those who are competent to assist him in making an exact diagnosis. Dentists who wish to be successful in the future must thoroughly educate themselves in both medicine and dentistry. The Council on Medical Education should discourage medical schools from conferring medical degrees on dentists who have not taken the prescribed medical course.

DR. W. C. FISHER, New York: Dr. Brown's suggestion as to the establishment of the B.M. degree is a good one. I read a paper last year entitled, "The Need of a New Degree, Bachelor of Medicine." I simply gave it as a suggestion at that time, and backed up my statements by those of Dr. Bevan of the Council on Medical Education of this Association, in which he stated that too much time was being wasted in preliminary preparation before men entered the medical schools; that we could not turn out competent medical men in twenty-nine or thirty years with the present restrictions, and that some new scheme would have to be devised whereby men could go out on their life work earlier. Professor Lowell of Harvard has said that instead of demanding the Bachelor of Arts degree before entrance to medical schools it would be better if we would incorporate some of these studies in the course leading to the Bachelor of Science degree and take one year from the four-year course, instead of spending a couple of years in studies that did not particularly relate to medical education. The man who has an excellent preparatory school education, and that does not mean two years in college, is qualified to undertake the study of the sciences of medicine and dentistry. When we speak of getting better men I am in favor of that, but I am opposed to anything that makes it so late in life before a man can acquire that degree which entitles him to go forth into the world and earn his livelihood.

DR. G. V. I. BROWN, Milwaukee, Wis.: We have been discussing subjects that my paper relates to ever since the meeting opened, and it is clear to me that, in the settlement of this question, the time element alone will not do it, but it is equally clear to me that we should not make any step in this direction until we know best what to do.

CONTINUOUS PAINLESS RENAL HEMORRHAGE AND ITS TREATMENT*

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The selection of the title for this paper has been made with the purpose in view of excluding all other conditions having hematuria as an accompanying symptom, and limiting the name to those cases in which there is constant and continuous bleeding from the kidney over a considerable period of time unaccompanied by pain except such as could easily be explained by the plugging of a ureter by a blood-clot. Objection might be had to the word "continuous," for in many cases the bleeding stops either spontaneously or after the administration of medicines or of serum, or as the result of mechanical therapy directed to the kidney pelvis. The bleeding may start again and continue for weeks or months, during which time blood in varying amounts may be recognized macroscopically in the urine. The term "essential renal hematuria" is in my opinion a proper name for this condition, because it is from the kidney substance that the bleeding takes place. This name, however, should be applied only to those cases in which absolutely nothing pathologic other than blood appears in the urine, and where no lesion can be demonstrated by a competent urologist. When epithelium from the tubules or the pelvis, or when pus, micro-organisms or crystals of any type are

found in the urine with the blood, or when with the Roentgen ray or by palpation a tumor or foreign body be diagnosed in this organ, or when intermittent or chronic hydronephrosis is found or suspected, it would be manifestly wrong to call such a condition essential renal hematuria just because blood is present in the urine. I shall have occasion later to refer in a more detailed manner to the subject of differentiating this condition from others in which bleeding exists.

The literature on this subject is too voluminous to review at length. To those who are interested I may refer to Kretschmer's article,¹ which brings the bibliography up to 1907, and White's paper,² dated 1910. The bibliography since these reviews were published is also quite extensive, and I shall only give the gist of the opinions expressed therein.

Pathologists are in apparent accord that the lesions found in renal hematuria are confined mostly to the glomeruli, and the bleeding has therefore been attributed to a glomerulonephritis. The changes are briefly as follows: while most of the glomeruli are normal, a few show absence of some of the endothelial cells lining Bowman's capsule, while others show hyaline degeneration, and some have undergone fibrosis. Kidneys that had bled for a long time present more advanced changes of the same kind with a beginning fibrosis of the intertubular tissue, in other words an interstitial nephritis—*identically the same changes which are found in kidneys which have been passively congested for some time.*

Senator³ cites a case in which he could find no lesion of the kidney either macroscopically or microscopically, and believes the condition to be a renal hemophilia. The general opinion, however, is that if these kidneys were scrutinized more carefully the changes described above would have been found.

