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There is probably not any condition which causes more worry, both to the general practitioner and obstetrician, than primary inertia of the uterus, and it is with a hope of imparting a better understanding of this abnormality that the author has studied the clinical signs and symptoms of this condition. In cases of primary inertia of the uterus, as distinguished from secondary inertia, the uterine inefficiency in pains is present from the beginning of labour; whereas secondary inertia is merely an expression of fatigue after normal uterine effort.

In primary inertia, there is a true lack of normal uterine response This lowered nerve-muscle irritability may manifest itself in a retardation of the normal time for the onset of labour, thereby causing unduly long pregnancies, with inordinately large children; or labour pains, of the type which we conceive to be true labour, may be preceded by days or weeks of distressing uterine cramps, of such severity as to cause inability to walk or sleep. These cramps may extend down the thighs and are usually greatly increased by exercise. When, in response to these ineffectual pains, the lower uterine segment stretches somewhat and a slight show of blood appears, we assume that normal labour has begun, but really the pains change very little in character from the preceding days and, owing to the lack of complete subsidence of the uterine spasm between pains, the patient is never completely free from distress and, therefore, lacks that complete rest and somnolence of the interval of normal labour pains. In other instances, labour sets in as in normal cases, but after some hours the advance is inordinately slight as compared with the effort, and the pains are usually badly borne owing to the lack of normal rhythm. The consequence is that the patient soon loses patience, her *morale* suffers, and now a train of distressing symptoms sets in. Firstly, the membranes frequently ruptures before conscious labour starts, and during the long hours and days of slow labour the amniotic sac becomes infected, and the afterwaters become fetid. Absorption leads to a rise in the tempera-

ture and to a rapid pulse-rate; vomiting frequently occurs after the second day, and owing to this, acidosis and dehydration follow rapidly, and eventually exhaustion and complete arrest of labour are the rule. The foetal heart gradually slows down to a dangerous rate, becomes irregular in rhythm and volume, and the life gradually peters out. If it should happen to be a posterior position, as it frequently is, a large caput succedaneum develops, and the inefficient pains cause a lack of internal rotation, so that the head is arrested in the transverse or posterior oblique diameter, and labour is usually ended, owing to either foetal or maternal distress by a difficult correction and forceps—or forceps without correction of the abnormal presentation. The end result is a depleted, infected mother, usually badly injured during instrumental delivery, and a high percentage of foetal antenatal death, or trauma and subsequent death.

It is possible to anticipate some of these cases during their ante-natal care. They conform to a type. Such patients are usually fat, short-necked, flat-nosed and nasal-speeched, and have thick unhealthy membranes everywhere. Many of these patients present male hirsutism, and others secondary male characteristics, including a male pelvis. But there are many which do not conform to this type.

Two years ago the author found that after these patients had been in labour for some hours, there appear a series of signs which are seldom absent and which stamp the cases at once as of the 'inertia' type. These signs are a very distended colon; the distention may affect either the sigmoid colon or the ascending part of the colon. It is not a matter of indifference which part of the colon is involved in the distension. The whole colon and rectum are under the influence of this paresis, but the lie of the uterus, and more particularly the lie of the child in the uterus, determines on which side of the mid-line of the abdomen the bulk of the uterus will lie, and that allows more room for the colon on the opposite side of the abdominal cavity. These factors initiate a progressive condition of increasing intestinal distension and uterine displacement. Eventually the uterus is crowded to one or the other side of the abdomen, and the opposite side is filled with a colon which is often the size of a small football. When the uterus contracts the bowel also seems to be simultaneously and weakly involved in spasm, and two mounds with a depression between them fill the abdomen. At times the stomach is similarly affected, but generally to a much lesser degree. The effect upon labour is to displace the axis of the

uterus and misdirect the uterine force, which adds further to the already great dystocia.

After the uterus has been in action some hours, it frequently assumes a peculiar hour-glass contraction, which may be quite alarming to the uninitiated, as suggesting a malpresentation. This appearance is often accentuated by a full bladder, and is due to a more forcible contraction of the circular over the longitudinal fibres, and is only another expression of a vitiated function.

Another sign of inertia is a paresis of the urinary bladder by account of which, even without engagement of the presenting part, this organ will not empty itself, becomes over-distended and catheterization has to be resorted to.

The syndrome, therefore, consists of an abnormal rhythm and tone affecting the hollow viscera of the abdomen, chiefly uterus, bowel and bladder, causing a malfunction in all of these organs. It is more than probable that other hollow viscera are similarly involved, though less easily detected.

This inertia frequently persists into the puerperium, causing grades of intestinal or vesical obstipation, and degrees of delayed uterine involution, and a great deal of epigastric burning and postpartum vomiting. What is the cause of this syndrome? The explanation is simple to a certain point, beyond which we cannot progress in our present stage of imperfect knowledge. First of all, a glance at the accompanying chart will show that all the organs affected by the inertia are fed by a common branch of the splanchnic nerve. We may further note that the organs involved in the inertia all suffer from a similar departure from the normal rhythm and tone of their muscular function. It is fair, therefore, to assume that it is a disturbance of the autonomic system of the splanchnic area, and that as the autonomic system is under the influence of the endocrine glands, the conclusion is logical that we are dealing primarily with an endocrine disturbance. The description given above of one of the individual types that is generally the subject of uterine inertia rather lends conformation to this hypothesis. That other cases of inertia do not conform to the type is readily explained by the statement that the former are congenital fixed types, the latter acquired and, as can be proved, not fixed, because they may differ in consecutive labours. However, such patients are nearly all uniform, in that they become very fat during pregnancy.

