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Primary uterine inertia

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PRIMARY UTERINE INERTIA

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INTRODUCTION.

Primary uterine inertia, is a condition seen by any doctor, who is concerned with the process of birth in his practice. Of course the greater his practice, the more of these cases he will see. Many hours will be spent in weary, watchful waiting, when he has a case of this kind. Many writers believe the incidence of inertia is increasing, and is being recognized more frequently now, than at any time before. Be that as it may, uterine inertia is one of the chief, indirect causes of obstetrical disasters, and as such deserves time, thought, and study.

In this thesis, it is my desire to bring out the most recent accepted ideas of why labor starts. Then, to determine, if possible, why it does not proceed normally, and how to care for a patient, who presents such a problem.

The classical definition of uterine inertia, is ' the uterus, acting without skill'. Stedman defines it as, ' Feebleness of the uterine contractions of labor'. Many limitations and additions, have been given to this condition, but in this thesis, I intend to limit my discussion, to the feeble uterine contractions, occuring in the first stage of labor, its manifestations, and treatment.

PHYSIOLOGY.

The onset of normal labor has been the subject of theorizing for a long time, and many theories have been advanced, some of which will be set down here, saving discussion for the theory most generally accepted at the time. As set forth by DeLee, these theories are:

Theories

1. " The ovum is prepared for separation by the degeneration of the decidua in the later months of pregnancy, and the disappearance of the placental septae, thus loosening the attachment of the latter to the uterine wall. The ovum, then, according to Simpson, Huvê, and Scanzoni, becomes a foreign body. The placenta does not loosen its living attachment to the uterine wall until the child is delivered, therefore this theory is untenable.

2. Thrombosis in the placental vessels causing an increase of dioxide of the uterine blood, has been advanced by Leopold. The thrombosis is not constant, and it too, must be explained, as it is pathologic.

3. Brown-Sequard proved that an increase of carbon dioxide in the blood will evoke uterine contractions. Lack of oxygen will do the same. The increased venosity of the fetal blood, which normally occurs in the later

months, due to the narrowing of the ductus Arantie and ductus Botalli, has also been suggested as provocative of uterine action- all not proved, but they indicate chemic lines of study instead of mechanical.

4. Products of altered metabolic activity of the mother or from the child, accumulating in the maternal blood, may evoke the pains, either by way of the uterine innervation or indirectly through the vasomotor system. Schaffer believes that the syncytium has an influence in limiting the passage of poison from the fetus to the mother. Late in pregnancy the action of the syncytium is insufficient. Others have referred to the possibility of the fetal posions stimulating the uterine muscle. Williams found that just before labor, there was diminished nitrogen output in the urine, with marked diuresis, confirming the suspicion that metabolic changes are involved in the causation of labor. It is possible that, by working this field, the active factor may be discovered. We know that diphtheria antitoxin sometimes bringson abortion.

5. Mauriceau believed that the excessive distention of the uterine wall resulted in labor. The uterine wall is not distended at term, therefore only in pathologic cases, as twins, polyhydramnion, could such a force act.

6. The increased irritability of the uterus is generally acknowledged to be an important factor. We know that the uterus contracts from the beginning of pregnancy, and also that the contractions become stronger and more easily elicited toward the end. This marked irritability is due to the great increase in the development of the muscular fibers and the nerves and cells of the cervical ganglion. When the end of pregnancy is near, some slight accident may make the contractions stronger, and each contraction stimulates the succeeding one until regularity is established.

7. The influence of the periodicity of the menstruation cannot be denied, though why the final effort of the uterus should come at the completion of the ten menstrual cycles is not understood. It is not believed that the ovaries have anything to do with it, since labor can occur without them. The same is true of the corpus luteum, though Born and Frankel have sought to connect the two. In some women, particulary those of a nervous type, there occur, during pregnancy, at the time of the usual menses, peculiar sensations and manifestations which show that some influences are at work. Neuralgic pains, especially in the sacrolumbar region, insomnia, skin erruptions, increase of the varicosities,

vomiting, nausea, diarrhea, constipation, decreased urine, sometimes small hemorrhages, and the tendency to abort, are greater at these periods.

