BRYSON, (J. P.)

THE ESSENTIAL FACTOR IN THE ÆTIOLOGY OF STRICTURE, AND ITS BEARING UPON THE QUESTION OF RADICAL CURE.

BY

JOHN P. BRYSON, M. D.,

ST. LOUIS.

REPRINTED FROM THE JOURNAL OF CUTANEOUS AND GENITO-URINARY DISEASES FOR AUGUST, 1889.

THE ESSENTIAL FACTOR IN THE ÆTIOLOGY OF STRICTURE, AND ITS BEARING UPON THE QUESTION OF RADICAL CURE.*

By JOHN P. BRYSON, M. D.,
St. Louis.

A STUDY of the ætiology and pathogenesis of urethral stricture involves, at the outset, a repudiation of that definition which makes of it a mere mechanical narrowing of the canal, for any such definition includes conditions which no one regards as stricture in the true sense of that term. Polypi and warty growths springing from the urethral wall cause unnatural narrowings, and are not in any sense strictures. The same is to be said of collections of fluid in the peri-urethral spaces, and of tumors pressing upon the duct. An acute inflammation of the prostate gland certainly causes a lessening of the dilatability of that part of the urethra, yet it is universally conceded that stricture, as a disease, does not affect this part of the canal at all. It is only when one regards stricture as a pathological condition of the urethral wall and subjacent tissues that he is in a position to study, in a rational, scientific manner, the factors concerned in its inception and development. It is, therefore, of stricture as a disease, and not as a mere mechanical narrowing of the duct, that I would speak here.

If, now, we compare a stricture with one of those congenital narrowings not uncommonly met with in practice, and which is quite as much an unnatural narrowing of the canal, we easily note several differences both in structure and in pathological behavior. In the case of the

* Read, in the discussion of "The Question of the Radical Cure of Deep Urethral Stricture," before the American Association of Genito-urinary Surgeons at its third annual meeting, May 21, 1889.

stricture, we observe that it not only causes a narrowing of the duct, but that this constantly increases. In the case of congenital coarctation the canal is narrow, it is true, but that narrowing does not increase, but remains stationary. There is, then, an activity manifested by the former disease (stricture) which is totally absent in the latter condition (congenital narrowing). We observe, secondly, an anatomical difference, and it is one of great importance. In the case of the malformation, the point of narrowing is lined by normal and healthy mucous membrane, while the stricturing bands are overlaid by a mucous membrane, which is altered by inflammation or otherwise diseased, or is totally absent (cicatricial stricture). If, now, we apply the same treatment to the two cases, we will observe a corresponding difference in the result obtained. Division of the congenital narrowing is not followed by recontraction, while in the case of the stricture the disposition to recontraction is so marked and distinct that to prevent it really constitutes the surgical problem the solution of which would rob this formidable disease of its terrors for surgeon and patient.

One may say, then, that the definitive characteristic of stricture, as a disease, is the persistent tendency of its constituent elements to multiply, to become more dense, and to contract toward the axis of the affected canal. This would exclude congenital narrowings from the list of diseases, and leave them where they belong—among the obstructing conditions, they being malformations and lacking the essential element of activity.

In studying the pathogenesis of this disease we must also make a distinction between those conditions which permit the process to begin and to continue, and the stricture-building itself. An ulcer, for instance, is not a stricture, and can never become one while it remains an ulcer. It is when the ulcer heals, and the site is replaced by a cicatrix, that the pathological processes begin, the sum of which is stricture-building. In a word, it is the changed condition of the mucous membrane which permits the stricture-forming. Mere inflammation of the urethral mucous membrane, even in its chronic form, is not stricture, and it is only in certain cases that the urethral mucosa is so altered by it as to permit of that periurethral fibrous overgrowth which constitutes the disease we name stricture. Here, again, we have an altered condition of the mucous membrane preceding the stricture development. Any number of times we see both of these pathological processes (inflammation and ulceration) approaching, without involving the urethral mucous membrane, from another direction, and still no stricture-building is inaugurated. If, however, either of them, coming from whatever direction, reaches and modifies the mucosa, we seem invariably to see the inception of the stricture disease, provided always that this altered state remains for a sufficient length of time. The essential thing seems to be that the mucous membranes undergo some not