Much has been written to show that hemorrhage from the kidney is fairly common in lesions of that organ. Elsner⁴ tabulates the cases in which blood was found in the urine as follows: Chronic tubal nephritis, 33 per cent.; acute tubal nephritis, 100 per cent.; *secondary congested kidney*, 100 per cent.; chronic interstitial nephritis, 14 per cent. In the same article he cites cases of profuse hemorrhage with no symptoms and no findings in the urine which would indicate nephritis, and yet these after a long time (in one case five years) presented symptoms or urinary findings which are expected in Bright's disease. In the vast majority of these cases the blood was microscopic, and they, therefore, cannot be dignified with the term "hematuria."

The question naturally arises, Why is there such profuse bleeding in essential renal hematuria when pathologic changes are so slight, whereas in the kidneys of patients who have succumbed to nephritis in which the lesions were a hundred times worse there had been no hematuria? Are these pathologic changes the cause of hematuria as is generally thought, or are they the result of the process which causes the bleeding? In my humble opinion the prime cause is a chronic passive congestion. Although positive proof of this assertion is still lacking, I shall essay to bring forth some facts which tend to favor this view.

1. Kretschmer, H. L.: Beitrag zur Frage der essentiellen Nierenblutung, Ztschr. f. Urol., 1907, i, 490.

2. White: Quart. Jour. Med., 1910-1911, iv, 509.

3. Senator, H.: Ueber essentielle Nierenblutung und renale Hämphilie, Berl. klin. Wchnschr., 1910, p. 205.

4. Elsner: New York State Jour. Med., 1913, xiii, 144.

* Read before the Section on Genito-Urinary Diseases at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

It is not uncommon to find blood in microscopic quantities in the urine from a kidney which is out of position, particularly when hydronephrosis is present. Occasionally even free bleeding is present. When the kidney is fixed properly in position this bleeding ceases. It is therefore fair to presume that the hemorrhage was due to a passive congestion, because while the inflowing blood-supply was not interfered with, the outflow was impaired. This would naturally come to pass in certain positions of the kidney because the caliber of the vein is more easily reduced in size than that of its companion artery for the following reasons:

1. Because its wall is thinner than that of the artery.

2. The pressure behind the inflow is many times greater than in the renal vein.

3. Because there are as a rule multiple renal arteries, while multiple renal veins are the exception.

If this passive congestion lasts a certain length of time, we shall find the identical changes that are seen in kidneys removed for essential renal hematuria. What more natural therefore than to assume that the cause of essential renal hematuria is a passive congestion?

Another reason for our belief is that these cases of essential renal hematuria cease to bleed spontaneously, and remain apparently well for varying periods, often never to recur. How may we account for this except for the explanation that the kidney after being twisted on a short pedicle so as to narrow the caliber of the vein subsequently resumes its proper position? The outflow of blood being no longer obstructed, the passive congestion clears up and the bleeding stops. In a week, a month, a year, the kidney may for some reason again be out of position, passive congestion once more occurs, and bleeding ensues.

We can thus account for the fact that Senator and others found no microscopic changes in some of these excised kidneys, while other observers have frequently found the glomerulonephritis previously described. If the kidney started to bleed as soon as it became congested and was removed before the pathologic changes could occur, it would be of course perfectly normal to the naked eye and under the microscope. In those cases, however, in which the passive congestion had continued over a longer period before removal, we would find this glomerulonephritis. If the kidneys were taken out at a still later stage we would probably find all the changes that accompany a chronic interstitial nephritis.

It seems to me that a passive congestion of one kidney can be accounted for only by a twisted short pedicle, for I can conceive of no other way, barring outside pressure on the pedicle, that would give rise to this condition. Several other observations have led me to believe that a twisted pedicle is the cause of this condition. In stereoscopic pictures of the collar-gol-filled kidney pelvis the organ occupies normally a certain fixed position, with the pelvis directed downward, inward and forward toward the ureteropelvic junction, while in cases in which the kidney cannot be called loose, but is nevertheless giving trouble, we have found its position so changed that the convex border instead of being almost parallel to the spinal column, is nearly at a right angle to the spine, as shown by the direction of the pelvis. In other cases again, particularly the one I recall to mind, in which a beginning hydronephrosis existed with blood in the urine, the kidney was so placed that its convex border

presented toward the abdominal wall. The patient was cured by fixing the kidney, which was not loose, in its proper position.

