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## ORIGINAL LECTURES.

### VERSIONS AND FLEXIONS OF THE UNIMPREGNATED UTERUS.

*A Course of Lectures delivered before the Boerhaavian Society.*

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#### LECTURE I.

##### ETIOLOGY.

CONFUSION, which grows more perplexing as gynecology advances, so surrounds the subject of versions and flexions of the uterus, as conditions demanding correction, that it is nearly impossible to reconcile the assertion on one hand, with the general denial on the other that defines the two schools of uterine pathology. I believe that in these lectures we will better serve the cause of sound teaching by ignoring these differences, and give to the facts—both of pathology and etiology—of the subject a careful analysis, and thus lay out a secure groundwork for the more difficult study of symptoms which will follow. In this way we shall know the relations of these lesions of form and position to what I may call the paradox of symptoms.

A glance at the various etiological groups will show that the causes range through nearly the whole field of morbid gynecological conditions. I divide these causes into five great groups:

- A. Causes existing in the uterus.
- B. Causes existing in pelvic inflammatory conditions.
- C. Causes due to non-inflammatory pelvic conditions.
- D. The condition of near parts as a cause.
- E. Systemic causes.

The first group (A) is of the first importance. To simplify its study, I divide it as follows:

- a. Developmental conditions of the uterus.
- b. Causes due to defective uterine involution.
- c. Causes existing in inflammatory and other conditions of the uterus due to parturition.
- d. Non-parturient inflammatory and other conditions existing as causes.
- e. Causes due to uterine new growths.

a. There can be no doubt that many errors in the period of uterine development tend to the production of versions and flexions of the organ. There are no reasons why these errors should not be assigned to the very earliest stages of uterine growth. But there has developed a thoughtless habit among gynecologists to apply the term "congenital" to those flexions of the uterus which evidently have their origin in early life. When we reflect that in infantile life the uterus consists of a neck with an undeveloped body and fundus attached; that there is no proper separation of the cavities of the neck and rudimentary body, but that it is one cavity with the *arbor vitae* extending throughout; and that the organ continues to maintain this relative pro-

portion between the body and neck until the sixth or eighth year of life, we have good reasons for rejecting the theory of connate flexions. Dr. Hagermann, in an elaborately illustrated monograph, clearly proves the difficulty of such an origin for flexions of the uterus.

In practice, we observe a large number of flexions of the organ in young women, in which the cervix, and usually the whole uterus, are small and exceedingly mobile. To this group, C. Mayer assigns as a cause the atrophy of the uterus in childhood. This is undoubtedly the cause if we substitute for the term atrophy, the term arrested development, for we cannot conceive of atrophy in an organ not yet developed. E. Martin also takes this ground. Virchow has expressed the opinion, from appearances observed in post-mortem sections, that this group of uterine flexions, is often caused by an unequal development of the lateral ligaments of the uterus, thus tending to draw the organ forwards, and toward the shortened side. Numerous authors take the ground of congenital flexions without defining very specifically what they mean. Meissner, Boivin and Dugès, Kiwisch, Flamm, Velpeau, Sommer, Picard, West, Jobert, Cazeaux, and Tilt (Hueter) take this view. Bennet describes an ante flexion of the uterus in which there is found no morbid condition of the cervix, either as to size or position, and which he explains as "the result of the congenital exaggeration of the natural anterior curve of the uterus." Comment upon such an explanation is not necessary. The latter author explains the etiology of latero-version, when not caused by adhesions, by "congenital" causes; excepting, however, a left to right position, which is often normal. In this latter opinion Bennet has the support of Huguier. In this connection, the opinion of such a careful observer as M. Goupil demands some comment. In the examination of the bodies of twenty-five infants at the Maternité, still-born, or dead within the first fortnight, he observed nineteen cases of lateral, right or left, versions and flexions, with double lateroflexions and a retroflexion in five others, leaving only one observation in which the uterus was straight.<sup>1</sup> In all these cases M. Corain proved that the round and ovarian ligaments were shorter on the side of the uterine inclination than on the opposite. This difference in length between the ligaments he explains by the position of the rectum and the distention of the sigmoid flexure with meconium; thus, when right lateral version was observed, the rectum was to the left, and in the opposite displacements to the right. In two of the twenty-five cases in which sinistro-version was noticed with the rectum also to the left, the exception was explained partly by press-

<sup>1</sup> The following note by Bernutz and Goupil is of bibliographic interest: Aran (*Archives générales de médecine*, 1858, 5th série, iif. 321) a parfaitement étudié cette disproportion des deux ligaments ronds et ovariens, dont il rapporte la première étude à Tiedemann, et qui avait été également signalée par M. Pichard. (*Thèse*, Paris, 1852, No. 76, p. 79), op. cit., p. 537.

ure of the sigmoid flexure and partly by the difference in the points of the emergence of the umbilical arteries, which turned the face of the uterus to the side opposite to the version.<sup>1</sup>

There are several reasons why the observations of Goupil and Corain should be given scant inductive value; first, they prove too much. They prove that lateral flexions and versions chiefly, but all forms in the aggregate, are the rule, and the straight, or so-called normal position and form, the exception. Secondly, the uterus during the period named is not acted upon by intrapelvic forces, but is one of the abdominal viscera, and exposed to the movements and pressure of the neighboring parts; and lastly, these authors are applying to a rudimentary organ the same laws of pathology that we are accustomed to apply to the developed uterus. One word will serve to illustrate this. Inequality being the law in the development of the uterine ligaments, it becomes the rare exception to find this lack of uniformity in adult life. The same error of induction exists in the assertion of Kiwisch and Manuel, that meteorism is the cause of congenital flexions.

Confining the etiological movement to the earliest period of uterine evolution, what are the forces which tend to produce pathological varieties of position and form? This question is not difficult to answer. Broadly, all the numerous forces which tend to arrest, retard, or pervert nutrition—more specifically, the diseases of assimilation and nutrition—the diatheses. Unhygienic conditions of food, air, and exercise; all the errors of moral and social environment which tend to sexual precocity, and thus interfere with the consecutive nutrition of all the parts.

There is marked clinical confirmation of this. The feature which is rarely absent in a case of developmental version or flexion is a defect in the size of the uterus, either of the whole organ or of a part of it, and which points to the arrested, or heteroclitic, nutrition of the part; further, these cases are usually associated with incurable sterility. The foregoing render the assumption a reasonable one that this group of flexions has its origin at the period of uterine evolution and not at the foetal period.

