PROLAPSE OF THE UTERUS IN NULLIPAROUS WOMEN.

BY

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Two cases of prolapsus uteri in women who have borne no children have come under my observation and serve as a basis for the consideration of prolapse of the uterus in nulliparous women. It is the most common of all lesions in the pelvis of multiparous women and one of the rarest of lesions in the nulliparous. Weinberg finds prolapse of the uterus in the new-born and in nulliparous women to constitute 3.45 per cent. of all cases of prolapsus, while Scanzoni would place the percentage at 13.15.

Virginal prolapse of the uterus occurs with greatest frequency in the new-born and in most instances there are associated congenital defects; notably spina bifida. Schaeffer reports the case of a fetus of the second half of intrauterine development, in which the uterus and vaginal walls were prolapsed and there was a well-marked spina bifida.

Procidentia in the new-born is rarely in evidence at the time of birth, but develops, as a rule, in the following week. Such infants rarely mature; death resulting from associated lesions rather than from the procidentia. The frequency with which spina bifida is associated with procidentia in the new-born is approximately 86 per cent. Nebesky records twenty cases, seventeen of which were associated with spina bifida and von Radwauski fourteen cases with spina bifida in twelve. If, however, we were to include the cases of spina bifida occulta, a lesion commonly overlooked, the percentage would be much higher. It is of peculiar interest to note the observations of Ebeler who rayed twenty-eight multiparous women with prolapsus uteri and found occult spina bifida in twenty-three (82.14 per cent.). Later twenty-eight multiparous women without prolapsus were rayed and three of this number revealed occult spina bifida. Ebeler argues, with much reason, that neural disturbances with secondary muscular insufficiency of the pelvic floor is an important

factor in the development of procidentia in multiparæ as well as in nulliparæ.

Associated with spina bifida and prolapsus uteri in the new-born are numerous congenital defects; *i.e.*, hydrocephalus, ankle clonus, club feet, scoliosis, kyphosis, flat or perpendicular pelvis, infantile uterus, flat and shallow vagina, elongated cervix, prolapse of rectum, inguinal hernia, paresis of perineal muscles and disturbed sensation in the lower extremities.

The relation of spina bifida to prolapsus uteri has been variously interpreted. Krause, together with many who have expressed an opinion, advances the theory of faulty innervation of the supporting structures in general; Ertzbischoff, to faulty innervation of the uterine ligaments alone; Haussen, to disturbances in the central nervous system, while Halban and Burger and the majority of writers on the subject account for the prolapsus on the theory of faulty innervation of the muscles of the pelvic floor. Halban and Burger noted atrophic changes in the levator ani muscles, which they ascribe to faulty innervation and Heil found hypoplasia of the fat, fascia, muscles and ligaments of the pelvis which he also ascribed to faulty innervation. It is to be borne in mind, however, that spina bifida is associated with prolapsus uteri in but a small per cent. of cases, hence the inference that there are contributing factors other than faulty innervation, such as prolonged physical exertion, malnutrition, general visceroptosis, congenital widening of the hiatus genitalis, congenital deepening of the rectovaginal pouch and oversized pelvis.

It is of interest to note that as far back as 1735 Munro reported to the Edinburgh Obstetrical Society, a case of procidentia in a girl three years of age. This case bridges over the gap between the congenital type found in the new-born and the acquired type of later years.

In several of the reported cases the procidentia occurred about the time of puberty, and in these cases it is recorded that the girls were poorly nourished; some with tuberculosis of the lungs associated with persistent coughing, others who were compelled to do hard labor.

A suggestion of the rarity of the lesion in the nulliparous women is found in the excellent contribution of Kepler who collected seventy cases in the literature up to 1911. To this number he added one of his own and eighty from personal correspondence, making in all 151 cases of procidentia uteri in nulliparous women. He classifies these cases as follows:

1. Cases due to congenital defects which occur in the new-born or at the time of puberty.

2. Cases not due to congenital defects occurring later in life.

Freund finds prolapsus occurring in the more advanced ages of nulliparous women begins as a hernia of the pouch of Douglas. As the pouch deepens the uterus and anterior vaginal wall descend. As a rule, this condition begins in early youth and develops slowly. Associated with the congenital deepening of the pouch of Douglas, Freund finds an abnormal inclination of the pelvis.

