

sacrouterine ligaments. Occasionally the cervix is held forward by shortening of the tissues anterior to the cervix from inflammation or other cause. When this is the case the contraction should be overcome by stretching or incision before an attempt is made to fasten the uterus forward. Generally, however, the tissues are lax all around the cervix.

In these cases, in addition to fastening the fundus forward while the abdomen is open, the cervix should be drawn backward and raised by shortening the sacro-uterine ligaments. This is accomplished by means of a puckering suture. Bovée has probably done more in the development of round ligament shortening by various methods than any one else.

We are interested now only in the shortening when the abdomen is open. With good exposure of the pelvic cavity and the intestines packed out of the way there is not much difficulty in accurately placing a suture that, when tied, will effectually pucker the ligament and lift the cervix upward and backward. As Dr. E. C. Dudley has pointed out, the suture should be passed rather superficially so as to avoid injuring vessels and other structures near the pelvic wall, and the surfaces to be brought together should be roughened so that agglutination will take place and adhesions follow. Both sutures could be placed before either are tied, for when the cervix is drawn back close to the rectum it is difficult to place a suture satisfactorily in this region.

In most of these cases of relaxed sacrouterine ligaments there has been marked laceration of the pelvic floor, the repair of which is indicated as a part of the operative work. Occasionally, however, such a condition of the sacrouterine ligaments is found in patients who have borne no children and have no laceration of the pelvic floor.

In cases with laceration, repair of the pelvic floor for the purpose of holding the cervix well back in the pelvis sometimes proves insufficient, the constant pressure of the unsupported cervix causing gradual stretching of the repaired floor and finally descent of the cervix to its old position and a return of the retrodisplacement. In these cases, shortening of the sacrouterine ligaments while the abdomen is open, in addition to fastening the fundus forward, adds much to the completeness and permanency of the replacement and may make the difference between success and failure in the final result.

WHEN THERE IS CONTRACTION OF THE POSTERIOR PART OF THE BROAD LIGAMENT.

There are certain cases of retrodisplacement with extensive chronic inflammation in which, after removing a disorganized tube and breaking up the adhesions posterior to the uterus and broad ligaments, there is still so much inflammatory infiltration limiting the mobility of the uterus that it can not be brought well forward without danger of injury to important structures or such resulting tension as will cause distress. The question is: What is the best procedure in such a case? Shall we persist in trying to loosen the uterus so that it will come far forward, at the risk of an injury to the large vessels along the pelvic brim which are firmly imbedded in the infiltrated tissue, shall we fasten the fundus forward to the abdominal wall under strong tension, or shall we leave it in its perpendicular or slightly backward position?

I think that, as a rule, the last-mentioned plan is the best in these exceptional cases. The principal symptoms are due to the inflammatory mass, which has been re-

moved. Usually the uterus can easily be brought forward to a position midway between the normal position and decided retrodisplacement, and this position, when maintained, practically never in itself causes troublesome symptoms.

Whether the uterus will stay in this elevated position or return to its former one depends on whether the tissues are pliable and the uterus movable. If they are, the uterus will not stay in this midposition; but when the tissues are fixed by infiltration, this with the scar tissue that results from the removal of the diseased tube or tubes so fixes the uterus that it is likely to be held in that position indefinitely.

To favor the maintenance of the uterus in this position, care should be taken, before closing the abdomen, to see that the intestines extend into the posterior part of the pelvis and that the omentum comes down over them and is, when long enough, tucked in back of the uterus. The intestines and omentum back of the uterus assist in holding the latter forward during the healing process and also tend to prevent adhesions which extend directly from the uterus to the posterior pelvic wall, the contraction of which would draw the uterus further back.

In certain of these cases some of the structures alongside of the uterus on one or both sides may be fastened forward, thus helping to maintain the uterus part way forward both during the healing process and afterward. In one especially troublesome case of this kind I was able to secure a satisfactory result by suturing the upper anterior margin of the broad ligament to the anterior abdominal wall for a considerable distance inward from the pelvic margin on each side.

