) Incomplete removal of the gland or capsular remnants. Such infections are usually controlled by the judicious use of urinary antiseptics. It may take three months for the prostatic cavity to heal completely, and urinary antiseptics may have to be used intermittently during this time. The choice of antiseptics includes the sulfas and mandelates Rarely is it necessary to resort to the antibiotics.
2. **Urethral stricture.** If infection continues, one should not hesitate to pass a metal sound, No. 20 Fr. or 22 Fr. size, in order to test the calibre of the urethra. Strictures may occur just inside the external urethral meatus from ulceration due to the indwelling catheter post-operatively, at the peno-scrotal junction for the same reason, or more rarely, at the bladder neck from cicatricial contracture associated with post-operative infection.
3. **Incontinence.** In the 1000 cases reported, there were sporadic cases of partial incontinence at the time of discharge from hospital. Most of these cleared completely within a few weeks. We know of two cases still with some difficulty, but have not been able to follow all cases. There has been no incontinence from any retropubic operation.
4. **Suprapubic fistula.** This only results from suprapubic prostatectomy, and usually follows continued obstruction either from stricture or incomplete removal of the gland. I know of no such cases in this series.
5. **Osteitis pubis.** There was one mild case in this series, which cleared spontaneously.

Carcinoma of the Prostate.

Carcinoma of the prostate is the commonest cancer of any internal organ in the male. In this series of 1000 prostatectomies, 19.5% were diagnosed pathologically as carcinoma.

The diagnosis of prostatic carcinoma rests almost entirely in the hands of the general practitioner. Often the condition is advanced before there are any symptoms, and when symptoms occur, it is usually too late for anything but palliative surgery. Carcinoma of the prostate is not necessarily associated with hyperplasia; in fact, in the early stages, a high percentage of cases have very little glandular enlargment associated with them. The early and initial diagnosis of carcinoma of the prostate is made on rectal examination. There is felt a sense of hardness that is stone-like, and in fact, prostatic cancer is often diagnostically confused with prostatic calculi. It is often stated that prostatic carcinoma develops more frequently in the remnant of the posterior lobe. This is perhaps true, but carcinoma of the prostate can develop in any section of the gland, and one should not be misled by incidence when one finds an isolated lesion at or near the periphery. Early cases of prostatic cancer, which are the only types that can be cured, must be diagnosed first on rectal examination.

It is therefore vitally important, if such cases are to be located in time, that rectal palpation be done on all males over the age of fifty whenever an examination is carried out.

Elevation of acid phosphatase, an enzyme produced by prostatic tissue, must not be construed as a test for early prostatic malignancy. True, the more prostatic tissue present, the more acid phosphatase will be found in the blood. However, unless there is a very great increase in prostatic tissue, such as is seen in the presence of metastatic lesions in the bone or chest, the acid phosphatase will not be significantly raised. It is therefore a test to be used only as confirmation when metastatic lesions are seen radiologically. As a test for prostatic malignancy when the lesion is confined to the region of the gland, it is useless.

Prostatic carcinoma is usually a slowly growing cancer. Many patients live out a normal span of life with carcinoma of the prostate, without having this lesion account for mortality. Other prostatic cancers, particularly in younger individuals, grow rapidly, and act in the same way as rapidly growing cancers in other parts of the body, with the same usually fatal outcome. The majority, however, by virtue of overgrowth of tissue, cause symptoms similar to those of benign prostatic obstruction, and require some form of palliative surgery.

Early cases of carcinoma of the prostate, that is, those cases where the tumor is confined to the gland, can be cured by radical prostatectomy. This includes total removal of the prostate, the capsule, the seminal vesicles, and the lower half inch of bladder, and anastomosis of the bladder to the membranous urethra. Unfortunately, such cases represent less than 10% of our total prostatic cancers, but the results in them have been excellent.

Palliation is the only treatment left for the remaining 90%. This treatment includes transurethral resection in patients with obstruction, with the addition of hormone therapy. Hormone therapy is based on the knowledge that androgens hasten the growth of most types of prostatic carcinoma. On withdrawing these androgens by means of castration or by neutralizing their effect with estrogens, or a combination of both, the growth of such tumors can be slowed and the patient kept relatively comfortable for an indefinite period. Sometimes even following castration, there is still a generous supply of androgens delivered by the adrenal glands. Surgical adrenalectomy or medical suppression of the adrenals by means of cortisone sometimes provides a measure of relief.

As to the outcome, we have no way to prognosticate accurately. Each patient is treated as an entity, and the extent of the surgical procedures depends upon the extent of the lesion as well as the age and condition of the patient. The majority live in reasonable comfort for many years, and with the judicious use of available therapy, this should be the rule rather than the exception.

The Urinary Calculus

F. Gordon Mack, M.D.

THE story of urinary calculus disease is well recorded in the history of medicine, and in fact, it made urology one of the earliest fields of specialization. Hippocrates, some 400 years B.C., charged physicians "that neither will I cut them that have a stone, but will leave this operation to those who are accustomed to perform it." Since then, much has been learned about the causes of stone formation, but in many cases we still do not know the exact etiology of the urine crystallization.