One class of developmental flexions often met with requires a few words. In the instance of a short vagina and a uterine cervix more slender and longer than usual, the vaginal portion of the uterus takes the short anterior curve of the lower part of the vagina, or of the perineum. The result is an ante flexion of the neck. Cases have been observed in which, while the vagina seemed of the usual capacity, the uterus was of diminished size, and rested permanently upon the floor of the pelvis. Martin has observed retroversion or flexions caused by defective development of the posterior wall.

b. Versions and flexions of the uterus due to defective involution.

<sup>1</sup> Orth reported to the Berlin Obstetrical Society (March 7, 1875) a case of ante flexion so exceptional that I give it in the original. A child of two years. The broad ligaments are equal; the fundus easily elevatable. As cause of the ante flexion, two abnormal folds appear in the recto-uterine excavation, which arise in the neighborhood of the internal os and coalesce by means of a broad band. They steady the cervix uteri in such a manner as to allow only the fundus to incline forwards when the bladder is emptied.—*Beitrag zur Geburtshilfe u. Gynaek.*, Bd. iv. S. 15.

In tracing the etiological conditions of versions and flexions closely in the parturient period, we must have regard to the fact that anteversion, or flexion, in a recently delivered uterus, is a normal position. Kiwisch alluded to this important fact first, and since then several observers (Hueter, Martin, Scanzoni) have explained the phenomenon. The enlarged uterus being in the cavity of the abdomen, the vertebral column defines the line of deflection of the organ; to fall backwards is not possible. If, under these circumstances, any cause should interfere with the completion of the involution process, the anteversion or flexion might be made permanent (Kiwisch). From this cause, however, retroflexion is more frequently met with than the opposite form of displacement (Martin, Hueter, Säxinger). The reason assigned is, that after delivery the round ligaments are relaxed, and no opposing force exists to the retroversion of the hypertrophied fundus. Klob has called attention to the fact that the contraction of the round ligaments can never wholly raise the organ. Martin seeks a cause of the puerperal tendency to flexions in the fact that the form of the uterus at this period is the inverse of the infantile organ; in the latter the cervix exists with the body at the minimum; in the former the fundus and body are enlarged, while the cervix remains at less than the *ante-partum* size. He states further, that for about nine days after delivery the uterus may be felt anterior to the cervix, and after that interval posterior to it (Beigel). To this arrest of the puerperal involution process, Dr. Henry Bennet attributes the increased size of the uterus in childbearing women, rather than to inflammation. Lesions of the cervix due to parturition seem to take a large share in this cause.

In this relation the effects of abortions must not be overlooked. Retroflexions especially may be so traced (Kiwisch, Arneth, Picard, Virchow, Scanzoni, Säxinger, Hueter). Injury to the connective-tissue stroma (Rokitansky), and the more lingering character of the retrograde metamorphosis (Scanzoni) after this accident, and which Beigel believes to be a prime cause of the great excess of acquired flexions among childbearing women. Among the poorer classes the same care against exertion and improper diet is not exercised after abortion as after a labor at term. The increased volume of the uterus, and the pressure of the abdominal contents in the relaxed state of the supports of the organ, cause the uterus to leave its normal position and become retroflexed. A state of previous flexion undoubtedly tends to abortion in the pregnant woman, and the two causes may become mutually intensive.

Repeated gestation has a cumulative effect upon the amount and character of the subinvolution. Meadows advanced this theory, and cites Oldham, Tilt, Bennet, West, Duncan, and others. Among 666 cases of versions and flexions observed in unimpregnated women by Martin, 395 cases had their origin in faults of evolution. Since in sterile women ante flexion is the rule, and the opposite form found in about forty per cent. excess among childbearing women, it is usual to find that arrested involution is the main factor in the last-named displacement (Heywood, Smith, Bernutz, Goupil).

The tendency of injuries to the cervix during labor to retard or arrest the retrograde movement to a normal size appears to be due, first, to the impeded circulation and irritative hyperæmia arising from a contiguous

focus of inflammation; and secondly, to the extension of inflammation as a result of anatomical continuity of tissue. Owing to the peculiar character of the uterine mural circulation, retarded circulation or hyperæmia may be confined within restricted areas, hence an injury to the posterior part of the cervix might lead to an arrested involution of the posterior uterine wall, with primarily a retroversion degenerating into a retroflexion as a secondary result. Relaxation of the uterus at the puerperal period gives rise to a softened condition of the parenchyma, resulting in flexion and catarrh of the organ. From such a state Klob holds that ante- is more often produced than retroflexion, due to an excess in bulk of the posterior over the anterior wall.

Arrest of the retrograde metamorphosis of the uterus affects the fundus and body of the organ rather than the neck (Hewitt); hence the body, not having support equal to the degree of hypertrophy, bends backwards.

From the softened condition of the parenchyma of the uterus after delivery, it is reasonable to expect that many mechanical causes will operate in the production of versions and flexions. In this condition, prolonged dorsal decubitus has been known to cause retroflexion (Martin, Sommer). Leaving the bed too soon after delivery has been noted by numerous authors as a cause of flexion (Kiwisch, Rigby, Moir, Martin, Säxinger, Hodge, Meissner); also bodily exertion or muscular efforts, such as laboring women are obliged to make, have been regarded as a potent cause of flexion (Lackner, Brünninghauser). From the first cause Scanzoni states that he has found it existing in the proportion of thirty-two in seventy-two cases.

Picard has traced cases of flexion to menstruation coming on prematurely, and causing a suspension of lactation, and hence an arrest of involution. Numerous authors have mentioned the causation of flexions in mothers neglecting to nurse their children (Martin, Picard). The mammary gland when excited by nursing, has undoubtedly a most active influence in promoting contraction of the uterus in the early periods after parturition, and thus acting as a physiological factor in normal involution. Among 54 women suffering from flexions, noted by Scanzoni, there were 196 children, only 57 of whom were nursed. It is among this class of women that we should look for the action of defective nutrition, so much insisted on by Martin as a cause of imperfect involution of the uterus. This imperfect general nutrition may be due to malassimilation of food, to the action of disturbing mental causes, to a depressed vitality, the result of frequent abortions or rapid childbearing, or, as is frequently the case, to a scanty supply of proper food, from either the injudicious orders of physicians and nurses shortly after confinement, or to the poverty of the woman. Martin mentions that many of the women in whom he observed the action of these causes were excessively thin.