In the after-treatment of the cases of this class, it is well to have the patient lie on her side and well forward, partially on the abdomen, most of the time, and not much on her back. If the cervix comes low, it is well also to have the patient wear a pessary that will hold it well back in the pelvis during the healing process, provided the instrument causes no severe pain.

TREATMENT OF TRAUMATIC GANGRENE.

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In this brief paper it is my intention again to call attention to a procedure which in my experience has proved of great utility in the management of these most troublesome and often fatal cases. I shall not take up the pathology of traumatic gangrene, but shall confine myself to a consideration of its treatment. By the term traumatic gangrene as herein used is to be understood that variety of gangrene following severe crushing injuries of the extremities complicated by virulent and often rapidly fatal sepsis.

On referring to the literature we find all authorities agree as to the extremely high mortality of this affection, Cheyne and Burghard giving it as high as 95 per cent. All authorities likewise agree that the only treatment of cases of this class is early amputation above the gangrenous area.

How often are we able to determine accurately the limits of the disease, particularly in those cases of rapidly spreading moist gangrene? Too often we are disappointed by the continuation of the process after amputation and are compelled to reamputate much higher, in spite of which the patient often dies.

If the gangrenous process does not involve the entire limb, the flaps frequently slough, necessitating another operation and the sacrifice of what is too often most valuable tissue so far as the future usefulness of the part is concerned. In other cases I have been convinced by subsequent events that in my effort to get well above the disease I have sacrificed tissue which might have been saved. In other words, I have amputated at a point higher than was necessary.

In casting about for the probable cause of the unsatisfactory course pursued by many of these cases, I became convinced that it was the attempt to form flaps from tissue, the vitality of which is always seriously in question, and the further impairment of that vitality by two procedures necessary to this method of operation: first, the dissection of the flaps; second, the introduction of sutures.

The dissection of the soft parts into suitable flaps necessarily interferes with their blood supply, which interference in the class of cases under consideration is often badly borne and frequently resented by sloughing of the flaps or the rapid extension of the gangrenous process up the limb. The introduction of sutures at this time has two distinct disadvantages: first, the retention within the wound of infectious elements which may have traveled up between the muscle planes or along tendon sheaths to a point higher than we may believe probable; second, the further interference with flap nutrition by the tension of the suture, for tension can not be entirely avoided, no matter how carefully the sutures may be placed. Believing the statements above made to be true, and that the bearing of these facts on the results secured in these cases was most important, in 1902 I described a method of amputation which had, in my hands, greatly reduced the mortality of traumatic gangrene. Since then I have had several opportunities to demonstrate further the value of the method.

The procedure is as follows: Being confronted with a case of traumatic gangrene of an extremity, estimate as exactly as possible the line between the diseased and healthy soft parts. Under anesthesia make a most careful and complete disinfection and cleansing of the skin, puncturing all bullæ and remove all discharges, envelop the gangrenous area in a sterile towel up to the line selected and then, at this point, make a circular amputation, cutting through soft tissues and bone at the same level. Ligate carefully all bleeding points, including none of the perivascular tissue in the bite of either the forceps or the ligature. Leave the wound open, not introducing a single suture, and apply moist dressings of gauze saturated with salt solution, these dressings to be changed from two to four times in twenty-four hours, as the circumstances of the particular case demand.

After seven to ten days, if the wound is perfectly clean and the condition of the patient favorable, the classic circular amputation may be made by dissecting up the flap already outlined and sawing the bone at the proper level. If for any reason the circular method may seem undesirable, any other procedure may be substituted, but in the class of cases under discussion a typical circular amputation will be found entirely satisfactory.

It is unnecessary to report in detail the cases treated by this method, but it may be stated that since adopting it in 1900 I have employed it nine times: twice in amputations in the subtrochanteric area, twice in the middle third of the thigh, four times in the upper third of the leg, and once in the forearm. The results were eight recoveries and one death.

The two cases of subtrochanteric amputation will be briefly described, as they presented all the difficulties common to the most severe and fatal types of traumatic gangrene.

