1972

Year Book of Urology

EDITED BY STAYHACK

THE YEAR BOOK
of
UROLOGY
1972

EDITED BY
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INTRODUCTION

Two aspects of a surgeon's life and his work stood out in my mind as this year's YEAR BOOK was put into final form. One had to do with prolonged uncertainty regarding undesirable sequelae after surgical intervention and the other, with the role of surgical procedures in the life of the surgeon and his patient. A type of tunnel vision that is not unique to surgeons and a requirement for the passage of time that is not unique to surgery are partially responsible for a failure to recognize a less than optimum result. The report by Kom *et al.* (this YEAR BOOK, chapter on the Genitalia) indicates an appreciable incidence and a complex mechanism for sexual dysfunction after retroperitoneal node dissection for testicular tumor. Even though a more selective dissection may avoid this complication, these observations emphasize the hazard in concluding that a surgical procedure does not remove tissue that will significantly affect the quality of life. My continued prejudice for use of node dissection in patients with testicular tumors other than pure seminoma now requires recognition that the assumption that retroperitoneal node dissection is associated with minimal mortality and almost no morbidity is not entirely valid. Similarly the report by Oschner *et al.* (this YEAR BOOK, chapter on the Prostate) confirms not unexpectedly that any type of revision of the bladder neck may interfere with sexual function despite the recognition that Gute's observations (Gute, D. G., *et al.*, J. Urol. 99:744, 1968) suggest Y-V bladder neck plasty would do so very infrequently.

Reviewing the article by Blakemore (Blakemore, W. S., *et al.*, Surgery 43:102, 1958), regarding the effect of adrenalectomy in patients with hypertension, in order to comment on the contribution by Sir George Pickering (this YEAR BOOK, chapter on the Kidney) brought to mind the accusation that surgeons carry out numbers of unnecessary operations. My qualitative if not quantitative acceptance of this indictment has been based on the impression that some patients seen by me have had procedures carried out that seemed ill advised in my experience. However, experiences differ and so do patients, so an absolute judgment, of necessity, seems difficult to achieve. Be that as it may, ample evidence exists to refute

the suggestion that blind adherence to operative procedures is characteristic of surgeons. Surgical intervention in the hypertensive patient has been altered appreciably in the past 15 years. Nephrectomy and arterial reconstruction enjoyed an exuberant acceptance but now take second place to medical management even in most patients with probable renovascular hypertension. Adrenalectomy has essentially been abandoned because other methods of controlling hypertension have had such significant success. Whether or not the proper place of surgical procedures in management of the hypertensive patient has been accurately defined at present, the utilization of a surgical approach has been significantly modified or essentially abandoned within a short period with concurrence of surgeons. Some unnecessary operations undoubtedly are done today and some necessary ones are not. Motivational factors in both behavioral patterns may be difficult to assess, but the demonstrated continued modification of practices on the basis of experience encourages me to question the more skeptical attitudes expressed so freely by some.

The publication date of the YEAR BOOK OF UROLOGY was changed this year in the hope of increasing its usefulness to those preparing for Board examination. Again, I am most grateful to Dr. William H. Boyce of the Bowman Gray School of Medicine, Dr. Thomas A. Stamey of Stanford University and Dr. Lowell R. King of our institution for their generous contributions of time and effort in preparing comments for selected articles in this YEAR BOOK. The effort of Dr. I. Bush and his associates in keeping the Selected Reference section current is also greatly appreciated.

JOHN T. GRAYHACK

GENERAL CONSIDERATIONS

Examination of Urine

Epithelial Cells in Urine Sediments. Most textbooks of clinical pathology describe the presence of various forms of epithelial cell in urine sediments, some of them derived from the upper urinary tract or tubular epithelium of the kidneys. Modern books on cytology do not describe tubular renal epithelial cells in urine. William H. Kern[1] (Hosp. of the Good Samaritan, Los Angeles) examined this discrepancy in the interpretation of epithelial cells present in urine specimens.