Martin defines several varieties of versions and flexions intimately connected with the puerperal condition, and based upon the theory of hypertrophy of limited portions of the uterine wall, the result of arrested involution within the areas of hypertrophy. The inference is not a true one, however, that this limited subinvolution is always the result of a morbid process. Dr. Hagermann, of Hanover, states that involution of the uterus after labor does not take place in all parts

of the organ with like rapidity, but takes more time at the placental site. Assuming that the rate of the retrograde changes is alike for all portions of the body of the organ, the inference is proper that the change is a longer time taking place in the thicker portions of the uterine wall, such as most observers admit to be the condition of the wall of the organ at the point of placental attachment.

Martin describes the following varieties:

1. Anteversion and flexion, in consequence of lengthening of the posterior uterine wall and defective involution of the placental site during the puerperal state and after.

From this cause there are two groups:

a. Recent anteversions and flexions.

β. Chronic anteversions and flexions.

Under this general head, Martin states that he has verified this etiological condition in 28 cases of living parturient women, and in 2 cases by post-mortem examination. The remote cause of this circumscribed subinvolution without regard to the situation of the placenta, is most frequently a metritis which may have had its origin during the pregnancy, at the time of the delivery, at the lying-in, or from indulging in too early sexual intercourse.

a. Recent (acute) anteversion and flexion in consequence of delayed involution of the posterior wall of the uterus when the site of the placenta. The author analyzes 55 cases, the majority being between the eighteenth and thirty-first years of age—the most active period of the sexual life. Of these, 28 were observed in primiparæ, 10 in the second confinement, 9 in the third, 2 in the fourth, 4 in the sixth, and 1 each in the seventh and eighth labors. Seven of the cases were abortions. The range in time after delivery extends from the fourth to the forty-second day. Of these parturient women, 20 were unable to suckle their children, and 25 stated that coitus was consummated in less than six weeks after labor. A large proportion of the cases were connected with endometritis, or colpitis; a high degree of anæmia was frequently observed.

β. Chronic anteversion or flexion in consequence of defective involution of the posterior uterine wall, when the site of the placenta.

In this group 86 cases were observed. The duration of the cases, when observed, ranges from nine weeks to twenty-one years: all counting their sickness from the last labor. Over one-third were primiparæ, and over four-fifths did not exceed the fourth confinement. More than fifty per cent. of the cases was observed between the ages of twenty and thirty years. The remote etiological relations were essentially the same as in the first group (a).

2. Retroversion or flexion of the uterus in consequence of lengthening and thickening of the anterior uterine wall when it is the placental seat.

Of this division, it is essential to their causation that the placenta have their site on the anterior wall, and not at the fundus, with part on the anterior or part on the posterior wall. Martin comes to the same conclusion with Rokitansky, Klob, and others, that in these instances of partial restitution of the organ, the submucous connective tissue is reconstructed in a faulty manner, and thus disposes the organ to version or flexion.

a. Recent (*frische*) retroversion and flexion, within eight weeks after labor.

β. Chronic retroversions and flexions.

a. Of the group of recent cases Martin analyzes 49. About twenty-eight years of age is the period of greatest frequency. The time during the parturient period in which the error of form or position was discovered was in 16 in the second week, 10 in the third, the remaining cases being distributed about equally in the weekly intervals up to the eighth week. In 14 cases the last preceding birth had been either premature or an abortion.

β. Chronic retroversions and flexions of this group were observed to the number of 205 cases. Abortion occurred in the history of one-fourth of the cases. In nearly 25 per cent. the cases were primiparæ, and in 85 per cent. the number of previous births did not exceed the fourth. The largest proportion of the cases observed, 61 per cent., occurred in the decade between 25 and 35 years, showing the potent etiological conditions existing in frequent labors or abortions. The majority of the cases were connected with impaired nutrition.

3. The following causes may be regarded as remotely affecting the restitution of the uterus after delivery, or as setting up morbid processes in the organ, which tend immediately to the production of versions or flexions. Partial spasm of the uterus after delivery, or adherent placenta (Siebald). Irregular labor-pain, tetanus of the uterus (Mende). Absence of uniformity of uterine contraction; injudicious drawing on the cord; shortness of the cord (Mende, C. Mayer); or, that flexions may be caused by cramp (Bauer).

c. Causes existing in inflammatory and other conditions due to parturition.

In the child-bearing woman it is difficult to isolate inflammatory processes due to parturition from those which may arise from other causes. So important a factor in the production of uterine disease is parturition and its attendant conditions, that it deserves separate consideration. One important effect of inflammatory action has been touched upon in the arrest or retardation of the involution process (β). There is left for us to consider those more evident inflammatory post-partum conditions.

A cause which leads to the speedy development of flexions is the relaxation of the mass of pelvic tissue due to intense inflammatory processes. Softening, as a primary result of acute inflammation, must precede all other etiological antecedents of flexions (Thudichum).

Interstitial hypertrophy and general or partial corporeal indurations must, from the nature of things, be secondary conditions. Inflammatory softening having occurred, two conditions develop flexions: pressure of the superincumbent abdominal viscera, and the gravitation of the uterus itself. It is probable that only in this state of the organ can Dr. John Williams's idea of flexion from gravitation be true. Its operation in any other condition of the uterus may be ignored. Inflammatory hypertrophy may be general, or corporeal, or cervical. In the former retroversion is the more common result; Bennet says anteversion. In hypertrophy of the cervix, anteversion of the neck usually results. Dr. Meadows says "that chronic metritis and so-called hypertrophy tend, sooner or later, to the production, first, of retroversion, and finally of retroflexion." We may, how-