Another argument: In cases of hydronephrosis with concomitant bleeding the latter symptom was arrested by simply fixing the kidney properly; and cases of essential renal hematuria in which no hydronephrosis was present have been cured by fixation.

The necessity of differentiating this condition from others accompanied by renal bleeding is quite apparent, but not always so easy. The microscope must show red blood-corpuscles unaccompanied by pus, microorganisms, casts or epithelium from the kidney or its pelvis. The bladder must be free of disease, the ureter mouths normal, the flow from the ureter catheters normal, the kidney must be impalpable, or if palpable no larger than normal and it must present no irregularities to the hand. The Roentgen ray must show the kidney clearly when a pyelogram is made, and the kidney pelvis must be normal in shape and size. If the contents of the kidney pelvis exceed 10 c.c.⁵ I do not consider it normal. With these restrictions we can exclude renal tuberculosis, stone, abscess, hydronephrosis and the commoner conditions in which hemorrhage occurs.

In tumors of the pelvis, by the time bleeding is continuous, one would expect epithelium in large quantities from the pelvis, together with debris from the constantly degenerating neoplasm. Continuous hemorrhage is extremely rare in tumors of the kidney until the enlarged growth has attained such a size as to be palpable.⁶ Pyelitis will show besides red corpuscles other elements in large numbers in the segregated urine. Nephritis presents well-known symptoms or urinary findings which make this condition recognizable. *Besides a true nephritis cannot be one-sided.*

A diagnosis of essential renal hematuria having been established, the question arises as to its proper treatment. Ordinarily we should keep the patient under observation unless the anemia begins to give grave concern. Many of these patients never become anemic. In the meantime we may try the injection of blood-serum, a rest in bed for a short time, and a properly fitted lower abdominal corset. As to the injection of epinephrin into the kidney pelvis,⁷ I consider this procedure more of a diagnostic than a therapeutic measure. If the bleeding arises from the pelvis the urine should be free from blood, or at least clearer for a short time following the injection, although it should not (theoretically) affect the bleeding in essential renal hematuria. In the three cases in which I have tried epinephrin the result was nil.

If the anemia progresses in spite of these procedures it is time to consider surgical intervention. One must, however, think long and arduously before daring to operate, and when operation is finally decided on, nephrectomy should be the last thought.

Years ago, before the advent of the present diagnostic facilities, the kidney was bisected at the operating table in order to determine whether stone or tumor was present. If neither was found the organ was securely sewed together. To the surprise of the operator the bleeding from the kidney stopped,

5. Childs, S. B., and Spitzer, W. M.: Roentgenographic Study of the Normal Kidney, Its Pelvis and Ureter, *THE JOURNAL, A. M. A.*, Sept. 30, 1913, 925.

6. Barney, J. Dellinger: Reports of the Meetings of the New England Branch of the American Urological Association for the Winter 1912-1913.

7. Suggested by Kretschmer, with report of case in which bleeding was stopped, *THE JOURNAL, A. M. A.*, July 5, 1913, p. 17.

although nothing had apparently been done to bring about this happy result. This occurred so often, and was so frequently reported in the literature, that it came to be adopted as a routine treatment for essential renal hematuria. Many a kidney has been bisected not for the sake of diagnosis but in the belief of the efficacy of nephrotomy. In all probability, however, it was the suturing of the kidney, with its consequent removal of a large portion of the kidney tissue from the renal circulation (partial nephrectomy) which produced the cure. Much has been said in praise of this bisection as a cure, and very little of the dangers that accompany it. In fact, a recent article by Payne⁸ asserted that not a single case has been reported in which this procedure failed to relieve this condition. While this may be true, it is surely contrary to reason to bisect a kidney if it be not the bisection which cures, when in a large number of cases the secondary bleeding from the incision is so great as to necessitate reopening the wound and removing the kidney.