Slides of 50 consecutive urine specimens, 6 of which were positive and 2 suspicious for malignancy, were reviewed and unusually small and vacuolated as well as the largest cells were measured. The specimens from patients with malignancy contained transitional epithelial cells. Specimens had been processed by adding 3 parts of 30% propanol to 1 part urine and by filtration through a Millipore apparatus.

Transitional epithelial cells and leukocytes were found in all specimens and squamous epithelial cells were seen in many. Transitional epithelial cells were 9.3-40 μ in diameter. Most contained fairly uniform round nuclei and they were often clustered. The cytoplasm was often vacuolated even in well-preserved cells. Small columnar cells were occasionally observed, especially in ureteral catheterization specimens. Smaller epithelial cells sometimes had eccentric nuclei and a small amount of cytoplasm (Fig. 1).

Renal parenchymal cells could not be distinguished from the much more numerous transitional epithelial cells. Well-preserved histiocytes were readily recognized by their eccentric, often lobed or bean-shaped nuclei and foamy or vacuolated cytoplasm. Some showed phagocytic activity. Small epithelial cells with shrunken irregular nuclei are difficult to differentiate from histiocytes. Large cells were seen in cases of poorly differentiated transitional cell carcinoma of the bladder but enlarged benign cells were sometimes seen in severe inflammatory reactions.

Re-evaluation of material from two quantitative studies of

(1) Am. J. Clin. Path. 56:67-72, July, 1971.

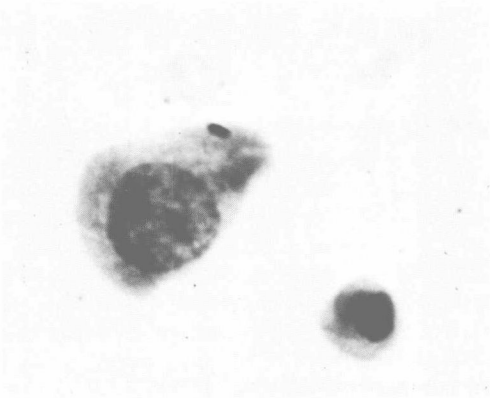

Fig. 1.—Size variation of normal transitional epithelial cells: reduced from ×1000. (Courtesy of Kern, W. H.: Am. J. Clin. Path. 56:67-72, July, 1971.)

epithelial cells from 350 specimens failed to show a distinct cell type characteristic of tubular epithelium. The mean diameter of 4,340 benign transitional epithelial cells was 19.6 μ, with no second peak incidence of cell size consistent with a different cell population.

Discrepancies in interpretation of cells in urine specimens are due to differences in technic. Many cells described in clinical pathology textbooks as being of apparent renal origin are probably transitional. Cells identifiable as of renal tubular origin cannot be recognized in routine sediment preparations. They should be accepted as renal only when they are clearly part of casts. The principal difficulty in classifying small cell types is in their differentiation from histiocytes.

Brown Urine as a Clue to Phenacetin Intoxication. A. L. Miller, L. R. Worsley and P. K. Chu[2] (Middlesex Hosp. Med. School, London) encountered a number of patients with syndromes believed to be wholly or partly attributable to ingestion of phenacetin who had abnormally dark urine. Five such cases are reported. Two patients were poisoned, acutely, whereas in 3 the diagnosis was not suspected until the unusual urine color was noted.

Man, 42, previously seen for thrombocytopenia of unknown cause,

(2) Lancet 2:1102-1104, Nov.28, 1970

was admitted semicomatose and disoriented, with profound methemoglobinemia. The urine was dark brown and reduced ammoniacal silver nitrate at room temperature. Severe hemolytic anemia and jaundice developed, with evidence of hepatocellular damage. The patient recovered slowly. No history of drug ingestion was obtained, but phenacetin metabolites subsequently were detected in the urine.