In my judgment there is an element of infantilism in most if not all the cases of procidentia in nulliparous women. The fact that these women are sterile is highly suggestive. In support of the theory of infantilism as an underlying factor in the development of procidentia, I have two cases to record.

CASE I.—Miss C., seen in the Home for the Feebleminded, Glenwood, Iowa. She was about thirty-five years of age, had acquired procidentia in early girlhood. Her sister was also feebleminded. There was lack of general development. All the pelvic supporting structures were atrophied and showed great relaxation. The uterus and vaginal walls were completely prolapsed. No observations were made as to the condition of the spine.

CASE II.—Mrs. L., fifty-five years of age, married thirty-one years, no children, menstrual periods began at fifteen years of age, always irregular (three to five weeks intervals), duration of flow five days, no pain. The menopause was established at forty-one years of age. For the past five years she suffered from frequent urination, getting up three or five times at night. She first noticed the protruding cervix three years ago at the age of forty-eight. The prolapsus gradually increased, but caused no great inconvenience until one year ago she experienced great difficulty in emptying the bladder. The prolapse was complete six months ago and remained so to the time of operation. The patient accounts for the "falling of the womb," by the years of hard work performed on the ranch where she daily carried heavy loads. She was exceptionally strong, nearly six feet in height and weighed about 180 pounds.

On examination the uterus was found completely prolapsed, the vaginal walls everted, and the pelvic floor greatly relaxed with pelvic floor muscles atrophied. No *x*-ray was taken to determine the possible existence of an occult spina bifida. The operation consisted in a vaginal hysterectomy followed by a colpoperineorrhaphy. Results satisfactory.

Other evidences of the effect of strain upon the uterine supports in the development of prolapsus, are the cases of Webster in which a wagon wheel passed over the abdomen and was shortly followed by prolapsus; the case of Green which followed the lifting of a piano and that of Lihotzky produced by strain at the age of seventy-two.

Poor nutrition in the early years of development has been cited as a predisposing factor. In two recorded cases tuberculosis of the lungs with accompanying persistent coughing brought on the prolapsus. In all these cases it is difficult to account for prolapsus in the absence of a pre-existing weakening of the uterine supports.

The relation of mental defects to prolapsus uteri is forcibly illustrated by the observations of Kepler who collected eighty cases of procidentia in nulliparous women and of this number thirty-eight were mentally defective. In this group were dementia precox, imbecility, idiocy, chronic mania, hysterical insanity, cretinism and nervousness of high degree. It has long been recognized that defective mental and physical developments go hand in hand and the casual relation of mental defects to prolapsus uteri is readily conceived.

Halban, in reporting the case of a nulliparous woman, twenty-seven years of age, whose uterus prolapsed after lifting a heavy load, expressed the opinion that the underlying factor in the development of the prolapsus was a congenital underdevelopment of the muscles of the pelvic floor. He referred to a dissection of a similar case in which Tandler found marked atrophy of the muscular supports. Martin observes that the levator ani muscle in prolapsus is often well developed. Halban admits this as an exceptional occurrence, but says that Martin overlooks the fact that the hiatus genitalis in these cases is relaxed, thereby implying that the inner portion of the levator ani muscle is defective and hence the weakened support to the uterus. Halban and Tandler do not believe with Martin that the weakening of the pelvic connective tissue and elastic fibers accounts for the prolapsus. They regard these structures as mere supports for the blood-vessels which can in no measure resist the force of intra-abdominal pressure. In support of their theory that the intra-abdominal pressure is resisted and the uterus supported by muscular structures alone, Halban and Tandler cite the cases of prolapsus in the new-born in which there is lack of muscle tone from defective innervation. Schultze is probably more near the truth when he contends that atrophy of the ligaments, connective tissue and muscles of the pelvis, all contribute to the production of prolapse of the uterus. Add to this the suggestion of Ziegenspeck that intraabdominal pressure plays the chief rôle in the development of prolapsus uteri and we have, in my judgment, the most rational solution of the problem.

BIBLIOGRAPHY.

Ballantyne and Thomson. AMER. JOUR. OBST., vol. XXXV, No. 2.

Boehm. Inaug. Diss. Strassburg, 1911; Ref. *Zent. f. Gyn.*, 1911, Nr. 51.