CASE 1.—A boy, aged 12, was run over by a disc harrow and the left leg frightfully mangled to condyles of femur. Brought to hospital by Dr. Evans of Emerson, Neb., on the third day after the accident, at which time gangrene had reached the lower third of the thigh and presented the usual clinical features of traumatic gangrene. The patient was delirious, having a temperature of 104 F. and a pulse of 136. Amputation as above described was made immediately at a point just below the great trochanter. Pulse and temperature reached normal within forty-eight hours, delirium had disappeared and the wound appeared clean. On the tenth day after the injury tetanus appeared and for four days death was hourly expected. Following the usual treatment, complete recovery from tetanus occurred, and two weeks later the flaps were dissected up, the head of the bone disarticulated and good union was secured. Since this time the patient has been well.

CASE 2.—J. M., man, aged 40, fell under a slowly moving train and sustained a severe crushing injury of the right foot and ankle. He was brought to the hospital thirty-six hours later by Dr. Langley of Oacoma, S. Dak. At this time gangrene had extended to the malleoli and the patient's condition was good. Not realizing the gravity of the case and thinking that, owing to almost total absence of constitutional symptoms the case was one of ordinary gangrene due to thrombosis, I made a classical amputation just below the knee. Twenty-four hours later pulse was 110 and temperature 102 F. The flaps looked suspicious. Thirty-six hours later the pulse was 158, temperature 105.5 F., and the patient in a low muttering delirium. There was gangrene of the flaps and discoloration to the middle third of the thigh. Realizing that prompt separation from the gangrenous and infected leg gave the only chance of life, I decided to reamputate.

The patient's condition would not permit use of general anesthesia and, owing to the condition of the limb, local anesthesia was considered inadvisable. With no anesthesia whatever amputation through bone and soft tissues as described was rapidly made slightly below trochanter. Owing to advanced sepsis the senses of the patient were so obtunded that he gave no evidence of pain except at the moment the knife severed the sciatic nerve. Despite the fact that we expected this man to die on the table or soon after, he began to manifest improvement a few hours after the operation. In twenty-four hours his pulse was 120 and temperature 101 F. Forty-eight hours later pulse and temperature became normal and so remained. Twelve days later, the wound being clean, flaps were dissected up and the bone sawed through at the trochanter and the wound closed. Primary union was secured, stitches were removed on the eighth day and the patient was up and about the ward on crutches. Was told one day that he might leave the next morning for home and that night died suddenly of pulmonary embolism.

The stump was absolutely clear and union perfect. This was the most desperately sick patient I have ever seen recover from gangrene and one on whom I could not think of operating by any other method. His tragic death after apparently complete recovery was particularly unfortunate and should not be charged against this particular plan of operation, as it might have followed any operation.

As my results by the old method can not be compared with those secured by this, I have ventured to present the operation for consideration. Its advantages may be summarized as follows:

1. By it we may speedily and with the minimum of shock remove from an enfeebled and exhausted patient the source of infection, namely, the gangrenous tissue.
2. We may frequently make the amputation at a lower point than would be possible by the old method, because

soft tissues whose vascular connections are left undisturbed are less apt to slough.

3. The freest possible drainage is provided for a sufficiently long time.

4. If the disease stops at the line first chosen, no unnecessary sacrifice of tissue has been made, as the flaps have been defined at the lowest limit of safety, and the bone would necessarily have to be sawn at the same point as at the second operation.

5. If the limits of the disease have not been placed sufficiently high, the same procedure may be repeated with less danger to the patient than a typical reamputation.

6. Patients too weak and prostrated by sepsis to withstand a typical amputation may endure this much shorter procedure and be built up during the interval before the second operation is necessary.

IS EYESTRAIN EVER AN ETIOLOGIC FACTOR IN EPILEPSY?*

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In reporting the following four cases of epilepsy in which the active causative agent appears to have been eyestrain and in which correction of the refraction errors relieved the greater affection, I am actuated by a desire to aid in promoting the educational work that other ophthalmologists have been doing in this direction, and possibly to benefit some other sufferers, rather than to enter into the dispute regarding the relationship of eyestrain to epilepsy. My own observations convince me that an epileptic seizure may be induced by reflex action from an ocular disease. There may be some physicians who do not want to be convinced of this, there are certainly some who deny the possibility of the eyes playing any important part in connection with epilepsy, and the great mass of the profession has probably never given any consideration to the matter and would have no answer prepared for the question expressed in the title of this paper.