Probably one of the most important factors etiologically is the volume and relative concentration of the urine. The lower the volume of output and the more concentrated the urine, the greater is the tendency for crystallization to occur, and the converse can be readily applied in prophylaxis against recurrence The reaction of the urine is another factor influencing calculus formation, certain types of calculi tending to form in an acid urine and others in an alkaline urine. Changing the pH. of the urine should therefore be of value in preventing further formation of stones. Diet also may play a part in predisposition to calculi—an excess calcium intake resulting in hypercalciuria, a high purine diet reflected by a high uric acid output, vitamin A deficiency leading to an unhealthy condition of the epithelium of the urinary tract, or an excess of vitamin D again causing hypercalciuria. Increased excretion of calcium in the urine may also occur in hyperparathyroidism due to a parathyroid adenoma; and it is good routine in all cases of calcium stones, particularly if recurrent, to determine the blood calcium and phosphorus, and in suspected cases, to proceed further with X-ray studies and urine calcium excretion studies. Immobilization and recumbency likewise, because of skeletal decalcification, result in hypercalciuria and promote the formation of renal stones. Stasis and infection, practically always secondary to some obstructive pathology in the urinary tract, are perhaps the best recognized causes of renal lithiasis.

It can be seen that practically all these known causes or predisposing causes of calculi have to do with the relative concentration of the precipitating ions, due to either their increased output or urine concentration. Much research work has been done on the crystalloid and colloid composition of the urine and chemical examination of actual calculi, in an attempt to determine the exact cause of crystalluria. Not too much attention, however, has been paid to the organic matrix of the calculus, upon which the inorganic crystals deposit. It would seem now that this latter approach may well be the one which will yield more information about the exact cause of stones, and perhaps enable us to be more specific in treatment and particularly in prophylaxis.

The treatment of the symptoms and of the actual calculus or calculi is quite well standardized. The recurrence rate in renal calculi, however, is high—some figures give it as 20 or 25%—and it is in prophylaxis against recurrence that we are mainly concerned here.

First, let it be said that there is no way of dissolving large stones and permitting them to pass by giving oral medication. Dissolution of calculi in the bladder or even the renal pelvis may in certain cases be brought about by continual lavage over a period of time with an irrigating solution such as "G" or "M", which is a citric acid buffered solution of an acid pH. The release of

obstruction by surgical methods, either endoscopic or by open surgery, the treatment of infection, and the maintenance of a sterile urine is at the present time the best way in which we can prevent recurrences, for it is in the stones that have formed in infected urine that the recurrence rate is highest. Many of the bacteria found in infected urine have an enzyme which breaks down urea, the hydrolysis resulting in ammonia formation with a highly alkaline urine, favoring the precipitation of phosphates. The urine can be checked periodically by the physician for pus, and if bacteriological facilities are available, the urine can be cultured. While initially the antibiotic to which the infecting organism has been shown to be sensitive is of value, the long term therapy which is so often necessary in these cases best makes use of one of the urinary antiseptics, such as the sulfas or mandelic acid compounds, in moderate dosage and in intermittent courses.

Increasing the patient's fluid intake to ensure a large volume output of dilute urine is one of the other very important factors over which we may exercise control. In dietary therapy, a low calcium, low phosphorus diet is often recommended when the stone has, on analysis, proved to be a calcium phosphate or mixed calcium one. A low purine diet can be used in those people who have formed uric acid or cystine stones. The acid and alkaline ash diets have been replaced, to a large extent, by other forms of therapy. The purpose of the acid ash diet was to promote an acid urine, but this is more easily accomplished by maintaining a sterile urine at its normal pH. The alkaline ash diet may still be used in cases of uric acid or cystine stones, where it is desirable to maintain an alkaline urine; but it is not a very palatable diet, and the alkalinity is fairly readily maintained by some alkalinizing agent, such as sodium citrate or sodium bicarbonate.

Physiotherapy and improved nursing techniques, plus an awareness of the gravity of complicating urolithiasis in many recumbent or immobilized patients, has resulted in a regime for these people which has practically eliminated this complication.

Many other forms and methods of therapy aimed at preventing recurrence have been advocated from time to time. The administration of one of the aluminum hydroxide gels, such as amphojel or basaljel, in a dosage of four ounces a day, is effective in limiting absorption of phsphates from the intestinal tract and hence reducing the amount of phosphates being excreted in the urine. This may be of value particularly in those stones formed in infected urine and where it is difficult to eliminate the infection. The injection of hyaluronidase was for a time thought to be effective in increasing the solubility of salts in the urine by virtue of the protein excreted in the urine in response to its administration. The citrate content of urine is known to exert quite a degree of influence on the solubility of calcium and, since estrogen administration increases the urinary output of citric acid, hormonal therapy was, for a time, hoped to be an answer to the problem of the recurring calcium stone. While administration of vitamin A may be helpful in aiding repair of a damaged urinary tract epithelium, the incidence of the stone formers suffering from a hypovitaminosis A must be very small. A relatively new approach to the problem of medical therapy and prophylaxis in calculus disease has been the use of aspirin. The background for this is the observation by some research workers that the presence of substances in the urine identified as glucuronides increases the solubility of calcium phosphate. The administration of certain compounds orally, such as the salicylates, increases the urine glucuronide output, and hence, it was

hoped, would increase the solubility of calcium salts. While this has shown promise, it is still too early to assess accurately the results of such therapy.