ever, believe with Beigel that a direct etiological condition consists of tissue alterations at the inner os, and enlargement of the fundus and body, in which case retroflexion could be a necessary result without a preliminary retroversion. With this inflammatory tissue-softening at either the inner os or in the body of the uterus, it is not remarkable that flexions result through the strong action of the abdominal muscles, as in lifting, springing, straining, coughing, sneezing, vomiting, or in difficult defecation (Rigby, Arneth, C. Mayer, Kiwisch, Sommer, Rockwitz, Moir, Hueter). Thickening of a wall of the uterus after delivery, subsequently followed in the healing process by retraction or atrophy of the affected side may act as a cause (Boivin and Dugès, Meissner). Martin found in twenty-eight cases of flexion an inflammatory post-partum thickening of the cervix. Post-partum engorgement and hypertrophy is a most frequent cause of versions due to the increased weight of the uterus (Henry Bennet, Boivin and Dugès, Lisfranc). Excessive vomiting under these conditions has been observed as causing flexions (Conway, Edwards). The inflammation due to obstetric operations frequently terminates in flexions of the uterus. The dorsal position, long maintained after delivery, particularly when the binder is firmly applied over the lower bowels, promotes retroversion or flexion (Martin). Premature exertion on the part of the lying-in woman while the uterus is normally undergoing the restitution process, and without necessarily exciting inflammation, but as the simple result of a mechanical force acting on a softened uterus may cause or predispose to version or retroflexion.

d. Causes due to inflammatory and other conditions not parturient. Much difference of opinion exists as to the possibility of a traumatic, or, what is nearly the same thing, an acute flexion. Kiwisch was the first (Hueter) to demonstrate that the normal uterus, taken from the dead subject, could not be given a flexed form at will, since the firm tissue, the considerable thickness, and slight length of the organ, with its marked resilience, resist such a change in form. Dr. Squarey assumes that traumatic flexions are due to the rupture of uterine fibres in women previously healthy; tissue-changes occurring at the seat of the injury being due to traumatism and not antecedent changes. Dr. Bantock, commenting on this theory of Dr. Squarey, says the curvature would not occur on the side opposite to the injury, except temporarily from swelling of the ruptured fibres, but that the true flexion would be found later at the seat of the injury, from atrophy of the injured tissues. The weight of authority is against any such view. Beigel does not admit the occurrence of so-called acute flexions, except as the result of previously existing uterine disease, as shown by the presence of symptoms referable to the uterus. Scanzoni and Rokitsky believe that tissue-changes, impairing the normal resilience of tissue, and especially the theory of the latter author of the destruction of the submucous connective-tissue layer,<sup>1</sup> exist as a preliminary to flexions.

<sup>1</sup> German authority upon the theory of Rokitsky is of considerable interest. Dr. Ludwig Joseph concludes an elaborate paper, *Beiträge zur Etiologie der Uterusflexionen*, in *Beiträge zur Geburtshilfe und Gynäkologie*, Bd. ii. p. 133, in the following emphatic words of disapproval: "Rokitsky's theory of the normal structure of the uterus and the origin of flexions is untenable, since the anatomical basis on which it rests does not exist."

Hueter simply admits that with these submucous connective-tissue changes at the inner os, strong muscular effort, particularly of the abdominal muscles, may increase the angle of flexion. Hewitt admits a modified acute flexion brought on by muscular effort during menstruation, and cites a case. West and Sommer hold, with Squarey, the idea of acute primary flexions; but we may safely exclude this from among etiological conditions except such as exist as a result of tissue-changes, and which are only fully realized in the chronic form of flexions.

The catarrhal inflammation of the uterine cavity, either of the neck or body, owing to the changes induced in the mucous membrane and contiguous layers, is a constant source of versions or flexions. So commonly is this inflammation present that it is rare indeed to find a childbearing woman suffering from a version or flexion in whom evidence of endometritis is not found. We have but to recall the obliteration of the internal os, the thickening and tenderness of the walls, the hemorrhagic and tender mucous lining of the fundus as the result of a chronic corporeal endometritis, in order to realize how readily these conditions drift into the necessary antecedents of versions or flexions. Papillary hypertrophy (so-called granular erosion) of the os, acting by the extension of the inflammatory process, in which it has its origin, to contiguous parts, and thus invading the cavity of the cervix, must be given causative value. When retroversion remains after the cure of erosion (Bennett), it is evidence that inflammation is yet lurking in the cavity of the cervix. The endometritic inflammatory conditions often taking place from deranged or arrested menstruation by taking cold, can also lead to those tissue-changes which result in flexions or versions. Cases of flexion which seemed to be traceable to these sudden outbursts of inflammation of the endometrium, led Martin to the idea that these flexions were often caused by rheumatism of the organ, from taking cold.

Specific inflammation must be regarded as a factor in the production of versions or flexions. It is rare to meet with a woman who has been infected with a blennorrhœa that is free from these errors of form or position. Profuse menstruation, or metrorrhagia, aside from their causative relation, may, by the ramollescence of uterine tissue, result in these displacements. For the same reasons, dysmenorrhœa, either congestive or obstructive, may, by inducing, first, softening of tissue, second, inflammatory congestion and hypertrophy, result in flexion. Injury to the cervix and to the cavity of the cervix by inflammation in near parts as a primary, and from atrophy, or cicatricial shrinking of the injured point as a secondary cause, has an important bearing. The latter cause, when acting on a point near the inner os, must almost inevitably result in flexion.

Atrophy of the uterine muscular substance, especially that form which follows puerperal involution (Klob), needs but the operation of an exciting cause to develop a flexion of the organ. This form occurring at an early period of childbearing life, may produce a serious form of the displacement. Marastic atrophy, taking place generally at a later period of life, is of less consequence in a surgical point of view, although equally potent as a cause of flexion. In aged women with a relaxation of the uterus, the pressure of the intestines upon the fun-

dus of the uterus causes an ante flexion (Klob). Rokitsky describes a peculiarly developed transverse vein in women who have borne many children, which passes through the anterior part of the cervix at about the inner os, and which, by developing its lumen at the expense of the resistance and solidity of the uterine wall at that point, permits the organ to ante flex. Klob mentions having seen these veins a line and a half in diameter. Cases of flexion have been traced to elongation of the utricular glands, and which, by growing into the submucous layer at the region of the internal os, materially weakens the organ, and thus permits the development of flexion. An exuberant production of the ovula Nabothi, and by their enlargement causing an atrophy of the uterine tissue, may tend to flex the uterus in the same way; or, by subsequently bursting, develop a loose, reticular structure, which renders it impossible for the uterus to hold its normal form (Rokitansky, Hueter, Klob).