The primary reason for stating the question is that it may serve as the basis for a dispassionate consideration of the subject, for it is an important matter that the proper answer should be determined and, if it be in the affirmative, as many of us believe it will be, it is equally important that the general profession should be so informed and should apply that knowledge for the prevention or alleviation of human suffering. It has been estimated that there is one epileptic in every 500 inhabitants in this country; applying that to the city of Baltimore, let us say, would mean that we have nearly 1,200 victims of epilepsy; or, furthermore, about 2,000 in the state of Maryland. Surely, it is worth while to give serious consideration to any disease that afflicts so many citizens in a community. Another most excellent reason for investigating any agency that might, even remotely, contribute as a causative factor is found in the fact that, when once it is firmly established, this disease has a bad prognosis. The percentage of patients cured by any or all forms of treatment is extremely small; the reports of some institutions in which epileptics are treated would seem to indicate that less than 1 per cent.

are ever restored to health. The saving of an individual, then, from the development of epilepsy or the cure of one in whom the disease had started is like snatching a brand from the burning! If only one case in 100 is found to be due to eye disease and curable by the proper treatment of that organ, it becomes possible to double the present percentage of cures.

Whenever one reports the cure of a series of cases of some disease which is admitted to have a grave prognosis and in which the percentage of cures by any treatment is exceedingly small, he may expect skepticism on the part of his hearers and perhaps some doubt as to his diagnoses. While the diagnosis in my cases was vouched for by others, I have taken pleasure in fortifying my knowledge of the disease by a recent careful reading of the latest authoritative publication on the subject, i. e., *Epilepsy and Its Treatment*, by William P. Spratling, superintendent of the Craig Colony for Epileptics, published in 1904.

I may be pardoned for quoting extensively from this book, for Dr. Spratling does not advocate the idea that eyestrain bears any relation to epilepsy, and probably did not dream that his book would ever be used to support such a theory. I believe, however, that he submits the best of evidence in support of that theory, and that his opposition to the theory in other writings, as well as in his book, is not only unjustified by, but is in defiance of his own observations.

What is the etiology and pathology of epilepsy? Regarding its etiology, Dr. Spratling says (page 58):

We may assemble the causes of all epilepsies primarily under two heads: Predisposing and Exciting. Under the former will be studied the comparatively few factors that prepare the individual to acquire the disease, but which may not actually produce it; under the latter, as many as possible of the numerous factors that actually produce epilepsy with or without the aid of influences that predispose to it.

In the majority of cases predisposing and exciting causes play a part and are complementary to each other. This well-known fact we may illustrate in this way: Given an individual in whom epilepsy may be induced under the influence of some irritation of which the climax—i. e., the point at which it causes the seizure to appear—is represented by 100. If there are already present 60 of these points from, we will say, a predisposition due to heredity, there will remain but 40 points to be supplied by some exciting cause to bring the disease to light; this exciting cause being, perhaps, one of the specific fevers, some kind of toxemia, an emotional shock; an acute attack of indigestion, trauma, some reflex irritation, or numerous other causes which will be fully described later on. In other words, epilepsy, etiologically considered, is often a symptom-complex dependent in the great majority of cases on two variables; either a maximum of inherited influence combined with a minimum of exciting cause, or a minimum of inherited influence combined with a maximum of exciting cause.

No one will question the importance of an hereditary predisposition in many cases, especially in those cases of direct hereditary transmission of epilepsy or in which the child acquired its predisposition from a parent who had some other nervous disease, such as alcoholism or insanity. The exciting causes are the ones bearing particularly on our question, and I would call attention to the list mentioned in the above statement regarding etiology. It will be noticed that with the exception of toxemia and trauma all of the exciting causes named must act reflexly. Just what that process of reflex action may be is not positively known, but it would seem most plausible to assume that, through the abnormal function of certain organs, toxins may be set free which will seriously injure the delicate structures of the cerebral cor-

* Read at the meeting of the Section on Clinical Medicine and Surgery, Baltimore City Medical Society, Dec. 7, 1906.