Thus it may be seen that an appreciation of the signs and symptoms indicative of calculus disease of the urinary tract has existed for a long time. Diagnostic methods and definitive treatment have become an exact science. Etiology and what might be called the medical aspects of this problem are, in the majority of instances, unknown. We will anticipate that the research urologist and biochemist will provide further enlightenment in the problem of etiology.

In the prevention of urinary calculi, it would seem to be of most importance to avoid supersaturation of the urine. An adequate fluid intake, particularly during the hot weather, when more prone to dehydration; dietary limitation; and certain medication aimed at reducing to a minimum the urinary excretion of calcium, phosphates, oxalates, uric acid, etc., will help in accomplishing this. Treatment and prevention of infection is another most important method of prophylaxis. In addition, in a limited number of patients, some of the other methods of therapy mentioned may be useful.

Infertility In The Male

W. Alan Ernst, M.D.

ANTONJ van Leeuwenhoek, the inventor of the microscope, related that a member of the medical profession, Doctor Hamm, first viewed with interest the motile spermatozoa in 1677, but until the beginning of this century, our profession was reluctant to accuse the male as a possible cause of barrenness. The eminent and courageous Dr. Marion Sims dared to challenge and threaten the almighty male ego by such a suggestion, and found cause to defend his belief, as he stated: "I was misrepresented, maligned, and positively abused, both here and abroad; for dabbling in the vagina with a speculum and syringe was considered to be incompatible with decency and self-respect." That statement was made some sixty years ago, and the male of today still clings to his delusion of potency until he is backed into the office of a urologist by his wife and his wife's obstetrician.

Infertility has been defined as failure of conception after twelve months of adequate cohabitation. About ten per cent of marriages are barren, and of this group the male is the etiological partner in about forty per cent of cases. Some degree of insight into causes of infertility in the male is obtained by consideration of the pathogenesis of this condition. The pathogenesis may be considered under three general headings. The first and most important of these are the deficiencies in maturation of the germ cell, since 95 per cent of infertile men have intrinsic spermatogenic defects ranging from complete germinal aplasia to partial maturation arrest of the germ cells. The second group includes obstruction of the conducting system, both congenital obstruction and absence of the ducts and obstruction due to acquired infection. In the third group are the more uncommon causes, such as endocrine disturbances, which include hypothyroidism and pituitary disturbances and disturbances of the adrenal glands.

The investigation of the potentially infertile male requires a detailed history and a complete physical examination, and included in the history should be a careful evaluation of the marital habits of the patient. Investigation of a barren marriage presents a unique situation in medicine, in that two individuals must be examined for a common problem. The routine laboratory procedures performed on the patient should include urinalysis, a prostatic smear hemoglobin, sed. rate, and white blood count. Other investigative procedures are utilized as indicated by the history and physical examination, and of course, the most important test is the semen analysis. This examination is relatively simple, and the technique will be found described in any number of standard tests.

The evaluation of the semen analysis, however, is not entirely didactic and free of controversy. The value of the semen analysis rests on the assumption that it affords a means of prognosticating the likelihood of conception. The evidence that such a value may be given to the semen analysis is based on a comparison of semen specimens of a large number of men who are fathers and the specimens of husbands who have no children. It was Dr. John MacLeod, of Cornell University, who, by his analysis of over 2000 semen specimens in both fertile and infertile males, contributed in a large measure to the standardization and evaluation of the semen analysis. The important features of the

semen analysis that will allow some degree of prognostication of the patient's fertility potential are the spermatozoa count per c.c., motile activity, and the morphology.

The relationship of the spermatozoa count to fertility has been and still is a debated subject. Earlier literature quoted the normal sperm count per c.c. as 60,000,000 to 150,000,000. This, however, was later lowered to 40,000,000 and more recently, MacLeod has stated that a count of 20,000,000 may be a truer level of the male fertility constant. In his own studies, MacLeod found 5 per cent of fertile men with counts between 1 and 20 millions. This is probably caution enough against labeling a patient with a count of 10,000,000 or less as infertile.

Although the cell count has proved to be an important feature in the prognostication of male fertility, it has, in recent years, been displaced from the limelight by the emergence of the motile activity of the spermatozoa as the dominating factor in human male fertility. The importance of good motility was illustrated by MacLeod in a follow-up of men in his infertile group who eventually produced conception as compared with those who did not. Two and one-half times as many men with good motility eventually produced conception as did men with poor motility. When the percentage of active cells was below 40 per cent, the conception delay was longest, but if the percentage of active cells was 60 per cent or above, conception took place significantly sooner. The cell count per c.c. and the degree of motility are the two most important qualities of the semen analysis used in the prognostication of male fertility. The relationship of these two qualities to frequency of intercourse in the normal male and in the relatively infertile male has recently been demonstrated. While both the count and the motility decreased with frequency of intercourse in the normal male, the decrease is relatively insignificant. However, in the relatively infertile male, there is a marked reduction in his fertility potential, as evidenced by a decrease in the motility and also the total count, which is produced by too frequent ejaculation.