c. Versions and flexions due to morbid uterine growths. Various morbid products developed in the uterine wall tend to produce versions or flexions. It may so act in two ways: first, by dragging the uterus toward the side of the greatest gravity—a version resulting; or, secondly, by inducing an hypertrophy of the wall involved in the direction of its long axis, and therefore inducing an approach of the fundus and cervix toward the shortest side—thus causing a flexion. The situation of the morbid growth in the uterine wall when high or toward the fundus, may retrovert or antevert the organ; when near the vaginal junction, upon the same side, it may force the uterus to the opposite side. The law seems to be: that for small fibroids—the size of a walnut—near the fundus, version occurs toward the side involved, as the result of gravity; when at the supravaginal cervix, ante flexion results toward the opposite side, from the pressure of the growths upon the near parts. Hueter expresses the law of version or flexions from this cause by a diagram in which a line intersecting the centre of gravity of the uterus, midway between the fundus and os externum, is drawn perpendicular to the horizon. Tumors situated laterally near the fundus, in either wall, ante flex the uterus, and when developed in the lower part or middle of the posterior wall, induce the opposite form of displacement. When the growth becomes large enough to impinge upon the brim of the pelvis, it acts as a displacing force, forcing the uterus in a direction opposite to its seat of development. Kiwisch has observed lateral flexions or torsions from the presence of uterine fibroids following a somewhat similar law of displacement. Intrauterine polypi, when of small size, produce, oftentimes, a retroversion similar to that caused physiologically by a pregnancy under the third month. When these growths are pedicular within the cervical cavity, ante version or flexion is generally the result. When of sufficient size to force the fundus uteri above the brim of the small pelvis, versions or flexions backwards, caused by the earlier stages of growth, disappear, and the uterus becomes anteverted or ante flexed (Boivin and Dugès, Kiwisch, Velpéau, Sommer, C. Mayer, Rockwitz, Scanzoni, Hodge Sims, Säxinger, Hueter, Gusserow).

B. Causes existing in pelvic inflammatory conditions.

a. Versions and flexions due to adhesions of the uterus to near parts.

b. Versions and flexions caused by inflammatory exudations.

c. Versions and flexions caused by cicatrization of pelvic tissue.

a. It is not a thoroughly settled point in etiology, whether, when adhesions are found coexistent with flexions of the uterus, the flexion of the organ was, not a condition prior to the adhesions. Tilt and Scanzoni both concur in the belief that adhesions are often the result of prolonged misplacement. The displacement and the adhesions may, however, be the co-result of a common factor, and the two conditions mutually prolong the existence of each (Bennet, John Williams). A common form of flexion from adhesion is that in which the vaginal portion has contracted adhesion with the vagina—generally that of the posterior periphery of the cervix to the posterior vaginal wall (Rockwitz, Velpeau). Sommer states that adhesions of this character are often the result of ulcerative processes of the uterine neck. In this situation one might readily confound the cicatricial contraction of old ulcers with simple adhesions. In this manner adhesion of the cervix may cause flexion, the body remaining unchanged in form, just as the body may become flexed while the cervix retains its normal form and position (Hueter).

According to Virchow, flexions are caused by adhesions of the uterus when the organ has not the necessary movement in the antero-posterior direction. Antelexion is produced when the fundus uteri is so fixed that in the expansion of the bladder it cannot be moved backwards. The fundus then lies close upon the posterior wall of the bladder, and as the cervix is fixed, it follows that the thinnest point on the uterine body—that where the body and cervix unite—is the seat of flexion. That flexions are for the most part the result of partial inflammations of the peritoneum, in which the exudation is organized into connective tissue which causes firm adhesions, and which, later, causes the flexion by contraction. The ligaments of the uterus may also participate in both the inflammatory action and the subsequent contraction. Hueter makes a proper comment upon this: It cannot, indeed, be denied that a local peritonitis can develop after a flexion has been a long time in existence. Velpeau, C. Mayer, Rockwitz, DePaul, Arneth, Picard, West, Manuel, Säxinger, and Hueter, speak of peritonitis with adhesions developing after a flexion has formed. According to Kiwisch, it is especially the exudations which lie to one side of the uterus that cause the lateral flexions. In these cases Martin has noticed the connection of antelexion with lateral version.

Martin has observed an extremely rare form of retroversion and flexion caused by shrinking of the lig. pubovesico-uterina, the result of inflammatory exudate, or from cicatricial contraction of the anterior vaginal vault. It may be observed to begin with an approach of the whole organ to the anterior wall of the pelvis (ante-position uteri). The lower anterior uterine segment is drawn forwards and fixed. Posterior to the neck the uterine body may be felt, in old-standing cases, forming a sharp angle with the vaginal portion. Of the 28 cases observed by Martin, 22 were married, of which 19 were sterile. The accident appears to be one belonging to early adult life, 19 of the cases coming under observation previous to the thirtieth year. Another variety of

this form of uterine displacement is caused by traction of a ligamentum ovarii and fixation due to a small ovarian formation. In this form the uterine body forms an angle with the cervix, backwards and laterally to the side on which the diseased ovary lies. This state of affairs exists only when the ovarian cyst is small. When the tumor increases in size the uterus becomes detached from its surface. In this class the uterine body may be either retroverted or retroflexed.

A rare form of anteversion and flexion has been traced by Martin, to contraction of one or both round ligaments, and by adhesive exudation upon the anterior peritoneal surface of the uterus. The body of the organ can be felt behind the os pubis with more or less absolute fixation. The flexion is generally at an obtuse angle, and when due to the shrinkage of one lig. rotunda is placed obliquely. Thirty cases were observed. Masturbation was noticed as a remote cause. The shrinkage involving the ligaments is usually due to exudative inflammatory processes in the peritoneal covering. Abegg has verified, by a post-mortem, that a state of complete anteversion was dependent on old adhesions anteriorly, and also between the posterior uterine surface and the small intestines, the lumen of which was much narrowed.<sup>1</sup>

Anteversion and flexion due to shrinkage of the sacro-uterine ligaments, or to inflammatory plastic exudation upon the peritoneal covering of these parts, is a comparatively common etiological condition.