Bonney. *Jour. Obst. and Gyn. Brit. Emp.*, xxv, No. 6.
Burger. *Arch. f. Gyn.*, Bd. 73, H. 2.
Bumm. Ref. *Zeit. f. Geb. u. Gyn.*, Bd. lxvi, H. 2, p. 468.
Ebeler and Duncker. *Zeitsch. f. Geb. u. Gyn.*, Bd. lxxvii, H. 1.
Graf. *Monatsch. f. Geb. u. Gyn.*, xxxiv, H. 6.
Halban. *Zent. f. Gyn.*, 1913, Nr. 23, p. 858.
Halban and Tandler. Anatomie u. Aetiologie d. Genital-prolapse des Weibes, 1907.
Heidenhain. *Monatsch. f. Geb. u. Gyn.*, xxxiii, H. 5.
Heil. *Arch. f. Gyn.*, Bd. 48, p. 155.
Kepler. Amer. Jour. Obst., vol. lxiii, No. 6.
Krause. *Zent. f. Gyn.*, 1897, Nr. 16, p. 422.
Lapeyre. Ref. *Zent. f. Gyn.*, 1908, Nr. 42, p. 1407.
Liebmann. *Zent. f. Gyn.*, 1894, p. 1002.
E. Martin. Ref. *Zeitsch. f. Geb. u. Gyn.*, Bd. lxvi, H. 2, p. 460.
Mayerhofer. Ref. *Zent. f. Gyn.*, 1915, Nr. 11, p. 165.
E. Martin. Der Haftapparat d. Weibl. Genitalien, Tl. I–II, Berlin, 1911.
Nebesky. *Arch. f. Gyn.*, Bd. 87, H. 3, p. 497.
Prochownick. *Arch. f. Gyn.*, Bd. 17.
Rosenthal. *Berl. klin. Wochensch.*, 1911, Nr. 25.
Schauta. Lehrb. d. Gesamten Gyn., II–Teil.
Schaeffer. *Arch. f. Gyn.*, Bd. 71, H. I.
Schiffmann and Ekler. *Monatsch. f. Geb. u. Gyn.*, xxxii, H. 3.
Schultz. *Monatsch. f. Geb. u. Gyn.*, xxxviii, H. 6.
Sippel. *Monatsch. f. Geb. u. Gyn.*, xxxiii, H. 5.
Sutter. *Monatsch. f. Geb. u. Gyn.*, xxiv, H. 4.
Veit. Handb. der Gynäkologie, Bd. i.

DISCUSSION.

Dr. Sigmar Stark, Cincinnati, Ohio.—I believe that all of us are familiar with the coexistence of pelvic prolapse and spina bifida, but that this state is a concomitant of occult spina bifida is extremely interesting. I am unfamiliar with this feature of the etiology, but I have given the subject considerable study from an anatomical standpoint. Three years ago I spent a few weeks in Berlin with Liepmann doing dissections on the pelvis in order to familiarize myself with the work done by Tandler and Halban, and came to the same conclusions that Martin did, namely, that damage to the muscular fibers of the pelvis had absolutely nothing to do with pelvic prolapse; that pelvic prolapse was solely due to damage to the connective tissue.

The coexistence of pelvic prolapse in cases of spina bifida can readily be explained on the same basis, namely, you have a defective development of the pelvic connective tissue due to nerve influence just as is the case with the muscular tissue of the pelvis. In the case of pelvic prolapse of the nullipara unassociated with manifest spina bifida, you can readily establish the deficiency of connective tissue

throughout the pelvis. The essayist called attention to the insufficient development, the infantilism of the pelvic organs that is present. You have a small uterus, but in particular do you have a shallow vagina, and although a fairly free mobility in an upward and downward direction exists, there is an inhibition of mobility in an anterior and posterior direction, all of which is due to a lack of connective tissue.

To go back again to the subject of my observations as opposed to the findings of Tandler and Halban, I wish to say that close study of their dissection reports and illustrations fails to reveal the evidence of a laceration transversely through the fibers of the levator ani. The pathological condition of the muscles is the result of atrophy and fatty degeneration due to overstretching, hematoma or thrombosis.

What I want to say in particular is, that the whole thing to my mind is a connective tissue disturbance, as connective tissue damage is the result of the trauma of labor, so in congenital prolapse there is a deficiency of connective tissue present in consequence of faulty innervation.

Dr. A. J. Rongy, New York City.—I wish to add three more cases of prolapse of the uterus in nulliparous women, two upon whom I have operated, and one that I am going to operate on, on my return to New York. The operation I did in these two women was the usual interposition operation. These women are highly neurasthenic, and if you take out the uterus they stop menstruating and become more nervous.