G.DePaoli studied these changes in thirty women, and found diminished lung activity; lower blood pressure; increases sensibility of the skin and of the reflexes; more rapid pulse. Pathologic conditions are more common at these times- for instance- eclampsia, fainting, hysteria, pains in the bones.

8. Pressure of the presenting part on the lower uterine segment on the cervix and the nerves of the great cervical and retrocervical ganglia, and other plexuses around the cervix and upper vagina is an old (Galen, Power) and favorite theory, but it will not explain all cases, such as breech and transverse presentations and the uterine contractions of extrauterine pregnancy. In some cases the head may be deep in the pelvis, and pressing hard upon the cervix, without evoking pains. Still, clinical experience shows that when the child settles well into the lower uterine segment, the dilatation of the cervix begins and labor comes on.

The methods employed in inducing labor nearly all operate by irritations applied to the nerves of this region e.g. the packing of the cervix with gauze, the

application of colpeurynters, bougies etc. We can often bring on labor by passing the finger around the internal os and stretching it a little.

9. The importande of accident must not be overlooked. When everything is ready for labor, the parts softened, the cervix begun to unfold, the uterine muscle well developed and having attained a high degree of irritability, it is easy to see why some slight cause, mechanical or emotional, may suddenly start the uterine contractions.

As soon as one contraction has occured it seems to form an irritant for another, or forces the ovum against the cervix, stimulating the nerves, so abundant here and thus labor is put into progress,

Such exciting causes are physical shocks, jolts, running up or down stairs, coitus, diarrhea, straining at stool, mental shocks sudden fright or joy, chemical, castor oil, quinine, or pituitrin may bring on labor at term."

Recent work reported by Allan and Dodds, also Reynolds, indicate that normones are probably the most important factor in the onset of labor. Corpus luteum normone prepares the uterus for implantation, innibits normal spontaneous contractions of the uterus, innibits estrin preventing ovulation.

Injection of corpus luteum prolongs gestation in animals and interferes with the process of parturition, and is antagonistic to posterior hormone, (corpus luteum is not necessary in humans after the first few months of pregnancy.)

"Estrin is indispensible to the act of parturition. This has been based upon three lines of evidence: 1) Estrin has a motility inducing property in animals as proved by animal experiments. 2) Induces motility in the human uterus. 3) is produced in increasing amounts during gestation,"

Estrogenic substances are the only ones capable of inducing rhythmid contractions in a quiescent uterus, the uterus of a castrated female rabbit.

The innervation of the uterus, has been studied by many workers. In 1847, Purkinje described fibers in the outer coat of the uterus, similar to the 'Purkinje' fibers in the heart. These findings were confirmed by Hofbauer, who found that these fibers were mudh more irritable than the normal muscle, and are much more sensitive to the action of the posterior pituitary hormone. He explains the rapid dispersion of the impulse through the uterus, and correlates the contraction of the uterine muscle, to a mecnanism similar to the 'Purkinje' fibers of the heart.

Polarity of the uterus is explained by the difference in the extrinsic and intrinsic factors, by Rudolph and Ivy. The sympathetic supply comes from the second, third, and fourth sacral, and presacral nerves, by way of the hypogastric plexus. The intrinsic factor is demonstrated in the uterine muscle, and although actual connections to the muscle cannot be satisfactorily demonstrated, there is sufficient evidence of the two factors and that it is the working together of these two mechanisms, which coordinates the contraction of the longitudinal fibers, and relaxation of the circular muscle fibers.

W.Blair Bell states, "From experimental evidence of pithing and clinical observation of paraplegias, it is clear that a part from emotional effects, the central nervous system exercises no essential control over the uterus, during parturition. This does not exclude however, that spinal nerves may not play some part."