Martin observed 37 cases of antelexion, and 18 cases of retropositio uteri from this cause in a total of 217 cases. From fixation of the lower uterine segment to the posterior pelvic wall, the organ is often in a state of retroposition, the corpus and cervix uteri both being flexed forwards. This condition is usually found early in the morbid process. Later, from the action of the rectal contents, retroposition disappears, and antelexion is the prominent feature. Martin's pathological investigations throw considerable light on this condition. The exudation of pseudo-membrane with spots of discoloration was found upon the posterior wall of the uterine neck, and upon the peritoneal covering of the hollow of the sacrum. Schultze observed 71 cases in a total of 250. Of this number, 22 were virgins, 37 had borne children with many abortions. Schultze lays stress upon mechanical causes, as in too violent coitus, coitus with a proportionately too long penis, drawing of the Douglas folds in great efforts at defecation, instrumental deliveries. Martin regards septic poisoning, taking cold during or near the menstrual period, bathing—especially sea-bathing—as common causes of perimetritis posterior. Gonorrhœal infection is a common cause (Martin, Duncan, Bernutz). Freund has described a chronic atrophic posterior perimetritis in which inflammatory hyperplasia of the connective tissue naturally leads to shrinking of Douglas folds by absorption of the exudate. P. Müller dissents from the conclusions of Schultze—that the condition of perimetritis posterior as a cause of flexion in multiparæ is not a frequent con-

<sup>1</sup> Aran says: Dans l'antelexion les lig. suspubiens sont tendus, résistants et surtout très-courts. Dans la latéroflexion, le lig. suspubiens correspondant au sous de la flexion est constamment raccourci tandis que celui du côté opposé est tendu et allongé. (Leçons clinique, Maladies de l'utérus, p. 389.)

dition; that when shortening of the lig. sacro-uterina does occur, it is probably an additional complication, and that functional arrest of the sexual organs is due to the flexion and not to the complication.

*b.* Versions and flexions caused by inflammatory exudations (phlegmons). In this group, the error of position is due to the displacing force exerted by the inflammatory mass thrown out in the intrapelvic cellular tissue. Antelexion with a lateral inclination is a common form of deviation of the uterus from this cause, as the Douglas pouch, or its lateral margins, is obnoxious to pelvic phlegmon or abscess. The largest of these inflammatory exudations, from often having their seat intraperitoneal, instead of intracellular, induce displacements of the uterus, the extent of which is only limited by the opposite pelvic wall. Usually, ante- or retro-positio uteri is due to a traction-force in the direction of the displacement; but, in this etiological group, these errors of position are caused by a displacing force pushing from behind. This displacement of the whole organ gradually disappears on the evacuation of the abscess or resorption of the phlegmon; and version or flexion occurs from the persistence of adhesions, or shrinkage in the wall of the abscess. While adhesions and fixation are marked features in the uterine displacement, yet the adhesions in the early stage take no part in the displacement, that, seemingly, being dependent upon the crowding of the inflammatory mass. The extent to which the uterus is sometimes distorted from its natural form and position by this cause is remarkable. Duncan mentions cases in which a hard tumor was felt in the iliac fossa—an extension, apparently, of the phlegmonous mass—but which, on an examination by the sound, proved to be the uterus, which had been driven out of the small pelvis by the exudation, and carried to one side by adhesions. Bernutz has described cases of periuterine lateral phlegmons involving the lower uterine segment, in which the cervix, instead of being pushed away, is drawn into the affected cul-de-sac. In other cases recorded by this author, latero-versions, which were the most frequent of all the displacements, were observed connected with torsion of the uterus on its axis as the margin of the tumor was advanced.

*c.* Versions and flexions caused by cicatrization of pelvic tissue. This is a group of uterine deviations valuable only in its etiological relations. These are caused mainly by (1) the contraction of abscesses in suppurative parametritis, by (2) fistulous openings into the bladder or rectum, and by (3) contractions following the too free use of caustic applications to the vaginal vault or intravaginal cervix.

The contraction of pelvic connective tissue involved in severe cases of parametritis is sometimes extensive, but usually not to the extent of arresting the function of the uterus. The lateral margins of the recto-vaginal sac being common seats of these abscesses, lateral versions and torsions are very common results. When developed in the vesico-uterine cellular connections within the common peritoneal covering of the two organs, and finding spontaneous exit for the contents of the abscess through the bladder, subsequent contraction of the sac may develop a strong and nearly immovable anteversion. When the abscess escapes through the anterior vaginal wall, subsequent contraction of the fistulous track and opening may cause an approach of

the whole uterus toward the pubes, when the entire vesico-uterine connection is involved. Cases may occur in which, while the fundus is movable, the cervix is drawn forwards by contraction of the anterior vaginal wall forcing the uterine fundus into the hollow of the sacrum. A retraction of the cervix from cicatricial contraction of the posterior vaginal cul-de-sac, may result from the persistent, free, or bungling use of caustics. In the days of free cautery, now happily past, it was not unusual to find the posterior cul-de-sac obliterated, and the posterior labium reduced to a trace, while the fundus was thrown forwards in a nearly incurable antelexion. There seems to be no reason why the posterior cul-de-sac and uterine lip should be the seat of this destructive cautery except that from the position of the patient upon her back these parts more directly confront the operator.

Vesico- and recto-vaginal fistulæ, with the more or less extensive slough attending these accidents, cause an almost endless variety of versions, flexions, and torsions. Sloughing at the base of the bladder quite generally causes retroversion. Cases are noted in which the uterine cervix becomes incarcerated in the contraction following an extensive slough, the organ becoming almost inverted and the uterine cavity opening into the bladder. The author has closed fistulæ so complicated, in which the subsequent menstrual fluid escaped through the urethra. The numerous cases recorded by Emmet and Hayes Agnew quite generally involve distortion of the uterine position or form.

*c.* Causes due to non-inflammatory pelvic conditions.

*a.* Versions and flexions due to pelvic and other malformations.

*b.* Versions and flexions caused by pelvic tumors, extrauterine pregnancy, and periuterine hematocoele.

*a.* Versions and flexions due to pelvic and other malformations. Stolz mentions having found lateral flexions in rachitic deformity of the pelvis. Boivin and Dugès mention as favoring conditions an increased curve of the sacrum backwards and an undue prominence of the sacro-vertebral angle. Excessive capaciousness of the pelvis tends to expose the uterus to the action of exciting causes (Hensley). Strong inclinations of the pelvis favor the development of antelexions, and a capacious pelvis and slighter inclinations dispose to retroflexion (Holst). A narrow and incomplete development of the pelvis—resembling, in many respects, that of the male—observed in cases of menischesis, is often found connected with flexions of developmental origin, with which, however, it seems to hold no etiological relations, but both are co-results of common factors.