Four of the 5 patients had dark urine of varying shades of brown to black, not attributable to blood pigments, although 3 patients had hematuria at some time. The urine reduced ammoniacal silver nitrate solution at room temperature in all cases. A rapid chromatographic method to detect phenacetin metabolites was developed. It was confirmed by examining the urine of rats poisoned with phenacetin. An orange-brown spot was seen just behind the solvent front on the chromatogram, a second well-defined brown spot immediately behind it corresponded to control p-aminophenol. The latter blackened on treatment with silver nitrate and sodium hydroxide in ethanol. Urinary N-acetyl-p-aminophenol was also determined quantitatively by extraction after acid hydrolysis of the urine.

The nature of the brown pigment seen in the urine of these patients is uncertain. The colored derivative may have a complex and possibly inconstant structure. The yellow-orange moving just behind the solvent front appeared to be specific for phenacetin ingestion. It may be a derivative of phenetidine. It was not found in a patient on paracetamol. There was no evidence that the abnormal urine color was due to blood pigments or any other secondary effect of phenacetin.

Characterization of Proteins in Urinary Casts: Fluorescent-Antibody Identification of Tamm-Horsfall Mucoprotein in Matrix and Serum Proteins in Granules. The genesis of granular casts has been controversial. Currently it is believed that they represent a phase in the breakdown of cellular casts. Gerald J. Rutecki, Carl Goldsmith and George E. Schreiner[3] (Washington, D.C.) attempted to determine whether serum proteins can be identified in cast granules, the hypothesis being that some granules might be explained by the precipitation of serum proteins into a matrix of Tamm-Horsfall mucoprotein. Urine was taken from 8 pa-

(3) New England J. Med. 284:1049-1052, May 13, 1971.

tients, 6 with nephrotic syndrome of varying etiology, 1 with hypertension and possibly nephrosclerosis and 1 without apparent renal disease, but with acute pancreatitis. Sediments were obtained from aliquots of 24-hour urine specimens and washed until negative for albumin. An indirect fluorescent-antibody technic was used for all proteins studied. Large numbers of hyaline and granular casts were available from each patient.

Pure hyaline casts, free of granules, stained homogeneously and intensely only for Tamm-Horsfall urinary mucoprotein. When present, granules stained for serum proteins. Casts with varying numbers of granules all stained for serum proteins and Tamm-Horsfall mucoprotein, the matrix staining for mucoprotein and the granules for serum proteins. All the human serum proteins sought except C-reactive protein and fibrinogen could be identified in cast granules. Cells present in casts did not stain for either mucoprotein or serum proteins. The casts and granules from sediments of all patients stained identically.

The belief that cast granules, coarse and fine, are derived from the degeneration of epithelial cells deposited within casts deserves re-evaluation. Some cast granules may represent aggregated serum proteins. Tamm-Horsfall urinary mucoprotein may constitute a part or all of cast matrix. The granular staining demonstrates that most if not all serum proteins can be found in at least some locations in the nephron distal to the glomerular basement membrane. The inability to detect fibrinogen in 2 cases of membranous glomerulonephritis is not understood.

Infections, Including Gonorrhea

Antibacterial Effect of Normal and Infected Urinary Bladder. Although the normal urinary bladder is known to eliminate bacteria that are introduced and the bladders of animals with chronic lower urinary tract infection are known to produce large amounts of immunoglobulins, the relationships of these observations has not been explored. W. Lee Hand, James W. Smith and Jay P. Sanford[4] (Univ. of Texas

(4) J. Lab. & Clin. Med. 77:605-615, April, 1971.

Southwestern Med. School) compared the antibacterial activity of the bladder in normal and infected animals and correlated it with antibody synthesis by the bladder.

Chronic lower urinary tract infection was produced in male rabbits by partially obstructing the bladder and injecting Escherichia coli 075 intravesically. Bacteriuria was eradicated by antibiotic therapy after at least 30 days of infection. The antibacterial effect was examined by inoculating ^{32}P-labeled bacteria into bladders, normal bladders or bladders altered by infection. Activity was also assessed in excised bladder tissue. Protein, immunoglobulin and specific antibody synthesis was determined, the latter by adding somatic antigen to aliquots of DEAE fractions of the supernatant of minced bladder tissue incubated in modified Eagle's medium.