The morphology of the spermatozoa is also important, but it tends to follow closely the quality of the count and motility. In those patients with good count and good motility, the number of normal forms was above 80 per cent in MacLeod's series, but with poor motility and low count, the number of abnormal forms increased. MacLeod and Gold also studied the relationship of morphology to miscarriage and other pregnancy accidents, and they concluded that they were unable to demonstrate a relationship between the abnormal sperm morphology and miscarriages.

It would appear then that the semen analysis, coupled with other data related to the marital habits, affords the means of general evaluation of the chances of conception. This appraisal is not absolute in the sense that the boundary line between fertility and infertility can be drawn. Such an absolute decision is warranted only at a point where the semen is devoid of spermatozoa.

The Treatment and Management of the Infertile Male

The general attitude of many practitioners towards the subfertile male is one of apathy and hoplessness. This attitude, no doubt, stems from the knowledge that about 95 per cent of **infertile** men have intrinsic spermatogenic defects, ranging from complete aplasia to partial maturation arrest. However, the **subfertile** male can be helped, and our responsibilities do not end with the semen analysis. Such general measures as proper rest, freedom from undue

tension, good nutrition, elimination of tobacco and alcohol, discontinuation of wearing jockey shorts, and the elimination of infection, while they may seem unimpressive, are important. The timing and frequency of intercourse, as previously mentioned, are of importance to the subfertile male. If thyroid or pituitary deficiencies are suspected, these, of course, must be investigated and treated.

More direct therapy of the subfertile male is directed on the seminiferous tubules themselves. Medications began with lotions and potions, and later progressed to the vitamins, and when these were exploited, we turned to the hormones. The most popular present day hormonal therapy of oligospermia is large doses of Testosterone, which produce the so-called rebound phenomena. The usual dosage of this drug is 50 to 75 mg. of Testosterone twice a week for eighteen weeks. Charles W. Charny, of Philadelphia, found, as a rule, that spermatozoa disappeared from the semen after twelve weeks of Testosterone administration. The injections were continued for an additional six weeks. In most patients, the spermatozoa first reappeared in the semen three months after the Testosterone withdrawal, and once present, the increase continued through the fourth and fifth months, when a peak was reached. In many of the unsuccessful cases, the semen count rose to the pre-treatment level three months after withdrawal, and remained unaltered thereafter. Testoterone, however, is not the only method of producing rebound phenomena. Estrogenic substances such as Ethinyl Estradiol, 0.5 mg. twice a day for one month, have been used with apparent good results. The average time for the maximum count, however, was ten months, but there was improvement both in the count and the motility in the few series reported. The rebound phenomena has also been produced by Pregnenolone. Other hormones, such as Cortisone, have been used, but these have proved to be very disappointing.

Surgery also has something to offer the infertile male. A patient with azospermia, whose testicles are normal in size and consistency, should have a testicular biopsy, since this examination may show normal spermatogenesis, and thus suggest obstruction in the conducting system. Such patients should be considered for an epididymovasostomy. Successful cases following this procedure have been recorded from 5 per cent to 20 per cent.

The idiopathic varicocele has been shown to be of importance to the subfertile male, since it has been shown that the presence of a varicocele reduces spermatogenesis. It is thought that this is produced by the increased local heat of the scrotum and its contents associated with the varicocele, and also by the long-standing congestion associated with this lesion.

Prophylactic surgery plays a part in the treatment of the infertile male. The early treatment of cryptorchism, that is, at age 6 to 9 years, is necessary if any hope is to be maintained of salvaging the seminiferous tubules. Gross recently has reported a 79 per cent fertility rate of cryptorchids who were treated prior to the age of 9 years. He pointed out that, in his series, the fertility rate in patients treated by the Thorek type of orchidopexy proved to have a much lower fertility potential. In the past, many young males have been rendered sterile, or at least subfertile, by testicular ischemia produced by too snug a repair of an inguinal hernia.

Probably the last procedure to be used to secure pregnancy is artificial insemination. However, it is generally considered that this procedure should be reserved until all other possible avenues have been explored in both the male and the female.

Perhaps there is justification for a somewhat pessimistic outlook regarding therapy in the subfertile male, but it is this attitude towards this unfortunate patient which has led to the neglect of many of the known factors that will increase his fertility potential. However, the truth remains that infertility in the male is a fertile field for research.

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The Significance of Hematuria

Clarence L. Gosse, M.D.

HEMATURIA means not only blood in the urine; it means cancer of the urinary tract. Let us not hold any other thoughts in our own minds unless we have proven otherwise. **Proof** involves a complete urinary tract investigation, including cystoscopy and intravenous pyelogram, and nothing short of this will satisfy good clinical practice or a court of law.