Changes in the lumbar curve induce changes in the direction of the long axis of the uterus relatively to its neighboring parts. Aveling gives the normal pelvic angle at 54°, but as the curve of the lumbar vertebræ lessens, the pelvic angle becomes debased. In the last condition the abdominal viscera, instead of being supported by the lower margins of the abdominal walls with a solid base over the os pubis, gravitate within the cavity of the pelvis and force the uterus downwards in a prolapsus, or forwards, or backwards. All exercise upon the feet exposes the pelvic viscera to a series of succussions, that intensify the action of the original cause. The lumbar curve was observed in a number of cases of

versions and flexions, by taking an impression of the back on a strip of sheet lead, and record made of the curve by drawing the outline of the lead, after being carefully removed, upon a sheet of paper. In nearly every case the deep arch normally observed at this portion of the spine was found to be more or less impaired. In the majority of these cases the general health was seriously involved, and the debased curve was explained by the weakening of all the muscular supports of the bony framework of the body. Aveling explains prolapsus of the uterus by this alteration of the pelvic angle, but when we conceive that this angle may, from this cause, be reduced to  $27^{\circ}$  or  $25^{\circ}$ , it is evident that prolapsus cannot be a more frequent result than versions or flexions. Figs 6 and 7 give a good idea of the mechanical effect of this alteration in the curvature of the lumbar vertebræ.

*b.* Versions and flexions caused by (1) pelvic tumors, (2) extrauterine pregnancy, and (3) periuterine hematocoele. In this group versions and flexions are due to displacing forces. The list might be extended to several other conditions which are rarely noticed, such as exostosis within the pelvic cavity, and displacing forces having their origin in the abdomen. Pelvic tumors, other than uterine, hold etiological relations to versions and flexions principally when of small size. New developments in the ovaries, broad ligaments, and tubes have been noticed by Rockwitz as frequent causes. Head has found retroflexion connected with small ovarian enlargements. When ovarian cysts attain considerable size, if the uterus preserves close adhesions to the tumor, the organ is generally distorted in the direction of the growth of the cyst. When growths of this nature have invaded the abdominal cavity and the uterus has become detached from its wall, the organ may become flexed by the downward pressure of the abdominal contents; the direction of the flexion depending very much upon the form of the displacement existing at an earlier stage in the growth of the tumor. Ascites may produce flexions in the same manner, and which, theoretically, in case of impaired lumbar curve, should be anteversion or version, and in cases in which the pelvis preserves its normal angle retroflexion should be the result. To the same class of displacing force meteorism belongs, which is mentioned by Kiwisch and Manuel as a cause of flexions. Parry has observed that extrauterine pregnancy most commonly produces an antero-lateral version with elevation of the uterus; at other times, when the vicarious uterus is developed anterior to the proper organ, retroversion has been observed.

Pelvic hematocoele, like the periuterine phlegmon or pelvic abscess, may produce version by displacement. From the action of this cause the displacing force acts uncomplicated by adhesions, since these are accidental, not necessary, attendants. Adhesions may develop subsequent to the primary cause, and make the uterine displacement permanent, or shrinkage of the pelvic cellular tissue following the resorption or evacuation of the extravasated blood may lead to flexion by traction.

*d.* Causes due to the condition of contiguous parts.

*a.* Versions and flexions caused by the action of the rectum.

*b.* Versions and flexions caused by the action of the bladder.

*c.* The perineum, the vagina, and the uterine ligaments in their effect on the form and position of the uterus.

*a.* That versions or flexions may be caused by the action of the rectum and the various sets of muscles involved in the act of defecation there can be no doubt. Dr. John Williams has carefully studied this subject. Throwing out his theory concerning any normal relation of the fundus of the uterus to a line continuous with the anterior surface of the spinal column, with which he has needlessly complicated the subject, he is undoubtedly correct. In the act of defecation the bladder is first emptied; secondly, the rectum. By the completion of urination, both the uterine fundus and the bladder sink lower in the pelvis. In case of constipation, so common among women, straining is then made and the fundus forced still lower, and, having reached the maximum of descent, becomes fixed. The cervix in this condition is pointed nearly horizontally backwards, and being now the most movable part of the uterus, bends upon either itself or the uterine body. This process, by frequent repetition, may become the cause of permanent anteversion. In case the uterus is in a state of slight retroversion from vesical distention, the straining effort then forces the fundus into complete retroversion, and the lowest point of descent is reached. At this stage the fundus becomes the fixed point of the uterus, the uterine body being also held by the posterior vaginal wall, while further straining effort forces down the cervix. Thus retroflexion results. The anterior vaginal wall may also take part in this process, when, by the fundus being forced deeply into the Douglas pouch, it is placed upon the stretch, and prevents further upward movement by the cervix.

Simple rectal distention from the accumulation of fecal matter, has been known to cause anteversion. Bedford records such a case. Gustav Carus refers an anteversion to the accumulation of feces in the sigmoid flexure of the colon and upper part of the rectum. Rectal distention united with the violent muscular efforts to expel the contents of the rectum, combine two of the factors of anteversion or flexion (Boivin and Dugès, Scanzoni).

*b.* The action of the bladder in causing versions and flexions may be regarded as a minor factor. Boivin and Dugès report a case in which distention of the bladder caused a retroversion, and regard this as a frequent cause of retroversion of the pregnant organ. After delivery, distention of the bladder may act as an exciting cause of retroversion (Protheroe Smith, Denman). Baker Browne cites Sir Charles Locock as reporting a case in which excessive distention of the bladder caused a retroversion in a young, newly married woman. Hewitt credits the bladder with more power to induce retroversion than the rectum has to cause the opposite form of displacement. The bladder, from its position, offers a certain amount of antagonism to a forward displacement of the uterus due to rectal distention.

*c.* The perineum, the vagina, and the uterine ligaments, may take a subordinate part in the etiology of versions and flexions. Tyler Smith believed that rupture, or impairment of the perineum was a frequent cause of retroflexion. If we regard the vagina as part of the machinery of uterine support, there is no doubt of the truth of this. As is already mentioned, the perineum,

in case of a very short vagina, often takes part in the production of developmental flexions of the cervix uteri. In very extensive lacerations of the perineum, prolapsus, when not complete, must always be associated with retroflexion, or version, which gradually disappears in the erect uterus as the prolapsus reaches the third degree.

