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HEMATOGENOUS INFECTIONS OF THE KIDNEY *

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The surgical aspects of nephritis have led to extensive discussion, and but little agreement exists with regard to the types that are not clearly surgical. Wide differences of opinion are also manifest among those who have given the most time and attention to the study of nephritis in general. The accumulated literature on the subject is monumental; much of it, unfortunately, is confusing, and often even the fundamentals are the subject of controversy. Recently, however, articles have been published that help to clear up some of the moot questions.

Historically, the chief point of interest with reference to nephritis is the profound knowledge of the various phases of the disease exhibited by Richard Bright,¹ one of a long line of brilliant workers in Guy's Hospital, London. In 1827 and again in 1836 Bright, in epoch marking papers on edema and on albuminuria, pointed out that fundamentally there are two kinds of nephritis: Type 1, the acute or "wet" nephritis; and Type 2, the chronic or "dry" nephritis.

Type 1, which involves the kidney filter, was to be recognized pathologically in cases in which the large white kidney was found. Clinically, the type was characterized by edema, especially in the acute stage, and by urine containing albumin and casts, and at times blood corpuscles, the urinary findings varying in extent with the acuteness of the process and the amount of involvement of the kidneys. Both kidneys are always involved, and exhibit a wide pathologic architecture. The morphology varies as the toxic agents vary in toxicity and affect different structures of the kidney.

BRIGHT'S TYPE 1 NEPHRITIS

Since the publication of Bright's work, we have learned that Type 1, or the "wet" type of nephritis, is usually the result of toxins developed in the course of infectious diseases. Diphtheria antitoxin affords the best known example of the effect of bacterial toxins on the kidney; it may produce changes in the kidney

analogous to the nephritis which sometimes develops in cases of diphtheria. The frequency with which Bright's Type 1 and infective nephritis follow lesions of the skin and mucous membrane, especially in children, is worthy of note, as shown by the nephritides following scarlet fever, tonsillitis, etc. In the adult, exposure to cold, especially of the cutaneous surfaces of the body, plays a definite rôle. The value of the skin as an aid to elimination in cases of defective kidney function has long been well known. The effect produced by hot packs and similar therapeutic measures is to cause a flow of perspiration which may eliminate large amounts of chlorids and some urea.

It is true that acute nephritis may be produced by other toxic agents, such as cantharides; but to all intents and purposes an infection lies behind it. The edema is to a large extent due to a failure of elimination of chlorids. Chlorids, usually taken in the form of common salt, are a constant requirement of all animal life. The herbivorous and omnivorous animals obtain the necessary amount in the form of salt, while the carnivorous obtain a sufficient amount from the flesh they consume. The chlorids are largely excreted by the kidneys. In acute nephritis, chlorid is not fully eliminated, and edema results. The physical and chemical reactions involved in the production of edema by chlorid retention are still greatly disputed, however, and it is apparent that there are varying factors to be considered.

BRIGHT'S TYPE 2 NEPHRITIS

Bright's Type 2, called the dry type because edema is not present, involves the connective tissue and blood vessels, especially the arteries; the patient suffers from headache, nausea, vomiting, hypertension and other symptoms of uremia. The symptoms may be latent for long periods. Urea retention is one of the final consequences of this type. While urea will not of itself, when in excess in the blood, produce uremia, it is closely associated with if not one of the agents which produce the uremic manifestations. Both kidneys are involved, and pathologically in the typical case the kidneys are contracted and granular. The urine is of low specific gravity, pale, and large in amount. The urinary findings may consist only of an occasional cast and a trace of albumin, although at times there may be large amounts of albumin. This type of Bright's disease is closely associated with or is a part of a general vascular disease, and perhaps should not be described as a true primary nephritis. Changes in the heart are so characteristic of chronic Bright's disease that the appellation of cardiorenal disease is at times not inappropriate. The pathologic architecture varies with the situation of the chief con-

* Read before the Section on Urology at the Seventieth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1919.