If the arguments as to the cause of essential renal hematuria be valid, what is the proper surgical treatment? Nephropexy with decapsulation, in the earlier stages, with a careful attempt to place the kidney in the position where it belongs. It may not be amiss at this point to call attention to the position of the kidney when properly placed. The kidneys are so situated that the upper poles are nearer together than the lower, the distance between the lower poles being almost twice as great as between the upper. The convex border of the kidney is posterior to the pelvis. If accessory arteries are found, they should be cut and tied. If the operation be not successful in stopping the hematuria, then at a subsequent operation a continuous mattress suture may be placed in the kidney, running from pole to pole, just as one would place it had the kidney been bisected. Experimental work on dogs by me has demonstrated that no evil results follow from this method of suture excepting the infarction that must of necessity ensue. There is no doubt that it is this partial nephrectomy which cures these patients, and not the bisection of the kidney. The rationale of the cure is probably the following: The vein capable of carrying only a certain percentage of blood back from the kidney is required to transport a smaller amount after this partial nephrectomy and is thus able to perform its function even though its patency is impaired.

I have had eight cases of this condition under observation, and will give a concise report of them. In one the bleeding ceased after four months, and did not return during the following eighteen months, after which time I lost sight of the patient. In four more the bleeding had persisted for periods of from six months to two years before they came under my observation. As the patients did not suffer from the hematuria I advised delay, and two of them passed from sight. I feel gratified that I did not advise surgical interference. Two others, with whom I am still in touch, seem to be in perfect condition despite hematuria of over two years' standing. In another case I advised surgical interference and assisted at a bisection of the kidney, which subsequently bled much worse than before by way of the bladder as well as from the wound, so that after eleven days I advised nephrectomy in order to save the patient's life. In still another case, nephropexy cured the condition. In the

remaining case the hemorrhage was very free, and the surgeon who had referred the case to me for diagnosis removed the kidney as soon as I traced the bleeding to its source. In the two cases in which the kidney was removed and in another one belonging to a colleague I had an opportunity to study the pathologic changes. In two of them the glomerular changes described above were the only lesions found, while in the third, in which bleeding had persisted for eighteen years, microscopic examination revealed intertubular fibrosis besides the glomerular changes.

CONCLUSIONS

1. The changes found in kidneys of essential hematuria are identical with those found in passive congestion and are therefore caused by passive congestion.
2. The bleeding is due to passive congestion, the kidney being an organ so constructed that it must of necessity bleed in the presence of passive congestion.
3. It is erroneous to ascribe the bleeding to nephritis, as there are no clinical symptoms or urinary findings indicative of nephritis, nor can the latter be unilateral. Still, it is admitted that if the bleeding continues, the pathologic changes in the kidney would be the same as in chronic interstitial nephritis.
4. The passive congestion occurring in one kidney only is due to some interference with the outflow of blood, which comes from a twisting of the kidney on a short pedicle.
5. Operative interference is warranted only when it becomes necessary to save the patient's life because of an increasing secondary anemia.
6. Bisection of the kidney for the cure of this condition is contra-indicated, and likewise dangerous.

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ABSTRACT OF DISCUSSION

DR. HERMAN L. KRETSCHMER, Chicago: The term "essential hematuria" originally was meant to be applied in those cases in which no reason could be found for the hemorrhage. That, of course, excludes cases of stone, tumor and tuberculosis. In view of the fact that Dr. Spitzer found aberrant blood-vessels and a movable kidney, these cases could not be put into the class of essential hematuria; because he found some reason for the bleeding. The term is one that is used very recklessly and carelessly. A great many times the term essential hematuria is "hooked" onto a case that is not sufficiently worked up. It is an expression of our limitations in pathology and diagnosis. If these cases are studied bacteriologically, sections made, not from a part, but from the entire kidney, and cultures made from the urine, in the large majority of instances some reason for the bleeding will be found. The ease with which lesions may be overlooked is illustrated in a recent case of Schupbach, who had a patient in whom the diagnosis of essential hematuria was made. The kidney was decapsulated, the bleeding did not stop, and it became necessary to remove the kidney. The kidney was cut open and no pathologic lesion found. Serial sections, however, revealed the presence of tuberculous lesions which had eroded a small blood-vessel, thus causing the hemorrhage. The more these cases are studied, the less we will use the term. I cannot agree with Dr. Spitzer that his case was one of essential hematuria, because he found a movable kidney. Nor can I agree with him that we have the same pathologic factor in this as in passive congestion. As regards the unilateral occurrence of the hemorrhage, we should consider the possibility of these lesions being bilateral. Dr. Spitzer says nephritis would not be unilateral. That is a good point, particularly when these patients are to be subjected to nephrectomy. I recall a case in which a patient