The sole reason for such a discussion on the significance of hematuria at this time is because of our persisting difficulties with this symptom. The incidence of **early** diagnosis of cancer of the urinary tract has improved in the last ten years. It still, however, is far from satisfactory. One of the main reasons for the delay in early diagnosis stems from errors in judgment. I know of no way in which to judge, with any hope of accuracy, a case of hematuria. All such cases require cystoscopy, and, with few exceptions, should also have an intravenous pyelogram. Intravenous pyelography of a very high quality is available in practically all hospitals in Nova Scotia. Cystoscopy for the purpose of gross examination of the bladder should also be available in every hospital. Where this is not so, I think the physicians of each hospital area should agree to have one of their number acquaint himself with the use of the cystoscope for the purpose of bladder visualization. Then, all physicians in that area should cooperate in asking him to cystoscope every case where there is blood in the urine. If this is not possible, then the individual physician should learn how to use the cystoscope for that limited procedure, or ensure that the patient gets examined by someone who is willing and capable of doing it.

The intravenous pyelogram is a relatively simple procedure which can be done on anybody. I know of no contraindication to an intravenous pyelogram except sensitization to iodine and an elevated N.P.N. The latter is only a contraindication because of the inability of the kidney to secrete the iodine compound, in which case the test gives negative and misleading information. Intravenous pyelography should be one of the first steps in any urological investigation.

Cystoscopy for diagnostic visualization of the bladder should be as readily practised as sigmoidoscopy for examination of the rectum and sigmoid. At Dalhousie University we are now teaching internes who spend sufficient time on our service the technique of visualization by cystoscopy in order to rule in or out tumors of the bladder. This centre would also be most happy to take any practising physician a week or so, and instruct him along the same lines.

You may say that surely it is not necessary to X-ray and cystoscope cases where there would appear to be some other cause than a tumor. I refer to such cases as hematuria in children, hematuria associated with post-marital cystitis, or where there is a history of trauma. To that I can only ask who knows whether or not the apparent cause is the real cause, or whether it is just a smoke screen obscuring the true state of affairs. Many of us have seen lesions of the breast, bones, and testicles where trauma was the presenting complaint, but where thorough investigation revealed a pre-existing malignant lesion.

Nearly 50% of cases of hematuria admitted to general hospitals for urological examination have tumors of the urinary tract. Of these patients with hematuria, over 60% have had more than one attack, and many have already been treated as a clinical entity.

Let us look at the forms in which hematuria may manifest itself:—

There is gross and microscopic. There is initial, terminal, and mixed. There is painless and painful, persistent and intermittent. There is hematuria in both males and females. There is hematuria in children, hematuria in adults, and hematuria in the aged. Statistically and academically, these forms have significance, but there is no clinical method of telling the benign from the malignant, the significant from insignificant, short of a cystoscopic survey.

The intermittent nature of many cases of hematuria is frequently the basis of investigative lethargy. A few days after the hematuria is first seen, the bleeding often subsides spontaneously. It usually recurs later, but may subside again. For some reason, this intermittency indicates a lack of seriousness to many patients, and valuable time is lost before going to their physicians.

Some patients go to their doctors with a history of passing blood in the urine, but at the time the urine may be quite clear. In view of the negative urine, physicians may then minimize the gravity of the symptom, and unfortunately, the patient is then spared further examination.

In the Victoria General Hospital, a high percentage of patients admitted for hematuria had their first bout between one and two years previously. In many instances, they thought that because the bleeding repeatedly ceased, there was nothing seriously wrong, and did not consult their doctor. In other instances, the patients consulted their doctors, and the symptom was treated as a clinical entity and examination deferred.

In some cases the source of bleeding can be suspected by the age or sex, or the form which the hematuria takes. Hematuria in the young adult is most likely due to inflammation. Painful hematuria is also more likely to be inflammation or stone. Painless hematuria, if terminal, i.e., at the end of micturition, is likely to be a benign bladder tumor. If the hematuria is initial, i.e., at the beginning of the stream, it must originate from the bladder neck outward, and most likely from the prostate or urethra. If the hematuria is mixed, i.e., with no change throughout the whole stream, it likely originates from the kidney or a malignant bladder tumor. If hematuria is not associated with frequency or dysuria, a diagnosis of tumor must first be considered.

Hematuria is the presenting symptom in over 85% of bladder tumors. Yet despite this most definite and obvious symptom, only 10% get diagnosed within one month after the onset of the bleeding, and only 50-55% within one year.

One does not suggest that hematuria is associated **only** with malignant disease. In the over-all picture, inflammatory lesions probably hold top place, but that is followed in order by tumor, calculi, the nephritides, and a host of other rarer conditions.

The table below indicates most of the lesions responsible for the finding of hematuria.

Incidentally, one must be sure that the red discoloration of the urine is really blood. Instances have been seen where the "so-called" hematuria was due to some food discoloration, biliary tract disease, or some rare entity causing a red or brownish tinge to the urine. Microscopic examination is absolutely necessary to be certain.