yet clearly defined change. This necessary thing being once accomplished, we have a steady and persistent growth of fibrous connective tissue underneath the affected portion of the membrane, pushing it more and more toward the axis on the one hand, and on the other involving in its meshes the underlying and adjacent spongy elements, causing their atrophic degeneration, and finally their total disappearance. Thus we see it is underneath the membrane that the pathological process is active; and even in those cases of cicatricial stricture, on the urethral side of which there is no mucous membrane whatever, it is in the peri-urethral tissues that development of new tissue goes on, the urethral side only undergoing a change marked by condensation. In other words, we have in all cases a chronic peri-urethritis, with a distinct tendency to contract toward the axis of the canal.

I would therefore propose the name of chronic contracting peri-urethritis by which to designate the stricture disease.

If we include under this designation all cases belonging there, we may at once eliminate the microbe as an essential factor in the ætiology. Certainly no one will accuse the traumatic cases of being microbian in origin or the microbe of having any essential pathogenetic relation to them. The researches of Oberländer and Neelson very clearly show the manner in which the microbe of gonorrhœa may, as it so frequently does, play the principal part in that modification of the mucous membrane which is apparently necessary to the inception and continuation of the disease. In fact, the disease gonorrhœa has long been known to hold this relation, the great majority of inflammatory strictures having followed it in such a way as to leave no room for doubt.

To an English surgeon is due the credit of making the most important contribution to our knowledge of the pathogenesis of chronic contracting peri-urethritis we have yet had. It was Mr. Reginald Harrison, of Liverpool, who showed that the chronic, persistent deposition of the so-called stricture-tissue was really a reaction of the underlying tissues against the leakage of urine or some of its numerous constituent elements through the altered mucous membrane; and this luminous demonstration not only adequately accounts for the different steps in the pathology of the disease, but it points the direction which radical treatment should take. Under this conception of the pathogenesis of the disease we have the changed condition of the mucosa, which permits of urine leakage as the essential though passive factor, and the leakage as the active element, rousing and maintaining the antagonism of the underlying tissues. This does away with the old mechanical one completely and adequately clears the field for treatment.

So long as the old mechanical conception held sway in the surgical mind it was but natural that a purely mechanical treatment should be

resorted to in order to conquer it. In a certain sense great benefit has resulted from this, for surgeons were stimulated to increased and never-ceasing efforts to devise new and better apparatus and technique. It was out of these efforts and the opportunities which they afforded for observation that our present admirable treatment known as "inflammatory atrophic dilatation" grew, and we have learned from our predecessors how to apply this method in a rational, scientific manner to the merely obstructive part of the disease. So well indeed has the mechanical side of the matter been met that it may be seriously questioned whether our present armamentarium will ever be improved upon in any essential particular, or whether there is need for such improvement. With the means at our present command and ordinary skill in their use, one can say that the mere obstruction to urination is, in the great majority of cases, capable of being easily, safely, and adequately overcome by the modern surgeon. Indeed, this does not constitute the difficulty any longer. Here, where we have under consideration the question of the radical cure, it is easily perceived that it is not a question of removal of the mechanical obstacle so much as it is one of permanency of result. It is that treatment which will forever prevent recontraction, or, in other words, which will inhibit the pathological changes whose activity constitutes the disease that chiefly concerns modern surgery, for in our day it is clearly recognized that here lies the danger to the patient.