1. Bright, Richard: Reports of Medical Cases with a View of Illustrating the Symptoms and Cure of Diseases by a Reference to Morbid Anatomy, London, Longmans, Green & Co., 1827-1831, 2 vols., xvi, 231 pp.; xl, 724 pp.; Cases and Observations Illustrative of Renal Disease Accompanied with Secretion of Albuminous Urine, Guy's Hosp. Rep. 1: 338-379, 1836; Tabular Review of the Morbid Appearances in One Hundred Cases Connected with Albuminous Urine, with Observations, Guy's Hosp. Rep. 1: 380-400, 1836.

nective tissue deposits and the changes in the renal vascular system. Bissell² and other pathologists call attention to the fact that many of the changes in the kidney called nephritis are regressive in character. Neither infections nor the products of infection, so far as is known, play an important part in the etiology of Bright's Type 2. The cause of the condition is as yet unknown, but it is probably due to disorders of metabolism.

Edema may develop in chronic Bright's disease (Type 2), but if it does it is almost invariably the result of cardiac failure and not of salt retention. Widal had divided nephritis functionally into two forms: the chloremic or salt retention type, corresponding to Bright's Type 1, and the azotemic, nitrogen or urea retention type, corresponding to Bright's Type 2. It has been asserted by various authorities that chronic Bright's Type 2 is the late result of Type 1. I am convinced, however, that Bright's Types 1 and 2 are entirely independent diseases.

A discussion of the differentiation is unnecessary here, but the confusion seems to arise from mixed types. In certain varieties of Bright's Type 1, chronicity may be a feature. This is especially true of a form of Type 1, caused by chronic focal infections. I have seen what were believed to be cases of Bright's Type 1, caused by chronic infections of the teeth, tonsils, gallbladder, or by duodenal ulcer, etc. These patients recovered from the nephritic disturbance after operative cure of the focus of infection. Subacute infections, with slowly sterilizing abscesses, will often cause symptoms of a nephritis which disappear as the cause is removed. Excessive connective tissue may develop in the kidneys in some cases of Type 1 so that the resemblance is close to the contracted kidneys of Type 2; but the vascular changes are different. Again, chronic Bright's disease, Type 2, acts as a cause for lowered resistance of the kidneys, and a secondary true nephritis of Type 1 may be added to the condition, especially as a terminal infection leading to the frequent postmortem finding of death from acute nephritis superimposed on chronic nephritis. This confusion is further increased in some cases by pathologic changes in the kidney, the late results of unrecognized hematogenous infection which might well be called Type 3.

FUNCTION OF THE KIDNEY

Rowntree³ calls attention to the fact that medicine of the last decade centers around the question of function, so far as the kidney is concerned. The capacity of the kidney to carry on its work, rather than its appearance, has come to be recognized as the important factor. This is well shown by the value clinically of the phenolsulphonephthalein test and the test of the chemistry of the blood. The percentage of urea in the blood or the phenolsulphonephthalein test will frequently give a clue to the state of the kidneys that cannot be obtained from the urinary findings.

That the kidney in health filters bacteria out of the blood, and by so doing receives no injury, is a fact too well known to require comment. It is the retention of the bacteria in the kidney which produces the

trouble. But we know much less about its ability to filter out toxic materials. In the greater number of cases of nephritis, no bacteria are found, and we must believe that it is some material, the result of infection, rather than the bacteria themselves that cause the trouble, although it is possible that an ultramicroscopic organism exists as the causative factor. The kidney normally filters out urea, which is the ash of protein derivatives, the amino-acids. The patient with a granular, contracted kidney (Bright's Type 2) must of necessity pass a large quantity of urine; the specific gravity is low because the kidney filter is damaged and fails to eliminate normal concentrations of urea and similar bodies. As these products are not threshold bodies, they are always to be found in both the blood and the urine.

On the contrary, sugar in the blood is a threshold body. When it rises beyond the normal, the excess flows off through the urine, and diabetes results. The threshold, however, is not an exact point, and many persons with crops of boils or carbuncles may, by testing the blood for sugar percentages, be shown to have an increased amount of blood sugar without having sugar in the urine, just as in others a low threshold permits the escape of sugar from slight temporary causes, so-called dietetic or alimentary glycosuria, which is without pathologic significance.