8. Payne: Surg., Gynec. and Obst., July, 1913, p. 93.

had a one-side hemorrhage. The diagnosis of essential hematuria was made, the kidney was decapsulated, and the bleeding stopped. I saw him a year later and he was bleeding from the other side. Sections from the second side showed extensive nephritic changes. If we had removed that patient's kidney it would have been very unfortunate, because both sides were diseased. I do not think we can call attention to this too often. All that nine out of ten surgeons require to know is which side is bleeding, and they do a nephrectomy. Cases of hydronephrosis associated with bleeding should not be included in these cases of essential hematuria. Dr. Braasch pointed out last year that in a certain number of cases of pyelitis profuse bleeding occurs. This is another argument in favor of care in analyzing these cases. Too many of these patients are operated on, the kidneys exposed, and the diagnosis of essential hematuria made. Even if such a kidney were cut into, unless the pelvis were opened, it is easy to see how a lesion in the renal pelvis could be overlooked. I therefore wish to be recorded as opposing nephrectomy in the treatment of this group of cases except as a last resort. In many of these cases of essential hematuria the bleeding stops with less radical measures. In a case reported a few years ago by Dr. Hagner the bleeding ceased following ureteral catheterization. Many cases of bleeding stop with a simple procedure. Catheterizing the bladder in one case stopped the bleeding. The injection of epinephrin into the kidney is a valuable procedure, and so far as I know was first used by Dr. Young. We must bear in mind that most of these cases have an organic basis and hence we must not treat them too long with the epinephrin injections.

DR. W. F. BRAASCH, Rochester, Minn.: I want to call attention to a condition which is easily overlooked and which may be regarded as due to essential hematuria, when in reality it is due to an inflammatory condition of the renal papillae. The accompanying clinical data are much the same as in the so-called essential hematuria with the exception that a varying number of pus-cells are found in the urine often together with colon bacilli. On section of the kidney, the papillae appear somewhat inflamed or slightly ulcerated and occasionally white areas, which are the result of necrosis, are visible at the tips. In the past year, we have found two cases in which we could say quite definitely that the hematuria was due to a condition which might be called chronic papillitis. Both of the cases were given pelvic lavage and autogenous vaccine without effect, so that nephrectomy was necessary.

DR. HUGH CABOT, Boston: This opens an interesting and difficult question, and I am not sure that I entirely understood Dr. Spitzer. He will correct me if I am wrong. I object to the use of the term essential hematuria, because it seems to me merely a cloak for ignorance. We had better say frankly we do not know what is the matter with a patient; but up to the point where absolutely nothing can be found let us classify these cases according to the cause of hemorrhage. I do not think that I agree with him in regard to bleeding from cases of chronic nephritis. I am sure that unilateral bleeding in chronic nephritis occurs. At least two cases were observed over a term of years in which intermittent bleeding occurred and a diagnosis of chronic nephritis was made, the bleeding having been first from one kidney and then from the other. We never happened to see it coming from both. I have no doubt that during that time a period of bilateral bleeding did occur. The bleeding in these cases of chronic nephritis I believe to be due to localized changes in the kidney, but I think it rarely occurs in chronic nephritis because there are few cases of such severity as to cause bleeding in which a diagnosis cannot be made by the other evidences of chronic nephritis. I think we are apt to overlook the fact that chronic nephritis is not a disease of the kidney; that there are other conditions, circulatory and otherwise.

Here is a point concerning which I am not at all sure that I understood Dr. Spitzer, but I rather gathered that he said that conditions causing passive congestion in the kidney

would ultimately cause changes in the kidney similar to chronic nephritis. That I believe to be based on pathologic error. I believe it necessarily occurs from a reaction in the kidney, and is always glomerular or arteriosclerotic. I do not believe any amount of change in the position of a kidney will produce the essential lesion of chronic nephritis. That mobility of the kidney can produce hematuria has been known for many years, and that the replacement of these movable kidneys will stop the hematuria is also well known. In those cases the diagnosis of kidney mobility and not essential hematuria should be made.