I can not help thinking that something may be gained by observing the manner in which "that cunningest pattern of excellence, Nature," goes about treating this disease on her own account. If we watch a case which has not been interfered with surgically, we will observe the following course of events: As the narrowing progresses there is a gradual dilatation of the uninvolved urethra immediately behind the stricture. The walls of this dilated portion are rather in a state of atrophy than of inflammation, for we get mucus and necrotic epithelia from its surface rather than pus, and we observe that it is in a state of venous congestion. There are thinning and devitalization; finally, an erosion or an ulcer forms. These changes are apt to be greatest at that part immediately posterior to the deeper edge of the stricturing band and on the inferior wall of the duct. So low are the normal reparative energies that no bands are thrown out to re-enforce it. Finally, we have urinary infiltration, or lower down, where there is yet enough energy remaining, an abscess cavity forms, points at the surface of the body, bursts or is opened, and gives exit to the urine, forming a fistula; but at first this is a fistula with soft and yielding walls. Gradually energy is gained, the tissues begin to resist the urinary encroachments, and we have eventually a fistula with hard walls, this hardening being the result of the development of the same kind of tissue that in the beginning caused the urethral narrowing; and this tissue behaves in the same way—

namely, increases in bulk, condenses, and tends to contract upon the opening so long as urine is permitted to flow through the tract. How of the stricture-band in the mean time? At first, when the fistula has formed and has soft walls, there is no great obstruction offered by it, and all the urine passes through by the new channel; but, however wide the fistulous opening may be, some urine comes in contact with the posterior edge of the stricture, perhaps remains constantly in contact, and so there is the necessary condition for a continuation of the peri-urethral contraction of the stricture-band, and especially of its posterior edge. This answers to this stimulation just as the stricture did originally, and tightly seals the urethral outlet of the pouch, forcing the urine to pass by the new way. It takes time for hard walls to form about the fistula, and more time for them to contract to such a degree as to offer great resistance to the urine. When they do, we have what seems a battle between the urethral narrowing and the obstructive efforts on the part of the fistulous opening, and it is quite interesting to observe the successful way in which the peri-urethral bands hold their own even when they are, as often occurs, only a line or two in breadth, for they are often seen to keep the urethra tightly closed while forcing the formation of one, two, several new fistulous outlets, which latter have at first soft and yielding walls which offer a minimum of resistance.

There is one other phenomenon in this series of changes which is of interest as well as of practical value, and that is the behavior of the anterior portion of the stricturing band, off which all urine is kept. For a long time I have been in the habit of calling the attention of my assistants and others about me to the fact that, when I was operating upon a case of this kind, where for a long time all the urine had apparently passed by fistulous openings, and none by the urethra, we always had to deal, at the critical moment, with an extremely narrow stricturing ring, in many cases quite resembling a thin diaphragm, through which there was still a very small hole to pass a probe. And this was not all. In some of the cases it was apparent, on inserting the Avery's threads to hold asunder the parts, that there was a pouch, or rather an unnaturally dilatable condition of the urethral walls in front of the diaphragm-like coarctation, as well as behind it, but presenting a different appearance upon inspection after the section was made; for the walls of the anterior pouch were pale pink and healthy-looking, while those of the one posterior to the coarctation were soft, friable, covered by necrotic epithelium, and with veins deeply injected. For a long time this state of things puzzled me, as did the fact that I never found a broad stricture-band encircling and compressing the urethra in these cases. I am now persuaded that this condition is the result of the atrophy and disappearance of the anterior portion of what was once a broader stricture-band, without

reproduction of the normal elements, which had undergone shrinkage from pressure of the previously existing connective-tissue elements. Such a state of things might be expected to come about after the withdrawal of the outer supports of the mucous membrane. And I am all the more persuaded that this is the real explanation by watching the changes taking place in the walls of the fistulæ after the urine was made to pass by the natural way or was withdrawn through a catheter; for here we observe the atrophy and disappearance of the same kind of tissue as the stricturing bands, in a manner quite striking. This, in my observation, goes on quite as rapidly and satisfactorily in the cases of fistulæ that are not as in those that are attacked by the knife. The essential thing seems to be the removal of urine from contact.