The general practitioner is usually the first consulted for hematuria. On him rests the burden of early diagnosis. If there is any procrastination on his part, he must assume the mental load of consigning a percentage of his patients

to a death from malignant disease. I think it important for us to remember as regards significance of hematuria—

"Hematuria is usually the first warning of urinary tract malignancy and is an absolute indication for cystoscopy."

CAUSES OF HEMATURIA*

	Relation to Micturition	Situation	Cause
Painful	Initial	Urethra	Acute urethritis. Caruncle. Rupture.
	Terminal	Bladder	Cystitis. Ulceration. Calculus. Growth (usually malignant). Carcinoma of prostate. Foreign bodies.
	Mixed	Ureter	Calculus. Blood clot. Ureteritis. Ureteric stricture.
		Kidney	Calculus. Hydro- or pyonephrosis. } some Tuberculosis. } cases. Laceration or gunshot wound. Pyelonephritis. Movable kidney.
Painful	Initial	Urethra	Growth (very rare).
	Terminal	Bladder	Growth (usually innocent). Congested prostate. Enlarged prostate. Stone (occasionally). Varicose vein. Schistosomiasis, especially in patients returning from overseas.
	Mixed	Ureter	Growth } very Ureterocele } rare.
		Kidney	Growth (usually malignant). Calculus. Hydro- or pyonephrosis. } some Tuberculosis. } cases. Congenital cystic kidney. Essential hematuria or hematuria from minute foci.
		Pre-renal	Purpura, scurvy. Arteriosclerosis. Mistral stenosis with fibrillation. Subacute bacterial endocarditis. Drugs or poisons, e.g., turpentine. High-protein diet. Embolus. Excessive exercise.

*C. C. Higgins, M.D., Modern Medicine June 1955.

Alcohol Pain as A Symptom of Disease

John O. Godden, M.D.*

A study of the incidence of alcohol pain in patients with Hodgkin's Disease, other lymphomas, other carcinomas, and in patients without malignant disease, is being carried out as a clinical research project at Dalhousie University. A medical student, Mr. W. L. M. King, is surveying all cases in the files of the Nova Scotia Tumor Clinic and selected ward patients in the Out-Patient Department and in the public wards of the Victoria General Hospital. However we are interested in knowing if any physicians in Nova Scotia have noted this symptom in patients in their practice. For this reason a short paper describing the characteristics of alcohol pain, as we know it, is presented at this time.

In certain patients with Hodgkin's Disease ingestion of small amounts of alcohol may cause severe pain. In 1950 Hoster[1] referred briefly to this phenomenon stating that the pain occurred in some of his patients and if some pain was present already it was increased at the site of the Hodgkin's deposits. Zanes[2] stated that "the amount of alcohol in a sherry wine gelatin dessert is sufficient to produce severe pain when disease activity is present." I reviewed this subject in 1954 and found 15 instances of this phenomenon in European literature and was able to add 4 more cases to this number.[3] Bichel and Bastrup-Madsen[4] found the symptom which they named "alcohol pain" in 9 of 62 patients with Hodgkin's disease. This peculiar response to alcohol ingestion was thought to be confined to patients with Hodgkin's disease but James[5] and co-workers very recently have reported alcohol-induced pain due to carcinoma in 2 patients. Although the primary site of the carcinoma was in slight doubt in both cases, it was almost certainly in the thymus in one and in the pancreas in the other.

In the 15 cases observed in Holland, Denmark, and England the pain caused by intake of alcohol had not been present previously. The pain was described as intense, acute, burning or "terrible" with onset during consumption of the beverage or varying from immediately after to 15 minutes later. The duration of the pain was from 10 minutes to an hour. It usually subsided spontaneously but 2 patients required opiates. The amount of alcohol required to produce pain in these cases was small, as little as one or two mouthfuls of beer, brandy, or sherry being sufficient. This bizarre symptom, when present, is extremely characteristic.

The pain occurred in the areas of granulomatous deposits found in Hodgkin's disease. Several patients felt the pain in new deposits that had recently appeared. In 8, the pain was localized in the chest extending in 3 of these into the arms. In 2 cases recurrence of the disease was heralded by the onset of alcohol pain at the site of the bone lesions in the ischium and sacrum respectively.[6] The pain was felt before there was any X-ray sign of recurrence in both patients. In 7 of the 15 patients the phenomenon of alcohol pain disappeared after treatment of the underlying disease.

The first individual I saw with this symptom was a 34 year old woman who came for examination on September 2nd, 1953, complaining of chest pain of 10 months duration. This pain had been characterized by a precordial

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tightness associated with a dull aching or twitching pain which extended upward to the left side of the neck into the left shoulder and down the medial aspect of the left arm to the fourth finger. It was first noticed by the patient in November 1952 when, after bowling, she drank a glass of beer. After 5 minutes or so a dragging pain began in the chest and shoulder. For the next 8 months this pain appeared after each use of an alcoholic beverage but after March of 1953 the patient began to have a little pain without alcohol. If she took a second highball the pain would occasionally abate, but more often it would increase in severity. She would sometimes take a cocktail "to check up on the pain" and was always able to reproduce it in the chest, shoulder and arm. About six weeks before admission, she stopped taking alcoholic beverages of any kind because the pain was so severe. The pain lasted from 1 to 3 hours and in this severity was brought on only by alcohol until just before admission when she noted the pain after exertion of raising a window that was stuck. The patient came for examination because mediastinal mass was discovered by her family physician.