Dr. Stark mentioned the fact that these women usually have shallow vaginas and that the cervix is inserted very low. Most likely these women start out with a congenital retroversion of the uterus, and the intraabdominal pressure from above causes the uterus to slide down into the vagina and prolapse ensues.

Dr. Charles L. Bonifield, Cincinnati, Ohio.—I wish to refer to one case I saw some years ago which bears out Dr. Findley's contention as to the etiology of these cases. This patient was a girl, fifteen years of age, an orphan, who lived with people who worked her very hard. They took her to market and she had to carry home on her arm a market basket overladen with produce, too heavy for her to carry. In order to get rid of this burden, as soon as she got into the house, she squatted on the floor, and just as she did so the uterus and vagina came out at the vulvar orifice.

Dr. Rongy is quite right in insisting that one great predisposition to these prolapses is congenital; that is, the uterus is either retroverted or retroflexed, because we all understand that as long as the uterus is of normal size and normally anteverted the increased intraabdominal pressure will only force the fundus forward; it cannot force it down. I believe that the connective tissue has some power of support. We all know that in regard to the kidney. Every one of us has seen patients exceedingly thin who have had trouble with movable kidney, and after they put on a lot of fat the kidney would move less and less, so that connective tissue must have something to do with it.

With what Dr. Stark has said I agree in the main, but I cannot agree with him that the muscle is not lacerated sometimes.

DR. STARK.—I said the levator ani.

DR. EDWARD J. ILL, Newark, New Jersey.—Dr. T. Gallaird Thomas said in his day that prolapse never occurs in a nulliparous woman. For a great many years I thought so too. The vast majority of cases of prolapse in nulliparæ are elongations of the cervix with secondary dragging down of the tissues and a secondary relaxation of the pelvic outlet. These cases do not present the condition of retroflexed uterus that Dr. Bonifield spoke of. They have an anteverted or flexed uterus and an amputation of the cervix invariably restores these patients. What shortening there may be in the vagina is atrophic, due to downward pressure. I do not think the line of demarcation has been clearly drawn between prolapse and infraelongation of the cervix. Because a cervix appears outside of the vulva does not constitute a prolapse of the uterus.

DR. HERMAN E. HAYD, Buffalo.—This paper has been very interesting and instructive to me. While I consider myself quite familiar with surgical literature, yet this is the first time my attention has been drawn to the association of spina bifida and adult uterine prolapse. Moreover, it is the first time my attention has been drawn to the use of the *x*-ray to find the incomplete spinal development. That point is exceedingly interesting, and I am obliged to Dr. Findley for bringing the matter before us. It seems to me, this question ought to be thoroughly discussed.

So far as the frequency of this condition in the nulliparous is concerned, the essayist has looked over the literature and records 143 cases. Gentlemen, there are thousands of these cases, but we do not report them. There is not one of us who has not seen one or two cases of procidentia in the virgin, and they are usually supraelongations of the cervix. I had one of these cases referred to me by the late Dr. Schroeder. The patient was fifteen years of age who, with her friend, went into the country, and they were playing with two pails of water, one in each hand. They jumped from a rail fence, and each one tried to beat the other in the jump and spill as little water as possible. One of these young girls came to me with complete procidentia; the cervix was not simply elongated an inch and a half, but really the whole body of the uterus came out in the prolapse. I think the condition is very much more common than the essayist records.

DR. O. H. ELBRECHT, St. Louis, Mo.—I am very glad Dr. Findley presented this paper for it seems to me we are getting closer and closer to the cause of this condition by the sort of studies he has detailed here.

There are three classes into which uterine procidentia can be divided.

First the congenital. We have all seen congenital defects that were responsible for uterine procidentia, and I certainly believe spina bifida can be put in that class just as much so as the total absence of the round ligaments or the absence of one of the broad