I did not hear Dr. Spitzer discuss those cases of varicose papillae which are now, I think, proved to be pathologic entities beyond the shadow of a doubt. They will give rise to renal hematuria without any other evidence but that of blood in the urine. This hematuria may be severe in character, may produce a high degree of secondary anemia, and has been treated by nephrectomy, which I believe to be entirely unnecessary. But the fact that it has been treated by nephrectomy has given us the pathologic material on which the diagnosis has been made with certainty.

That a condition that we may be required to call essential hematuria may occur cannot, I think, be denied. It simply is a confession of ignorance, not a confession of wisdom, and more and more as these cases are narrowly examined with the best methods we shall find them in this inflammatory category to which Dr. Braasch referred, or with some demonstrable lesion as the cause of the bleeding. That the bleeding occurs as an act of God, without any reason whatever, is still to be shown.

DR. J. W. SQUIRES, Charlotte, N. C.: A case of renal hematuria came under my observation. Ureteral catheterization was done and the functional output from each kidney as shown by phenolsulphonephthalein, was 15 per cent. About one month ago, ten months after the first ureteral catheterization, the patient came in for another catheterization. At this time the right or bleeding kidney, was putting out 5 per cent. in one hour and the left 25 per cent. in one hour. When the patient first came into my hands I made a diagnosis of essential hematuria because of the fact that the functional output was normal and I could not find any pathologic lesion or any etiologic factor for the bleeding from this kidney. After the last catheterization I told him that there was now a demand for an operative procedure, and advised it. In the operation we found a hypernephroma of the right kidney. I wish to emphasize that in unilateral renal hematuria the function of the two kidneys may be practically the same when the case first comes under observation, but if kept under observation for sufficient time some pathologic lesion will be detected.

DR. W. M. SPITZER, Denver: Dr. Kretschmer, I think, misunderstood me. I stated that at the time of operation one should replace the kidney and sew it in its proper place, and if accessory arteries are found they should be cut. In my paper I distinctly limited these cases of essential hematuria, so-called, to cases in which a competent urologist could find no lesion in the kidney, no micro-organisms in the urine, no pus, no epithelial cells from either the pelvis of the kidney or the kidney itself; in which no tumor was palpable, and in which the roentgenogram of the kidney as well as one of the collargol-filled kidney pelvis, showed no tumor; and I also excluded hydronephrosis, and then called the rest of the cases those of essential renal hematuria. As to bleeding from both sides, I have never seen a case. I have never even seen bleeding first from one side and then from the other, but if I should see such a case I should very strongly suspect a nephritis, although I agree with Dr. Cabot, that signs of nephritis and urinary findings should be present. In the cases of lesions in the papillae, mentioned by Dr. Braasch, we should find enough pelvic epithelium in the urine to help us diagnose them as such. I mentioned that I considered the injection of epinephrin a diagnostic measure. We might get a cessation of hemorrhage by injecting epinephrin in cases in which the papillae are bleeding. I do think, however, that this is a rare cause of bleeding. I do not see how

we can refuse to call these conditions cases of essential hematuria when they are narrowed down. It is the kidney substance itself that is bleeding. It is a cloak for our ignorance to call it idiopathic renal hematuria, but it is not so to call it essential renal hematuria. Dr. Cabot doubts that the changes can be the same in these kidneys as those we find in chronic interstitial nephritis. They will be the same eventually. If a chronic passive congestion of the kidney exists, the same changes that I have described will be found, and after that a round-cell infiltration and finally an intertubular fibrosis, and thus, eventually, all the changes of chronic interstitial nephritis. Dr. Cabot also spoke of varix of the papilla. The reason I did not mention it was that I was afraid to. Harry Fenwick has described six cases and Dr. Cabot has described one. I do not see how it could be demonstrated microscopically after the kidney was excised. It may be it can be demonstrated macroscopically while operating. Lately there have been no cases of varix of the papilla reported. Dr. Squires speaks of a case of tumor of the kidney with accompanying hemorrhage, in which there was a low output of phthalein. This is as it should be. If careful roentgenograms had been taken in his case, I do not doubt that the diagnosis of tumor would have been made before the patient was operated on.