A roentgenogram of the chest revealed an enlarged left hilar shadow which appeared to be nodular on first examination Thoracotomy disclosed an infiltrating mass that apparently arose from the thymus and involved the pericardium and pleura anteriorly and included the left phrenic nerve. The mass was removed by resecting the left lower lobe of the thymus and the involved pericardium and pleura. All of the large left hilar lymph nodes were dissected away from the bronchi and great vessels. A frozen section taken at the time of surgery showed the presence of Hodgkin's disease. After an uneventful recovery the patient received radiation therapy to the mediastinum. An alcoholic drink taken before leaving the hospital produced no pain and there was no alcohol pain during the 10 months following operation.

The cause of alcohol pain in patients with Hodgkin's disease is not known although several hypotheses have been proposed. It does not occur in a large percentage of patients but seems to be more than a coincidence in those who have the intolerance. Other workers suggest that alcohol pain may be of value in detecting a recurrence of the disease in patients being treated, and as a means of evaluating the results of treatment. It is recommended that patients suspected of having Hodgkin's disease be questioned about symptoms relating to the ingestion of alcohol and that anyone reporting pain on drinking alcoholic beverages be carefully examined for signs of systemic disease.

The current survey is being conducted by means of two questionnaires. The first questionnaire consists of three questions. The patient is asked if he has ever taken alcohol by mouth, whether or not he has ever had an unpleasant sensation after taking a small amount of alcohol, and finally if he has ever stopped taking alcohol for any reason. The second questionnaire, which is much longer, investigates the occurrence of this symptom in relation to the patient's medical history and utilizes whatever records are available. If any physician reading this report has a patient in whom he believes he has detected alcohol pain, we would be glad to send him copies of our questionnaire. It can be completed under his supervision or we will send the questionnaire directly to the patient with his permission. This can be arranged by communicating with Mr. W. L. M. King, c/o Nova Scotia Tumor Clinic, Halifax, Nova Scotia.

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Society Meeting

The annual meeting of the Western Nova Scotia Medical Society was held at Lakeside Inn, Wednesday, July 17th, with over fifty Doctors and their wives attending.

Following a social hour in the Boat House, we enjoyed one of Lakeside Inn's famous Smorgasbord Dinners in the Tuna Room. Later the Doctors adjourned to the Boat House for a business meeting, while their wives played Bridge in the Mezzanine.

The meeting was under the Chairmanship of Dr. D. S. Robb of Shelburne, serving in the absence of Dr. B. J. d'Eon. He called on Dr. Lee Steeves of Halifax to introduce the special speaker, Dr. Ian MacKenzie, newly appointed Professor of Surgery at Dalhousie University, who gave an illustrated lecture on "Vascular Insufficiency in the Lower Limbs and It's Treatment by Autogenous Graphs." Dr. J. E. LeBlanc led the discussion and thanked the speaker. Dr. Robb then called for the report of the Nominating Committee, chaired by Dr. Sutherland and the slate of officers was declared elected.

President	Dr. D. S. Robb, Shelburne
Vice-Presidents:	Dr. P. E. Belliveau, Meteghan
	Dr. J. Balmanno, Yarmouth
	Dr. J. Jeffry, Shelburne
Sect.-Treasurer:	Dr. D. F. Macdonald, Yarmouth

Member of the Nominating Committee of N.S.M.S.—Dr. P. E. Belliveau
alternate Dr. D.F. Macdonald

Nomination to N.S.M.S. Executive: Dr. D. R. Campbell
alternate Dr. D. F. Macdonald

Representative to Maritime Medical Care: Dr. D. F. Macdonald

Dr. Lee Steeves addressed the Society in his new position as Director of the Post Graduate Committee at Dalhousie University and offered us a series of lectures, once weekly for six weeks if we wished to organize a course in the local Hospital, which offer was accepted. Miss Joan Hudson, representing the Canadian Arthritis and Rheumatism Society, also spoke briefly and asked our support of the Society in organizing Mobile Physiotherapy and Home Service in this area.

Dr. G. B. Shaw of Maritime Medical Care and Dr. C. J. W. Beckwith, Executive Secretary of the N.S.M.S., also spoke briefly and the meeting adjourned at a late hour.

D. F. MACDONALD, M.D., C.M.
Secretary, Western Nova Scotia Medical Society.

Experimental Research into Problems of Ageing

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wishing to encourage well-conceived research relevant to basic problems of ageing, invite candidates to submit papers descriptive of work in the field for

AWARDS FOR 1958.

Copies of the Regulations and Form of Application must be obtained from the undersigned before an entry is submitted, but in general candidates should note:—

(*a*) Not less than five awards, of an average value of £300 each, are available for 1958. The announcement of awards will be made in July 1958.