Bladders of normal animals produced little immunoglobulin and no anti-E. coli antibody, whereas chronically inflamed bladders of recently infected animals produced large amounts of IgG (26% of total protein synthesis) and specific anti-E. coli 075 antibody (13% of synthesized IgG). Bladders of normal and recently infected animals showed a similar antibacterial effect, comparable against each of the organisms tested (E. coli 075, E. coli 014, Proteus mirabilis C). Most organisms were killed within a few minutes of contact with the bladder; the effect was persistent and often progressive over a period of 4 hours.

The inherent antibacterial effect of the bladder is intact in infection. The effect is independent of local specific antibody production and is not improved by an increase in local immunoglobulin and specific antibody synthesis. The exact mechanism of bladder antibacterial activity is unknown. Probably an unidentified factor concentrated at the bladder mucosal surface is able to kill bacteria in contact with the bladder. ► [It is interesting that the presence of infection did not stimulate the antibacterial effect of normal bladder mucosa.—Thomas A. Stamey.]

Bacterial Excretion Rates in Diagnosis of Urinary Tract Infections. Currently significant bacteriuria is defined as a bacterial concentration of 100,000/ml. or higher in midstream urine specimens; colony counts below 10,000/ml. are considered insignificant. The influence of variations in urinary flow on the rate of bacteria excretion in the urine has

not, however, been defined. I. Lampert and G. M. Berlyne[5] (Beersheba, Israel) compared the excretion rate and the concentration of organisms in the urine in patients with and without significant urinary tract infection. Forty patients, including 15 with clinical urinary tract infection, were studied. The other 25 had minor ailments or miscellaneous nonrenal diseases. The total volume of urine passed was measured, and standard cultures were carried out on blood-agar medium. Plates were incubated at 37 C. for 24 hours.

Urine volumes ranged from 0.15 to 4.17 ml. per minute (mean, 0.64). All patients with urinary infections had bacterial excretion rates exceeding 10^4 colonies per minute. Six had less than 100,000 colonies per ml. urine, indicating bacteriuria of doubtful significance according to Kass's classification using bacterial concentrations. Infections were not seen with an excretion rate below 10^4 clones per minute.

Early morning urine specimens are not suitable for clone counting. A timed specimen taken in the forenoon is preferable since the forenoon sample is often the first passed since the initial morning micturition on rising.

► [The authors fail to recognize that the midstream urine of uninfected males is contaminated by urethral bacteria far less (several logs) than the midstream urine of uninfected females. Twenty of the 25 "controls" were males, thereby skewing their normal bacterial excretion rates in favor of their proposal. Had they compared excretion rates of uninfected women with those of infected women, and assuming they counted all contaminating bacteria, the result would have been many false positives from heavy perineal contamination of uninfected urine. They also seem unaware that Doctor Kass's data are based on the total voided specimen, not the midstream urine. Lastly, the critical reader will not accept as evidence of bacteriuria the simple statement that they (15 patients) "had clinical evidence of urinary tract infections." Either suprapubic needle aspiration of the bladder or a carefully collected catheterized urine would be necessary to *prove* the presence of bacteriuria. Despite the few disadvantages to quantitative urine cultures, simple bacterial counting of the midstream urine is most unlikely to be replaced by bacterial excretion rates.—Thomas A. Stamey.]

Experimental Production of Pyeloureteritis Cystica and Glandularis. Cystitis cystica, ureteritis cystica and pyelitis cystica and glandularis are seen not infrequently at autopsy, and occasionally may cause urologic symptoms. There has been no direct proof that gram-negative bacteria can cause metaplasia and cyst formation. Gary S. Hill[6] (Johns Hopkins Univ.) found that Escherichia coli infection can cause epi-

(5) Lancet 1:51-52, Jan. 9, 1971.
(6) Invest. Urol. 9:1-9, July, 1971.