EXTRAPERITONEAL RUPTURE OF THE BLADDER

ITS SURGICAL MANAGEMENT *

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Extraperitoneal rupture of the bladder, for a serious injury, has received little specialized study or attention, and when this can be said of a surgical condition of such magnitude it follows almost as a matter of course that its management has been, as a rule, inefficient and unsatisfactory. Some may criticize this statement on the ground that hospital statistics show that a large percentage of patients with such injuries are at present discharged from the hospital alive and with the record "recovered." Still, if one will take the trouble to investigate these patients so discharged, it will be found that almost all of them have recovered only sufficiently to be discharged from hospital, and that most of them have been left invalided—the majority, severely so.

Peritoneal rupture, on the contrary, has been carefully studied, and the surgery applied to it has been radical and eminently satisfactory. Severe violence directed against the suprapubic or perineal region may result in extraperitoneal rupture. When violence is simultaneously exerted in both these directions, the bladder has small chance of escaping severe damage. The worst cases of extraperitoneal rupture are those associated with fracture of the pelvis. In many such cases violence has been severe enough to cause general destruction and to make death inevitable. In these extreme instances surgery, at its best, may be ineffectual. It is in the cases uncomplicated by pelvic fracture that surgery has a chance to show its efficiency.

Extraperitoneal rupture generally occurs in the region of the trigonum; less frequently above, under the space of Retzius, and occasionally at the side of the vesical neck. In most instances the injury consists of one tear, less frequently of two, and occasion-

ally of even more. Multiple ruptures usually occur in connection with fracture of the pelvis, and some of them are in the nature of punctures caused by the sharp ends of the fractured bone. If a rupture is small and perhaps incomplete, the amount of urine extravasated may be correspondingly slight, the result being simply a circumscribed area of involvement. A case such as this, treated expectantly, can leave the patient with nothing more than a corresponding area of pericystic sclerosis. The symptoms arising from such sclerosis may be slight or severe, according to the extent and position of the lesion. In the great majority of cases, however, owing to the volume of the extravasation or to a complicating secondary infection, expectant treatment invites a fatal termination. Following such injury, the extravasated fluid tends as it increases in amount—owing to a continuance of vesical distention or to pus formation from secondary infection—rapidly to extend itself, so that after the extravascular space about the site of the rupture is filled, active burrowing follows. The directions which can be taken by such burrowings are various. One point, however, seems from my experience to be reasonably assured, and that is that the most dependent part of the extravascular space—in other words, the postprostatic area in which the seminal vesicles lie—always becomes involved; primarily, when the extravascular rupture is, as is usual, at the base of the organ and secondarily, when it is elsewhere. This is a point of the utmost importance to be kept in mind, as will be seen in considering surgical treatment.

Further burrowings point outside the pelvis. They may extend behind the peritoneum—sufficiently, perhaps, to involve the perinephritic region on one or both sides—or they may mount upward, filling the space of Retzius in the middle, or the inguinal regions to either side. The extension above the space of Retzius may result in a marked hypogastric tumefaction, liable to be mistaken for an overdistended bladder. Or, following extrapelvic deviations of the pelvic fascia, the burrowing may invade the thigh, passing out of the pelvis under Poupart's ligament, generally externally to the internal iliac vessels; or it may reach the scrotum, following along and externally to the structures occupying the inguinal canal. The buttocks can be invaded, the paths being via the obturator or the great sacrosclatic foramina. These last paths, however, are rarely taken, as they are narrow and devious. Extravasations can break through the pelvic fascia into the ischio-rectal space, the perineum and elsewhere, but the assumption of such directions is not to be expected unless the trauma causing the bladder rent likewise breaks down other structures which would otherwise serve as natural barriers.

There are no strikingly characteristic clinical symptoms associated with extraperitoneal vesical rupture, and it is on this account that often the injury remains undetected until inflammatory changes or burrowings in connection with the extravasation attract attention. Hematuria is present after the accident, perhaps to such an extent as to fill the bladder with clot; or it may be so slight as to cause no noticeable discoloration. When the bleeding is so slight as not to cause the formation of clot, and when the laceration does not involve the vesical neck, there is not apt to be retention. There is always some pain on urination, and this may be severe. The pain, however, associated with other parts which have been injured by the

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