Embryologically, the kidney has a double origin: First, the ureter, the pelvis, the calices, and the straight collecting tubules all lined with pavement epithelium, which are derived from the wolffian duct and have no function beyond that of collecting urine as it is formed. The chief response of the part of the kidney thus derived, when diseased, consists in the development of infections, calculi, etc., and if the disease is a malignant one, it is a true carcinoma. Obstructions to the urinary outflow at any point are most potential of mischief through failure of drainage. The medullary part of the kidney is composed of the urinary collecting channels just described and of the venous channels which carry back the purified blood to the general circulation. Second, the filtering portion of the kidney, which arises from the mesothelium and forms in large part the cortical substance which is the arterial side of the kidney, bringing blood from the general circulation for purification. The common, solid tumors of the kidney that we have misnamed hypernephroma are, as pointed out by Wilson,⁴ true mesotheliomas; that is, malignant neoplasms arising in the kidney filter. I think we may agree with Cushny⁵ that the kidney does not secrete urine, but filters it. The kidney may be aptly compared to a separator. The arterial blood enters the kidney cortex, the urine is filtered out, and the venous blood passes back to the general circulation. Weld,⁶ by his experimental injections of mediums, demonstrated the physics of the process outlined. The partial failure of the two halves of the kidneys to unite, so that the urine, when filtered, is not freely admitted into the collecting tubules, produces the so-called congenital polycystic kidneys that are eventually associated with chronic nephritis.

4. Wilson, L. B.: *Hypernephromata*, Old Dominion J. M. & S. 10: 238-252, 1910.

5. Cushny, A. R.: *The Secretion of Urine*, New York, Longmans, Green & Co., 1917, 251 pp.

6. Weld, E. H.: *Renal Absorption with Particular Reference to Pyelographic Mediums*, Thesis submitted to the Graduate Faculty of the University of Minnesota in partial fulfillment of the requirements for the Degree of Master of Science in Surgery, June, 1919.

2. Bissell, W. W., and Le Count, E. R.: A Consideration of the Relative Frequency of the Various Forms of Coma with Special Reference to Uremia, J. A. M. A. 64: 1041-1045 (March 27) 1915.

3. Rowntree, L. G., and Geraghty, J. T.: An Experimental and Clinical Study of the Functional Activity of the Kidneys by Means of Phenolsulphonephthalein, J. Pharmacol. & Exper. Therap. 1: 579-660 (July) 1909-1910.

TYPE 3 NEPHRITIS

Nephritis, the result of living organisms, may be called Type 3. True nephritis is concerned with the filter portion of the kidney, and the failure to filter out all the bacteria and their retention is responsible for the occurrence of one form of the disease, which is of great surgical importance. As to just how often such infections may be ascending or lymphogenous rather than hematogenous in origin there is a diversity of opinion. My own opinion is that such infections, other than hematogenous, are extremely rare so far as the kidney filter is concerned, though it is possible they may be more frequent in that part of the kidney devoted to collecting urine.

Accepting the idea that there is a common form of true nephritis which differs from Bright's Types 1 and 2 and is caused by a bacterial infection, we quickly see that the effect on the kidney will depend on the nature of the bacteria, their number and on the condition of the kidney itself, on whether, for example, there is an anomaly present, such as hydronephrosis or calculi, which makes the kidney more vulnerable. The importance of this has been shown by Cabot and Crabtree,⁷ who demonstrated that the pus cocci affect the cortex of the kidney and often follow lines of least resistance toward the periphery; and that the colon bacteria affect the straight collecting tubules of the pyramids, extend from them to the pelvis, and there produce a pyelitis. Pyogenic infections may lead to cortical abscesses and other evidences visible to the eye; but with scanty urinary findings beyond a trace of albumin in the acute stage, and a few microscopic pus and blood cells, the urinary evidences are so slight as to be overlooked unless great care is exercised. In the subacute and chronic forms the kidney may be more or less destroyed, and the common forms of pyonephrosis will follow. The pyogenic cocci are short lived and often are not to be found in the pathologic changes their action initiates. On the contrary, colon bacteria, by the production of copious, purulent sediment in the urine, give abundant evidence of infection without abscess formation in the kidney. Acute nephritis (Bright's Type 1) is the result of toxic products, not of living organisms, as in diphtherial and scarlatinal nephritis, and the two kidneys are equally involved. When the nephritis is the result of living bacteria (Type 3) the kidneys may be involved unequally or unilaterally, the unilateral infection being in the kidney which is more vulnerable because of some physical defect. The pus-producing organisms affect the kidney much as do tubercle bacilli, and those who have studied tuberculosis of the kidney will recognize the resemblance.