Fitzgibbon gives all of the credit to pressure on the cervical ganglion and bases his idea on the ability to induce labor, by rupturing the membranes, which permits the presenting part to press on this area. Also by the use of bougies, and manual dilatation, in bringing on good, strong, regular, pains by these methods, which are entirely pressure, where before they were feeble,

irregular and incoordinated.

Now to consider the evidence and theories of these various workers and to correlate their various statements into one theory upon which they all agree.

At the beginning of gestation the corpus luteum hormone is necessary to prepare the uterus for implantation of the fertilized ovum. It inhibits the action of estrin, which sensitizes the muscle. It also inhibits the pituitary hormone from producing another cycle and bringing about menses. This functions in the female human, in the early months of pregnancy after which time the action is aided or maybe taken over entirely, by the anterior pituitary hormone.

Estrin, produced in the ovary, gradually increases in amount in the maternal blood stream. The placenta may produce estrin too, as shown by Zondek. This rise gradually continues and is greatest at the time of parturition, as shown by Reynolds, Allan, Dodd and others. Estrin sensitizes the uterus to the action of pituitrin as shown by W.Blair Bell, Datnow, and Jeffcoate, Allan and Dodd, and Hofbauer. Just prior to parturition there is an increase in the amount of posterior pituitary hormone in the maternal blood, probably ariseing from hormonal stimulation of the gland as shown by Samuel Morris. With the increasing irritability of the uterus,

and the decrease in inhibiting substances, the uterine contractions which have been occuring all through pregnancy become regular and rhythmic, tending to expell its contents and thus creates pressure on the cervical ganglion. With each contraction wave the action becomes more and more coordinated and expulsive in nature. The lower segment forms, due to the relaxing of the circular fibers, the cervix thins and dilates and the infant is expelled through the birth canal.

In this explanation it is evident that there some factors omitted and others perhaps not given full value, such as, mechanical pressure, innervation, and accident. These may, at all times, be more or less important in all labors. Another thing, the power has hardly been mentioned, except in coordinated action. The passages have not been considered, nor the passenger, except as a dilator, These are all important, not only in normal labor but also in consideration of our subject, inertia.

MECHANISM INVOLVED IN PRIMARY INERTIA.

Uterine inertia, atonia uteri, delayed labor, prolonged labor, and many other names have been applied to the condition I wish to discuss but they all apply to the same or to a similar clinical condition.

To define the term ' uterine inertia', the classical derivation means, "without skill". In primary uterine inertia, the uterine action is sluggish and irregular from the onset. This is true inertia. Inertia, developing in a later stage of labor is exhaustive and by some authorities, is called secondary inertia. It is truly a misnomer. In this thesis, we are concerned with the former.

There are many factors involved in the delay of labor and they will have to be differentiated. They can be divided into two types, due to one or both of two causes, either because there is too little power, or too great a load. In other words, the inertia type or the obstructed type. In well marked cases, clinical differentiation is not difficult.

In true inertia, the action of the uterus is slow, sluggish, and irregular many times, and feeble. Although the contractions may be painful, there is only a brief hardening of the uterus, and vaginal examination will

reveal very slow dilatation of the cervix, with slow or no descent of the presenting part. There is no evidence of obstruction. The contractions merely lack expulsive force.

In the obstruction type, the 'pains' of labor start gradually, become regular and of good quality, and they may continue normally for sometime with normal progress. After a time progress ceases, the contractions continue,but soon there is evidence of fatigue and exhaustion. The strong regular contractions decrease and become feeble. Most cases of obstructed labor are however, of minor degree, because the obvious cases of obstruction are discovered during pregnancy. Since the obstruction is minor, uterine inertia enters the picture, in that the expulsive force is not sufficient to overcome the resistance present. In these cases the real factor for the delay maybe hard to determine.

M.W.Vaux lists the etiological factors of uterine inertia as follows:

"1.Defective innervation (paralysis of nerves which presides over uterine contraction.)