Dr. Williams has mentioned cases of retroversion due to elongation of the anterior vaginal wall. Sims also alludes to this cause. In the earlier stages of cystocele, anteversion results from the removal of the usual support of the vesico-uterine connection; but as the vaginal prolapse continues and the uterus also begins its descent, the anteversion disappears. The fundus, before the third degree of prolapsus is reached, may even occupy the cavity of the sacrum. Rectocele and prolapsus vaginæ are of doubtful etiological relation to the subject. Prolapsus of the uterus so quickly develops either primary or secondary in its relations to the vaginal vault, that any other form of uterine displacement is lost sight of.

Boivin and Dugès report a case in which anteversion was caused by an inflammatory congestion of the suprapubic ligaments, occurring at each menstrual period. The inflammation began in the left labium, and the thickened ligament could be traced to the inguinal ring. The same authors regard the utero-sacral and the suprapubic (round ligaments), while being shortened, as the result of long-standing displacement, yet this may ultimately exist with the force of a cause, and render the displacement incurable.

Klob, while giving the round ligaments but little value as a means of support of the uterine fundus, believes that they may render an extreme degree of retroflexion difficult of reposition by fixing the fundus. In this instance the line of traction of the ligaments falls below the junction of the uterine body with the uterine neck, and thus tends to double the uterus upon itself when in the third degree of retroflexion. It is evident from the experiments of Savage, on the structures supporting the uterus, that no slight degree of contraction of the round ligaments would tend to develop version or flexion of the uterus forwards.

#### E. General and special causes.

a. Flexions and versions caused by posture, incidental to occupation.

b. Excessive sexual indulgence and masturbation as causes of versions.

c. Impaired nutrition and other causes of a general nature, of flexions and versions.

a. The effect of posture, and especially such as is incidental to occupation, may have a marked influence upon the tendency to version or flexion of the uterus. The relation of long-maintained dorsal position to the causation of retroversion in the newly delivered woman has been already alluded to. Many of the modern industries which so largely employ women, are, without doubt, active causes of uterine malposition. This cause is also found to be combined with errors of diet, deficient food, and bad ventilation. The use of the sewing machine is regarded as a frequent cause of versions and flexions (Hewitt, Squarey). Occupations that require long standing upon the feet, such as saleswomen and mill operatives, favor the development of flexions.

b. Overindulgence in sexual intercourse is alluded to

by many authors as a cause of flexions. Bennet believes that it may cause a retroflexion of the cervix, and especially if the cervix is rigid from inflammation, and the body of the organ yields to the leverage. In such a condition the cervix is thrust back in ante-, and forwards in retroversion. Routh has expressed the opinion that the uterine congestion that attends orgasm may, by too frequent repetition, become permanent, and thus lead to retroversion. While nothing can be objected to the theory involved in this, yet the examination of prostitutes fails to show that versions and flexions are more frequently met with in this class than in married women. An examination of a large number of the female members of the Oneida Community shows that these women, as a class, are remarkably free from versions and flexions.

Masturbation is frequently alluded to by German authors as a cause of uterine flexions. Martin cites this as a cause of anteversion from shortening of the round ligaments and adhesive inflammation of their covering. Such cases of masturbation as the author has seen were attended with either versions or flexions, but nutrition was greatly impaired, and extreme nervous depression present, which may have been the more active causes of the uterine displacement.

c. Impaired nutrition, a state of the general health, called general debility and want of tone, from any cause, are regarded by Hewitt as important predisposing causes of versions or flexions. The impaired vital resistance and general arrest of nutrition that attend excessive lactation and rapid childbearing, are often found associated with flexions of the uterus in very evident etiological relations. Emmet states that mental disquietude, ill-assorted marriages, and the various means used to prevent conception are causes of flexions. Diseases of remote organs, especially such as involve the circulation, have been believed to have a bearing upon the causation of changes in uterine form and position. Sommer states that diseases of the heart, attended with obstructed circulation, cause these changes by producing relaxation of the uterine parenchyma. Wright traces such results to retarded portal circulation, which leads to a venous stasis of the uterine tissue.

From the foregoing review of the etiology of versions and flexions, it is evident that the question how these changes—one of form and the other of position of the uterus—may be brought about by nearly identical local conditions, is one of considerable interest. But that these different results of common etiological factors are not mutually interchangeable, the following figures prove beyond doubt. Ten German observers place the proportion of anteversions at 65 per cent., and of retroflexions at 34 per cent. Emmet, who has had excellent opportunities for observation, makes the difference still greater, being 58 per cent. of the former to .17 of the latter. An examination of the statistics of many writers gives the ratio of retroversions to anteversions as three of the former to one of the latter. Now, while a common cause may in one instance produce either a backward or forward displacement of the uterus, the above figures clearly show that these are not chance results, but are produced by governing causes. Emmet's idea—that a retroflexion is a deviation from a previously existing retroversion—is one that is found running through the writings of various authors, and, to a certain

extent, is probably correct. As a preliminary to version, the uterus must possess a normal condition of stiffness and resistance (Squarey). This retards the development of flexion. It is an established pathological fact that changes in the structure of the uterus are indispensable to the existence of flexions (except such as may be caused by parametritis and displacing forces), either in the sub-mucous connective tissue (Rokitansky), or through the parenchyma of the organ (Sommer). These changes may be the direct precedent of the flexion as the result of antecedent pathological conditions, or may supervene upon a previous state of version. The only instance in which a causative condition seems to produce either a version backwards or forwards is in the abnormally movable uterus of Savage. Here, what is anteversion at one visit may be retroversion at the next—most probably due to the direction in which the superincumbent intestines exert their pressure upon the uterus. Bernutz and Goupil have met with similar cases. Squarey tries to show that the uterus is always at right angles to whatever pelvic plane the organ may chance to be situated. Consequently, when the uterus is well up in the pelvic cavity, it is directed forwards; and when low, the fundus inclines backwards. When, therefore, the organ holds the former relation to the pelvis, the operation of a displacing cause produces anteflexion, and the same cause operating in the latter relation produces retroflexion.