ligaments, etc. The first case of prolapse of this class I ever saw was in a woman eighteen years of age who had a normal delivery without laceration and within two months following it a complete polapse. At operation I found a complete congenital absence of the round ligament and broad ligament, tube and ovary on one side. Spina bifida can come under the same classification as a causative factor, but it is of functional origin, the result of traction, encroachment on the cord. Accordingly we find club-foot, paralysis of the sphincters, uterine prolapse, etc., and in this way the prolapse is only one symptom (if I may use this term for the sake of clearness) of a group of defects caused by the traction of nerve roots on the cord resulting in improper innervation to the parts. The second class of cases are those resulting from traumatism during childbirth which we all know represent the most common form. The third class can be called idiopathic pelvic hernias. In this class there are no demonstrable congenital defects or evidences of traumatism and the sum and substance of this type is faulty development or atrophy. In this connection I desire to call attention to the case just referred to by Dr. Hayd and Dr. Bonifield. They spoke of the squatting position and lifting causing it. What is the usual history of the onset in inguinal or umbilical hernia. Sudden and forceful lifting or straining. All classes of uterine prolapse are herniations, but the latter class, *i.e.*, idiopathic prolapse, is certainly only one phase of hernias in the general sense arising from the same causative factors at work in the development of inguinal or the umbilical type. Proctologists in analyzing the etiological factors of rectal prolapse must cover the same ground and draw the same conclusion as we do in this field if they go back and investigate the bigger causes at work in the production of this condition.

DR. CHANNING W. BARRETT, Chicago, Illinois.—It is understood that spina bifida has much to do with early prolapse of the pelvic structures. I was glad to see the conservative position taken by Dr. Findley. The question he raises is not, whether this is the only cause of prolapse of the uterus, but he points out that spina bifida causes prolapse in women who have not borne children.

Some questions have been raised in the discussion that are of considerable importance in relation to prolapse of the uterus. As Dr. Elbrecht said, this whole question is one of hernia, and when we stop considering prolapse of the uterus as something entirely apart from other hernias, we will get at the facts more nearly. To say that inguinal hernia is due to elongation of the mesentery allowing the bowel to come out through the opening, is rather absurd. To say that prolapse of the uterus is entirely due to lack of innervation is absurd; to say that it is due to weakness of the connective tissue alone is equally absurd; to say that it is entirely due to weakness of the muscles is no less absurd. It is everything that causes or allows the structures in the abdomen to get outside of the abdomen, and therein prolapses are hernias.

Now we have no doubt but that the muscles may at times be weak, because they are poorly supplied with nerves, and if muscles are

weak they will allow structures to drag down, and the weakness of the muscles of the outlet, inasmuch as there must be an opening, will allow the opening to become larger, but to say that this is the only thing that can cause enlargement of the opening and allow herniation is going farther than most of us will want to go.

Now it is perfectly plain to me that the levator ani muscle does get injured. It is also perfectly plain to me that just as patients can have a congenital weakness of the inguinal region, a congenital weakness of the umbilical region, so they can have a congenital weakness of the vaginal region, and then if added to that a congenital weakening of muscle, we have a weakened or defective innervation, the muscle will do its work poorly. A woman can have a poor pelvic floor, and yet things not come out, but that does not say that the pelvic floor is of no value to hold the structures in. We can have a big pen with a bull in it and gate open, and the bull not come out, but that is not saying the open gate is not a weak point in the enclosure. Now then, prolapse of the organs is a herniation; that herniation will take place by reason of the upper structures being weak, by reason of the pelvic floor being weak, by reason of its being enlarged, by reason of the patient having to lift, and there is no reason on earth why a woman with weakened muscles, although well otherwise, may not have a herniation that is not due to child-birth injury. Of course, most of them come from injury of the supports during childbirth.

Dr. Findley (closing the discussion).—We cannot get away from the fact that spina bifida occurs in a large percentage of cases of prolapsus uteri in the newborn. Not a single man who has written on the subject fails to agree that it is due to faulty innervation of the pelvic supports. I was not particularly interested in what are the pelvic supports, whether muscles, fascia, ligaments, connective tissue or what not, but I wanted to bring out the point that faulty innervation is undoubtedly one of the many factors in the development of procidentia uteri, and to call attention to the very interesting observations of Epler who has taken the trouble to use the *x*-ray in cases of prolapse in nulliparous women. Who else has done it? He has found in twenty-eight cases without prolapsus that there were only three in which spina bifida oculta did exist with the prolapse. In twenty-eight cases with prolapsus, twenty-five of the twenty-eight showed spina bifida occulta. That is rather something for us to think about until we meet again. Defective innervation is only one factor in bringing about prolapsus. When I see a woman shot to pieces, where the uterus does not come down, it is simply raising the question as to faulty innervation in relation to prolapsus as one of many factors. I cannot agree with those who are trying to establish a single factor for the support of the uterus. We must conclude that not one but many factors are involved as Dr. Barrett has said.