2. Defective development of uterine musculature.

3. Abnormal shape of the uterus, due to anomalies.

4. Abnormal position of the uterus, as seen in antiflexion of pendulous abdomens, also prolapsis uteri.

5. Over distention of the uterus, as in poly-hydramnious, and multiple pregnancies.
6. Neoplasms of the uterine wall and cervix.
7. Adhesions between the amnion and the uterine wall.
8. Rapidly succeeding pregnancies.
9. Hormonal factors not being in proper balance."

In these factors of etiology, neither disproportion between fetus and pelvis, nor obstructions are listed. Another mechanical abnormality is the presenting part and its position, such as, breech presentation, occipito-posterior presentation, which may delay labor and many times produce true inertia type of pains.

Bright Bannister also includes: "Incoordinate action of the uterine muscle, (as described earlier). Causes from higher centers such as, fear", which is also described thoroughly, by A. C. Bourne. As to how these various factors produce inertia the following explanations are offered.

#(1.) Deffective innervation can act in two main ways. First, in not giving peristoltic contraction, but merely producing localspasm. Goodall, Rudolph and Ivy, Wentworth Taylor, and others, state this may occur.

Second, they also all agree that, there must be coordinate action, i.e. contraction of the fundus, with relaxation of the cervix, or circular muscle fibers. The lack of this sometimes diagnosed as cervical rigidity, Bright Bannister and J. S. Fair bairn believe so called cervical rigidity is wrongly diagnosed in a nigh percent of cases.

#(2) Deffctive musculature ; #(5) Overdistention; #(6) Neaplasms ; #(7) Adhesions between the amnion and the uterine wall; #(8) Rapidly succeeding pregnancies; are all considered by Fairbairn to be more of an assumption than a knowledge of how, and why, they cause inertia.

#(3) Abnormal shape may inhibit peristoltic action of the contractions and as Rudolph and Ivy have shown, may be due to defective fusion of the two analogen in the formation of the uterus.

#(4) Abnormal position the uterus, causes more or less mechanical obstruction, as well as changing the axis upon which the powers are working.

#(9) Hormonal factors. As shown by their experiments on the uteri of animals, Allan and Dodd delayed the onset of labor by the injection of corpus luteum. The injection of estrin followed by

injection of pituitrin, was much more efficient in causing the onset of regular contractions, than the injection of either one singly. This finding was corroborated by Blair Bell, Bourne and Bell, and Reynolds.

Samuel Morris found in experiments performed on animals and humans, that the products eliminated at the time of parturition, correspond definitly to those eliminated when posterior pituitary substance was injected into a normal subject, and so concludes, that there is a rise in the content of the maternal blood of posterior pituitary substance.

#(10) Incoordinate action of the uterus. This term means, that the correlation of the action of the uterus and cervix, are not in harmony. Wentworth Taylor showed that dilatation of the cervix is not just a mechanical process alone, but the two acts are coordinated.

Rudolph and Ivy from their work, believe that coordination id due to the extrinsic and intrinsic nerve elements, One causes contraction of the longitudinal muscles, the other, relaxation of the circular fibers. Hofbauer, in discussion of a band of special-

ized tissue, in the midline of the uterus, believes that this band of tissue is important in the coordination of action, because waves of contraction spread from this band. When the uterus is not coordinated, the expulsive force is weak and the contracted circular fibers act as an obstruction.

#(11) Causes from higher centers. Fear, we learn in physiology, releases adrenalin into the blood stream. Bourne and Bell, have shown, that adrenalin inhibits uterine contraction. When fear, or pain, causes the patient to contract her voluntary muscles at the time of contractions, labor is delayed, because this forms a muscular obstruction.

CLINICAL MANIFESTATIONS

Frequency:

A diagnosis of uterine inertia is usually not difficult. In fact W. Blair Bell, believes all labor starts as a primary inertia, and it is beneficial in preventing too rapid dilatation of the cervix, and, if there is slight dis proportion, to allow slow descent and rotation of the head, thus preventing injury,

A. C. Bourne and Bell, in their seriesof cases did not consider any cases as inertia, unless the first stage of labor lasted fourty-eight hours or more. This was just an arbitrary time decided upon by them.