Hematogenous nephritis is often caused by cocci found in the skin, especially staphylococci derived from boils, carbuncles, etc., and from focal infections generally. The staphylococcus is short lived and often affects only one kidney. Acute streptococcal infections are most malignant. Subacute and chronic streptococcal infections occur commonly as a result of septic endocarditis, and appear in the kidney as a terminal infection, embolic in character.

In the fulminating type of hematogenous pyogenic infection, unless nephrectomy is performed, death may often result within a few days. The acute condition is often confused with acute intraperitoneal infections;

on the right side, especially, is the differentiation from cholecystitis and appendicitis necessary. This may also be true of the less acute form. In the subacute and chronic forms, natural processes may localize and sterilize the foci of infection, and the patients may fully recover, or partial recovery may later be followed by chronic infection, and the kidney will be converted into a pyonephrosis. The fact should be recalled that septic infarcts rupturing through the capsule of the kidney are the most common cause of perinephritic abscess.

Bright's disease, both Type 1 and Type 2, is of surgical interest only because of complications. But all the nephritides caused by living bacteria (Type 3) are of great surgical interest and have, until recently, been confused with Bright's disease. Hematogenous kidney infection is the one bright picture in a group of maladies that have been discouraging in the extreme. The surgeon of today is as greatly interested in nephritis as is the internist.

In reviewing the large mass of literature on nephritis, rather superficially, it is true, I have been particularly impressed with the clear and logical presentation made by Brewer⁸ in a series of published papers beginning in 1911, in which he demonstrates hematogenous pyogenic infections of the cortex of the kidney clinically, experimentally and pathologically. In this connection, I wish to comment on the value of the contributions of the surgeon to such problems. Nephritis has been studied largely from a clinical and a necropsy standpoint. Clinical observations in these cases are notoriously unreliable, while the necropsy shows the terminal conditions that cause death. This exposition of terminal change, determined at necropsy, is exceedingly difficult to interpret in the living, and the final catastrophe leading to the death of the patient produces pathologic changes obscuring those that existed during life. In no other way can I explain the various theories and controversies which have marked the history of the study of nephritis. The clinician attempts, by study of the urine and the symptoms of the patient, to explain the terminal conditions found after death, which may not have existed at the time the clinical examinations were made. The surgeon, not by reason of greater acumen but by opportunity, now furnishes the missing link in the investigation which carries the truth, and makes possible an exact study of the kidney before the terminal infections, which are encountered at necropsy, obscure the picture.

Payne and MacNider,⁹ and Buerger¹⁰ have shown the hematogenous origin of certain of the so-called chronic, essential hematurias, demonstrating that infection in and about the straight tubules, resulting in the development of scar tissue, which interferes with the venous circulation, causes congestion and varicosity of papillae and leads to rupture and renal hemorrhage. This gives a pathologic explanation for several cases in which we explored to find the cause of renal hemorrhage, and in which one or more papillae were found to be the seat of varicosities.

8. Brewer, G. E.: The Present State of Our Knowledge of Acute Renal Infections, with a Report of Some Animal Experiments, *J. A. M. A.* **57**: 179-187 (July 15) 1911; A Rare Type of Unilateral Hematogenous Infection of the Kidney, *Yale M. J.* **17**: 237-252 (Jan.) 1910-1911; Observations on Acute Haemic Infections of the Kidney, *Am. J. Urol.* **9**: 549-570, 1913; Hematogenous Infections of the Kidneys, a Summary of Our Present Knowledge, *New York M. J.* **101**: 556-560 (March 20) 1915.

9. Payne, R. L., and MacNider, W. B.: The Surgical Problem of Unilateral Symptomatic Hematuria, *J. A. M. A.* **67**: 918-923 (Sept. 23) 1916.

10. Buerger, Leo: Concerning Certain Types of Hemorrhagic Nephritis, *M. Rec.* **94**: 1057-1061 (Dec. 21) 1918.

7. Cabot, Hugh, and Crabtree, E. G.: A Classification of Renal Infection with Particular Reference to Treatment, *Boston M. & S. J.* **174**: 789-785 (June 1) 1916.