Mr. Harrison ("Lettsomian Lectures," 1888, p. 14), in speaking of the intention manifested by the tissue development in stricture, says: "In this strengthening of the urethra we recognize, in the first instance, a conservative action; eventually, however, as in other compensating processes, certain inconveniences follow which constitute, as it were, an independent disease." With that part of this view which regards the development of the stricture-bands in support of the urine-tight state of the urethra as conservative I agree; but if I correctly interpret the author to mean that the effort of the process to close up the urethra is but an accidental and vicious one, or one not equally conservative, I must respectfully dissent from that view. To my thinking, the intent to close up the canal at the point of narrowing is manifest from the very beginning, and it would seem to be quite as conservative as any other of these phenomena. For, if the mucous membrane is no longer functionally efficient, it is manifest very soon that, develop and condense as it may, neither is the stricturing neoplasm capable of preventing entirely urinary resorption. If the effort was solely to strengthen the urethra against the urine, the tissue development might just as well be eccentric, whereas it is in an opposite direction, and with an evident intention. In a word, Nature seems early to have realized that the affected mucous membrane is best protected and cared for by being squeezed together tightly enough to keep urine off it altogether.

The rate at which this urethral narrowing goes on is another feature of practical importance as well as of theoretical interest. We all know that the stricture-building process is a distinctly chronic one, requiring months and even years in most cases to extensively interfere with the functions of the parts, and we can not help regarding this slow rate of progress as an important item of conservatism so far, at least, as the life of the affected individual is concerned, for it affords opportunity for the development of those changes in the tissues immediately posterior to the lesion in such a way as least to jeopardize other organs which may be necessarily affected.

It is only by this slow progress that the bladder, ureters, and kidneys are protected, even in a small way, by being allowed time for adjustment to the new order of things. One sees a different and far more dangerous condition arising in those rapidly developing cases of traumatic lesion where the passages may be shut up in a few hours by the mere swelling and distortion from laceration.

It is universally conceded that the pathological process concerned in the development of the stricture-bands is one of the forms of inflammation—contracting, non-suppurative inflammation. The modern pathology requires us to believe that we must have a factor at work which is in some degree commensurate with the duration of the disease, or, in other words, that the inflammation will exist only so long as the causative factor is present and efficient. Remove this ætiological factor and the process ceases at once, and at least and in many cases there is a retrogressive action in the newly formed elements which may and often does go on until they have totally disappeared. One of the methods of dealing with the purely obstructive part of the disease is to change the constructive into a suppurative inflammation. Atrophic, inflammatory dilatation of a stricture does this and nothing more. May we not accomplish the same retrogressive change by simply removing the factor concerned in the development of the newly formed tissues? I believe we can, not perhaps as rapidly as is required in many cases; but one is hardly permitted to doubt that it can be done when he watches the rapid and complete retrogression which takes place in the hardened walls of an old urinary fistula after he has removed the urine from contact. If we could, in the case of inflammatory stricture where we still have mucous membrane left, restore that membrane to its normal condition in every respect, we would, I think, be able to cure the disease permanently without either dilatation, cutting, or splitting, and the cure or restoration of the normal state of the parts would in every respect resemble the changes we observe in the fistulous walls after diverting the urinary stream; and it would be just as permanent or radical. If, however, we should fail in this because of the development of a cicatricial mass incapable of absorption, we would at least arrest the process of stricture-building. In the case of cicatricial stricture we could accomplish a like result by covering the altered portion of the urethral wall with healthy normal mucous membrane (transplantation, urethroplasty), though not here to the same extent might the caliber of the canal be raised. We would rather in this instance bring about a state of things closely resembling those non-progressive and far less dangerous states known to us as congenital narrowings. We would then have malformation, but not stricture in the sense here intended.

I conclude, then:

1. That the essential ætiological factor in urethral stricture is a modi-

fication of the mucous membrane in such a way as to permit of the leakage of urine or of some of its constituents.

2. That the surgical indications thus afforded are (*a*) to restore the mucous membrane to its normal condition, if that be possible, as it often is in inflammatory stricture; or, (*b*) failing in all efforts to restore the membrane, to remove the urine from contact by providing an artificial channel for its escape.