When we realize that the condition of inertia is most frequently encountered in the primigravida and that according to the series of cases reviewed byBourne an Bell one in sixty primigravidae had labor which lasted over forty-eight hours, in the first stage, it is easily seen that a slighter degree of inertia might be present in even more patients. The age of greatest frequency is between twenty and thirty. Wilfred Shaw, believes the patients most apt to have inertia, are "'fat', primiparae, between the ages of twenty-five and thirty-five, who are quite high strung and flighty".

The same is true in multiparae, except that inertia only occurs in one out of three hundred cases. In one

series of cases, thirty out of forty-nine patients had premature rupture of the membranes.

Heredity is thought by many authorities to be an important factor, but this does not always hold true.

Clinical Types:

When the pains of labor are weak, infrequent, irregular, and short in duration, they do not cause very great discomfort to the patient, and they may continue thus for many hours without giving rise to exhaustion, but are more apt to cause apprehension on the part of the patient and her family.

If there is incoordination of the uterus, there is failure of the lower segment to relax and the cervix does not dilate, and no progress is made by the presenting part. In this type, the pains may be more noticeable, to the patient, causing more distress and fatigue. These cases are often termed, ' Rigid Cervix', but the term is incorrect. The cervix may not have a disproportionate amount of connective tissue, but merely does not dilate, because the powers of expulsion are not strong enough, nor well enough organized, to dilate the cervix.

Then lastly, there are those patients, who have the typical pains of inertia, but, in the very early stages, the membranes rupture. Naturally these patients are predisposed to infection. Wilfred Shaw, believes

that signs of fetal distress are more apt to appear early, in these patients.

Causes of inertia, from higher centers, is quite easily recognized, because the state of fear the patient is in can be seen readily, by her talk and actions.

Examination:

Examination abdominally, reveals a uterus which becomes firm,or even hard,during contraction, but the contraction is not maintained, It merely comes to a peak of contraction and relaxes immediately. Examination of the cervix usually reveals a cervix that is perhaps two or three fingers dilated, many times less, that is edematous and boggy. Subsequent examinations reveal little or no change, in the cervix, nor in the progress of the presenting part, down the birth canal.

It is most important to try to determine the cause of the inertia. If it is due to obstruction it is of importance to find how extensive the obstruction is and the possibility of the passage of the infant, through the birth canal. If the cause is fear, reassurance and elimination of the fear is in order. If due to feeble effort, on the part of the uterus, try to correct it.

CLINICAL TREATMENT.

Treatment of inertia, naturally begins early in pregnancy, first by ruling out obstruction, uterine anomalies, and other anatomic defects. Then during antenatal care, insight into, and understanding of the patient should be attained, and reassurance given so that the patient may approach labor, with the proper mental attitude, and faith in her attending physician. A.C.Bell states," Inertia cannot be anticipated, except as fear enters in as a factor."

In discussing the treatment, I wish to divide inertia into two groups; mild inertia, and severe inertia.

Mild inertia is considered, in those patients who have ineffectual, colicky, short, irregular contractions, but the cervix slowly thins and dilates, and is soft and yielding.

General measures are used first. Warm enemas, and emptying of the bladder, may relieve the patient, of some bowel or bladder obstruction, and stimulate the uterus into normal regular contractions.

Fear can be removed, if it is a factor, by assurance and explanation, to the patient and her family. Sedation may be a very helpful measure, in cases where fear is present.

If there are signs of fatigue, on thepart of the patient, sedation is very important, because it has been found by many practitioners, that after the patient has rested, the pains become regular and vigorous.

The greatest part of the treatment ,is patience and suggestion, along with mental and moral support. Morphine 1/6 gr. hypodermically, is the sedative chosen by the most men, although hyoscine, scopolamine, chloral, bromides, and the barbiturates are used extensively, with success. DeLee suggests that 'uterine massage' is helpful.