I have been greatly interested in the chronic form of hematogenous nephritis of bacterial origin. In a number of instances, I have explored and found small, cortical, pimple-like collections of fluid in the kidney in various stages of sterilization. I have been able to link up another most interesting sequel to the condition, namely, the occasional deposit of calcium carbonate in such an infected area, usually close to or connected with the capsule, which produces a roentgen-ray shadow resembling that of stone. The diagnosis of stone is often justified by the history of the acute, severe attack which marks the onset of infective nephritis. I have known such deposits of lime to form as soon as two months after the primary symptoms. Why, in some persons, lime is deposited during the process of the cure of the infection, I do not know. The kidneys in such cases are often painful. Our patients were relieved by the excision of the lime masses, and by decapsulation. In several cases, the deposits of lime were deeper in the cortex. There are almost no urinary signs or symptoms in these cases, as the masses are in the cortex with little communication with the collecting parts of the kidney. It is very evident, too, that in some painful kidneys, with dense scars in the capsule, the origin is similar, and the condition may be relieved by decapsulation. I have been slow to admit that capsular compression of the kidney could be the cause of symptoms, but I have seen at least three cases in which hypertrophy of the remaining kidney within its fibrous capsule, after the removal of its fellow for disease, produced pain from the stretching of the capsule. The kidneys were low and movable, and the patients thin, and I was able to follow the compensatory hypertrophic enlargement by palpation, and to satisfy myself that the hypertrophy caused the pain.

In our experience, decapsulation has been valuable in this small group of cases in which there are scars and lime deposits in the capsule of the kidney, and in another group still more rare, that of acute nephritis (Bright's Type 1), in which, as pointed out by Morris,¹¹ the operation occasionally enables the kidneys to functionate when urinary function has ceased and the patients are apparently in a dying condition. For movable kidney, etc., we have seen no good effect from nephrorrhaphy other than the psychic.

ABSTRACT OF DISCUSSION

DR. HUGH H. YOUNG, Baltimore: Dr. Mayo's paper is somewhat parallel to an article I wrote a number of years ago, in which I took up the history of urology. Among other things it was easy to prove that John Hunter was originally a urologist, although the pathologists claimed him as the father of pathology, and the internists claim him as the father of modern medicine. In regard to this society, as one of the original founders of it, I may say that the reason we called it the section on genito-urinary diseases, was just with the idea of grouping here all types of scientific men interested in the urinary tract, not only general surgeons but also internists and physiologists and all others who are interested in the urinary tract. Dr. Mayo's point that the surgeon has played a very important part in clearing up the pathology of many parts of the body is, I think, very well taken. We know how the pathologist and the internist groped for many, many centuries over typhlitis and perityphlitis, and had no conception of the internal pathology or

the real fundamental basis of the condition until the surgeon cleared it up. Real pathology of the kidney made very little progress since the time of Bright, and it is interesting to note that Dr. Mayo goes back to the classification of Bright as the standard classification of renal infections. For a number of years the question as to the possibility of a unilateral nephritis has been a mooted one. Casper claimed that unilateral nephritis was possible. The trouble was that these cases were not seen early, or that the disease was not studied thoroughly. Undoubtedly those cases are apt to be caused by some obstruction below. Undoubtedly the kidneys filter out bacteria constantly, which probably is one of the ways the body has of getting rid of bacteria, and happily the kidneys are able to take care of it, and there is no infection in the large majority of cases. When the bacteria are retained there is probably some condition below which prevents their elimination. I think that obstructive conditions of the urinary tract are present in a far greater number of cases than we realize. Whatever is the cause of these conditions is a question that needs working out in every individual case. Dr. Mayo spoke of focal infections in the kidney, one of the most interesting and puzzling conditions met with in the kidney. His fulminating type is rarely seen. They are not generally seen except by the internist or the general surgeon who is called to operate on a man who is desperately sick, and he may accidentally find the condition present. It has been extremely rare in my experience. The relation of that condition to the more chronic types is not very definite, because those patients very frequently die before the condition becomes chronic.