(*b*) Entries must be received by the undersigned not later than 1st January, 1958.

(*c*) Entries will be judged by an international panel of distinguished scientists including: Prof. C. H. Best (Toronto), Prof. E. Braun-Menendez (Buenos Aires), Prof. E. J. Conway (Dublin), Prof. G. W. Corner (New York), Prof. A. Haddow (London), Prof. V. R. Khanolkar (Bombay), Prof. R. Nicolaysen (Oslo), Dr. A. S. Parkes (London), Prof. F. Verzár (Basle), and Prof. F. G. Young (Cambridge). They will advise the Executive Council of the Foundation on their findings and will also have power to recommend variation in the size and number of the awards according to the standard of entries. The decisions of the Executive Council will be final.

(*d*) In making the awards preference will be given to younger workers.

(*e*) The papers may be in the candidate's own language. Papers should not be more than 7,000 words in length and in all cases a summary in English not exceeding in words 3% of the length of the paper must be attached. If possible, 10 copies of reprints in English should be provided.

(*f*) Where there is one or more co-author, the name of the leading author should be indicated; it is to him that the award will normally be made, and it will be left to his discretion to share this award appropriately with his co-authors.

G. E. W. WOLSTENHOLME

Director
and Secretary to the Executive Council.

Personal Interest Notes

Dr. R. L. Aiken of Halifax, a specialist in internal medicine and diseases of the chest, was elected a fellow of the American College of Chest Physicians at the annual convention held in New York, July 17, 1957.

Dr. T. B. Acker, a Halifax orthopedic surgeon, has been elected vice-president of the 1958 meeting of the combined British, American and Canadian Orthopedic Associations, to meet next year in Washington, D.C. Dr. Acker was attending the Canadian Orthopedic Association at Murray Bay, P.Q., at the time of his election.

Dr. Kenneth A. MacKenzie, retired specialist in internal medicine of Halifax, was a guest of honour at a testimonial dinner held on June 22 in the Nova Scotian Hotel. The dinner was held by the Royal Sussex Masonic Lodge, and was in honour of his fifty years membership in the Masonic Order. Following the dinner, Dr. MacKenzie was presented with a fifty-year jewel of the Order, and a toast was proposed by Dr. A. E. Doull.

Obituary

Dr. Shirley Ebenezer Bishop, age forty-nine passed away at Blanchard Fraser Memorial Hospital on Sunday, July 7. He had undergone a major operation fifteen months ago and since that time had been in poor health. Born at Freeport, Digby County, N. S., he was a son of Mrs. Lena Woodroffe Bishop and the late Dr. Bradford S. Bishop. Dr. Bishop was a Bachelor of Science, Dalhousie University, and graduated in medicine from Queens University in 1939. He engaged in general practice at Kentville from 1939 until 1943 when he joined the R.C.A.F. Following the war he took post-graduate studies at the Polyclinic Hospital, New York, and then resumed his general practice in Kentville and was one of the town's most popular and finest citizens. He was Medical Officer for the West Nova Scotia Regiment Reserve, Aldershot Camp, and for the Kentville Air Cadets, 405 Lions Squadron. He had been prominent in the Valley Medical Association, Nova Scotia Medical Association, and was a member of the Kentville Masonic Lodge.

He is survived by his wife, the former Maude R. Tully, R.N., one son Robert, and his mother, Mrs. B. S. Bishop, and one sister, Helena, Mrs. Ewan Clark, of Halifax.

Dr. William Thomas Morris MacKinnon of Amherst, passed away at the Highland View Hospital on Sunday, July 2, after a long illness. He was born at Baddeck, Cape Breton, and was a graduate from Dalhousie University

in 1898 and from the University of Toronto in 1903, with his medical degree. In 1953, after practising for fifty years, he received his Honorary Degree from the University of Toronto. He served overseas with the R.C.A.M.C. during World War I, and received the C.M.G. from King George V at an investiture in London. During the Second World War he served as medical examiner in Ottawa, and was awarded the O.B.E. He returned to Amherst in 1949, and practised until the time of his death in the specialty of eyes, ears and throat. He was an active member of the Acadian Lodge A.F. & A.M. and was in his fifth term as a member of the Town Council.

He is survived by one daughter, Elizabeth, Mrs. Gordon Ferguson, Dartmouth.

Dr. Robert Arthur Haliburton MacKeen, D.A.B., died in Camp Hill Hospital on July 12 at the age of fifty-seven. Cape Breton born, most of his life had been spent in the Maritimes. He graduated from McGill University in 1924 and for a time was on the staff of the Halifax Pathological Institute. Later he was appointed Provincial Pathologist of New Brunswick, and was the Director of Laboratory Services, N.B. Health Department.

During the second World War he served overseas with the rank of Lieutenant-Colonel in the R.C.A.M.C. He held the appointment of Assistant Professor of Pathology at Dalhousie University. To mourn his loss he leaves his widow, Catherine Robert Wilson, a son and a daughter.