Theoretically, the use of oxytoxics is correct in trying to overcome inertia, but care must be taken and obstruction ruled out. The dosage advised by Wilfred Shaw is two units of pituitrin as a maximum, during the first stage. A. C. Bourne finds two and one-half ($2\frac{1}{2}$) units, can be used with impunity, andmay be repeated often. He also finds it is more successful if estrin has first been given. Other authors seem hesitant to use pituitrin, because of its dangers. DeLee suggests its use as a later resort, giving fifteen (15) minims every three hours, for three doses.

A. C. Bourne advises rupture of the membranes in cases where the os of the cervix is three-fourths dilated, but advises against bags, or manual dilatation, and

believes that the use of forceps, or version, are to be considered for actual delivery.

In the severe types of inertia, where the cervix is still hard, and not dilated or dilating, the foregoing measures should be tried first, avoiding too active manipulation.

When the membranes are not ruptured, too active measures are not indicated and waiting is much safer.

If the membranes have ruptured early, and the patient is dis tressed, or if there is fetal distress, caesarian section is the treatment, advised by Shaw, Bourne, and Maliphant. R. G. Newton, however, advises trachelotomy, rather than caesarian.

A recent report on the use of acetyl-choline, in the treatment of uterine inertia, has been presented by Bell and Playfair. Their method was to select cases for treatment that had lasted forty-eight or more hours, whether due to weak pains, or colicky contractions, either case being associated with dilatation of the cervix. 0.2 gm. of acetyl-choline (acetyl B methyl choline)was given intramuscularly, every three hours, for four doses. If the patient delivered in less than twenty-four hours after treatment was instituted, they considered the treatment successful. In the series of twenty-three cases, eighteen were successful, three

failed, and two were borderline cases. Of those sucessfully treated, the average time of delivery, after treatment was begun, was eleven hours. These results seem very good, and this treatment may prove to be very valuable, and practical.

General measures such as diet, dehydration, and prevention of infection must be instituted. Fetal distress must always be kept in mind, and looked for in these long labors, and it is considered one of the important signs, for early use of the more drastic methods.

In treating any case of uterine inertia, it is important, as Maliphant says," to treat the patient , not just the condition".

CONCLUSIONS.

The onset of labor is probably due to the rise in the estrin content of maternal blood, and the decrease of inhibitory substances. This rise in estrin makes the uterus more irritable and sensitive to the posterior pituitary substances, and the change in hormonal balance causes secretion of the posterior pituitary hormone, in the last few days of pregnancy.

When the concentration of pituitrin is great enough, the sensitized uterus begins to contract rhythmically, producing pains of labor. With each succeeding contraction, the efforts become more and more expulsive and coordinated, causing pressure changes, which probably assist in maintaining the rhythmic contractions of labor.

Primary uterine inertia is the uterus acting with out skill. The obstructive type of inertia, the malpositions, and abnormal presentations, are not considered.

Causes of true inertia are: defective innervation; abnormal shape; incoordinate action; hormonal unballance; and fear.

Uterine inertia occurs in one out of every sixty primigravidae, and one in every three hundred multiparae. It cannot be anticipated, except in ruling out fear and obstruction.

Manifestations clinically, are weak, irregular, colicky pains of short duration, causing the patient more or less distress, but producing only slow dilatation of the cervix, if any at all. The membranes may or may not rupture early.

Clinical treatment is, first of all, assurance to the patient, then, mechanical measures such as, hot enemas, hot water bag to the fundus, sedation such as morphine, hyosine, chloral, bromides, scopolamine, and the barbiturates.

Pituitrin 2 1/2 units as a maximum dosage, may be given and repeated often.

Rupture of the membranes, if the os is 3/4 dilated is many times very beneficial.

Acetyl-choline 0.2 gm. intramuscularly every three hours for four doses, has given good results.

Version or forceps are very successful in many selected cases for the termination of labor.

In cases of fetal and maternal distress, caesarian section is the method of choice, even though the membranes are ruptured.

Above all, reassurance, patience, and vigilance, remembering to treat the patient, not just a condition, is of paramount importance and must be followed, in treating uterine inertia successfully.

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