DR. WILLIAM LINDER, Brooklyn: The subject of hematogenous infection of the kidney is of great interest to the surgeon as well as to the general practitioner. It has been pointed out that this disease early involves only one kidney. The clinical picture is one of an acute upper abdomen. The history is one of acute onset, ushered in with a chill and frequently preceded by some external lesion, such as a carbuncle or a sore throat. Examination will reveal a distended abdomen, tenderness in the upper abdomen but no rigidity. This alone differentiates it on the right side from a perforated gastric or duodenal ulcer. It, however, must also be differentiated from an acute perforative appendicitis in the retrocecal region. But the history of appendicitis will aid us not to be misled, i. e., general abdominal pain, vomiting then localization to the right side and finally rise of temperature will point to appendicitis. The most important sign or symptom is tenderness and a sense of rigidity in the costovertebral angle. The Murphy fist percussion sign is of great aid. The urinary findings in this condition do not help us; one may find a few white and red blood cells. Therefore, it can easily be differentiated from an acute pyelitis where pus is frequently found in the urine. In my service at the Jewish Hospital I have operated in more than 100 cases of this type of kidney infection. Some of these patients were pregnant women and a diagnosis of unilateral kidney infection was made and confirmed at the operating table. The treatment in these cases of the fulminating type is nephrectomy; fortunately these cases are few in number. In the subacute type, decapsulation, if the patient is seen early, will save both the kidney and the patient. Hence, the importance of early diagnosis. These lesions, as has been pointed out by Dr. Mayo, are cortical and a metastatic infection is carried by the blood stream. They are minute miliary abscesses and decapsulation allows these abscesses to rupture and drain.

DR. GUY L. HUNTER, Baltimore: I would like to ask Dr. Mayo if he thinks it is really safe teaching to recommend nephrectomy in many of these cases? I am familiar with Brewer's recommendations in that line, but for some reason they have not appealed to me. In the first place, we cannot differentiate, within the first few days, in the acute cases, between these acute cortical abscess cases and mere pyelitis cases. Even in the acute pyelitis case the urine contains albumen, blood and casts, that we usually consider characteristic of these acute cortical infections. There is the same high temperature, the chills, the prostration and, as

11. Morris, H.: Notes on the Surgical Treatment of Affections of the Kidney, *Ann. Surg.* 5: 289-305, 1887; *Surgical Diseases of the Kidney and Ureter, Including Malformations and Misplacements*, New York, Cassell & Co., 1901 2, 670 pp.

far as I know, it is rather impossible to differentiate which the patient has. If you are going to operate at all it probably ought to be done early, and that is Brewer's teaching. I think Dr. Mayo will agree that some of these patients undoubtedly recover spontaneously if we leave them alone and treat them as we would a pyelitis patient. He has spoken of a series of cases in which he finds evidences of past infection, with the lime deposits, showing that some of those patients have recovered. So altogether it seems to me that if we are going to operate at all in these cases it would be safer teaching for the average surgeon to recommend mere drainage, or, perhaps, decortication. If I operated at all on a case of that sort I should prefer drainage. In such cases a few kidneys will be saved, and a few, of course, will remain infected, and perhaps have to be removed later. But in any of these cases, another point in favor of some form of conservative operation is the fact that we cannot tell whether they are unilateral, or bilateral. The symptoms may all be referred to one side; but I have had cases where later, after the patient had recovered, investigation showed that both kidneys were infected, whereas in the acute attack we considered but one kidney to be infected. I would like to ask Dr. Mayo's opinion about these points.

DR. LEON LOURIA, Brooklyn: I have learned of the type of infection of the kidney to which Dr. Mayo refers only during the last few years. The subject was entirely new to me, and it was only by observing the findings on the operating table that I could clarify in my mind the pathology of this disease. Hematogenous infection of the kidney is distinct from pyelitis, it is due to a general infection, with localization in the kidney. There are fulminating cases, which are not recognized at all—or only too late to save the patient's life. At the Jewish Hospital of Brooklyn, I have seen a number of these cases and referred them to the surgeon. The internist sees them first, and he must make a diagnosis. We deal with a general infection very frequently following a tonsillitis, with metastases in one kidney. Sore throat is often followed by chill and fever, but the patient does not localize the pain; still, if you keep in mind this type of kidney infection, and press your finger firmly in the costovertebral angle you easily elicit an acute pain; and if you can exonerate the appendix and gallbladder, the diagnosis is made. After the conclusion has been reached, our attitude in these cases should be identical with that in cases of appendicitis. Nobody can predict the course of an acute appendicitis; so then, in acute hematogenous kidney we cannot be sure, *a priori*, that the infection, left alone, will not destroy the life of the patient, or at least one kidney, while both may be saved by timely surgical intervention. Internists have learned that the appendix belongs to them only until they make the diagnosis, and then it becomes the property of the surgeon. I think the same principle ought to be applied to the acute kidney. A surgeon ought to be consulted; if experience has taught him that this particular kidney can be saved without operation, well and good, we will stand by him; otherwise an operation ought to be performed. No single surgical procedure should be binding, the surgeon ought to proceed without a contract; no promise of saving the kidney can be made to the patient in advance. If on inspection of the kidney the surgeon feels that the organ can be saved, he will not do a nephrectomy, but he will resort to a minor operation, decapsulation, nephrotomy or partial excision. If you are sure that the patient has another kidney, and the partner is functioning properly, the hazard is not great, as we know that man can live with one kidney. A few years ago I said that I felt that nature was provident in giving us two kidneys, in that she consigned one to the surgeon and left one for the patient.

DR. FRANK HINMAN, San Francisco: Two points might be emphasized in this discussion: In the first place, the type of infection. We all know that the colon bacillus does not produce abscesses in the kidney, and it seems to me that this discussion is confused by the fact that many of these acute infections of the kidney, probably the great majority of them, are due to the colon bacillus. And these kidneys can always be left alone. They do not demand operation in

the acute stages. On the other hand, streptococcus or staphylococcus infections of the kidney are the type of acute infections of the kidney that are so frequently hematogenous and surgical and are the kind that sometimes demand immediate operation to save the patient's life. I inferred from Dr. Mayo's paper that this was the type he referred to particularly. The other fact that should be emphasized is that the kidney is not an excretory organ for bacteria by choice; it is only that when the circulation is overwhelmed with bacteria, or when the kidney is injured, or is nonresistant that infections of the kidney occur. This can be shown experimentally in a great many ways. If you inject animals with living cultures of bacteria you will find that the liver and the lungs, the gallbladder, and the other organs of this animal will be loaded with bacteria, and the kidney will show relatively few. The kidney, therefore, is not an excretory organ for bacteria. Marked infection of the kidney occurs much more often with potato cultures than with broth cultures due to the clumping and the resultant capillary emboli in the former instance. The type of infection, whether colon bacillus or coccus, and a knowledge of the particular renal injury present, in view of the fact that the kidney is not, by choice, an excretory organ, can often shed considerable light on questions of surgery, particularly in reference to these acute septic kidneys.

DR. WILLIAM J. MAYO, Rochester, Minn.: I was particularly interested in having Dr. Hugh Young lead this discussion because I believe that in the Brady Institute which is under his charge there is a great opportunity to solve such problems as this by attacking them from every side. Organized effort will clear up moot questions in urology just as has been done in other fields. Dr. Hunner has brought up an important point with regard to nephrectomy. Nephrectomy is only for the exceptional case. In a very high percentage of these acute cases the patients get well or eventually develop a chronic condition of pyonephrosis and come to operation later. Many cases may be in their inception so mild that we do not appreciate the condition as an acute, or even a subacute process presenting so few symptoms. There are cases, however, as pointed out by Brewer, in which nephrectomy is necessary to save life. I have removed a kidney in a few such cases. On two occasions I operated from in front, expecting to find acute suppurative cholecystitis, but on exploration I discovered that the real seat of the trouble was in the kidney. The kidney when removed was found to be riddled with acute abscesses. I could not conceive that such a kidney might be cured by decapsulation or drainage. It should not be assumed for a moment that the kidney should be removed early in all cases; probably nine out of ten such patients in this condition will either enter into a chronic or get well. I wish again to call attention to the fact that these cases may sometimes be seen in the process of sterilization. In some instances little pimple-like masses were shown to be bacteria free, and others contained bacteria, and in the late stages of cure carbonate of lime may be deposited in the sterilized area giving rise to roentgen-ray shadows.

A Literary Fragment on Syphilis and Tobacco.—The following quotation from a curious document printed in London in 1604, entitled "A Counterblaste to Tobacco," affords some insight, both into the belief of that day as to the origin of syphilis and as to its treatment: "For Tobacco being a common herbe, which (though under divers names) growes almost every where, was first found out by some of the barbarous Indians, to be a Preservative, or Antidot against the Pockes, a filthy disease, whereunto these barbarous people are (as all men know) very much subject, what through the uncleanly and adust constitution of their bodies, and what through the intemperate heate of their Climat: so that as from them was first brought into Christendome, that most detestable disease, so from them likewise was brought this use of Tobacco, as a stinking and unsavorie Antidot, for so corrupted and execrable a Maladie, the stinking Suffumigation whereof they yet use against that disease, making so one canker or venime to eate out another."