The clinical influences of the inertia syndrome are numerous and serious. They involve degrees of maternal exhaustion, some

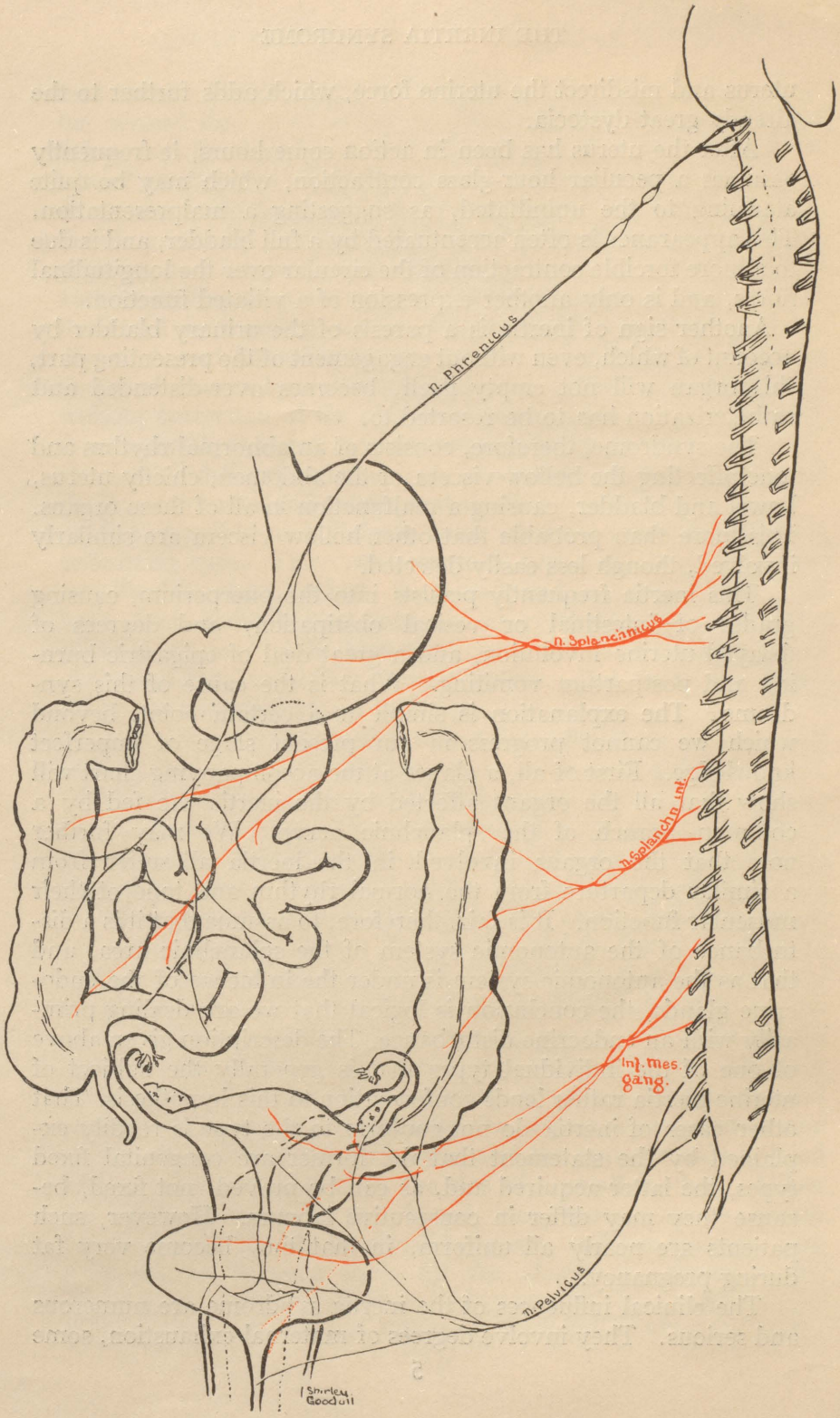

DIAGRAM OF THE SYMPATHETIC AND PARASYMPATHETIC NERVE DISTRIBUTION IN THE ABDOMEN AND PELVIS.

of which are alarming; vomiting, dehydration, acidosis, early spontaneous rupture of the membranes, infected amniotic sac, difficult instrumental delivery, severe birth traumas (which, in the presence of an infected ovisac, spell puerperal infection), intestinal and vesical obstinacy, the use of the catheter and cystitis, and sub-involution due both to infection and inertia. The foetus will die either in the first or second stage, from rapid or slow cumulative asphyxia—caused either by tetanic uterine contraction of an abnormal stimulus, or slow asphyxiation, since the insufficient interval between pains does not allow the child fully to recover its aeration, so that each pain adds its small quota to a a cumulative asphyxia. Furthermore, cerebral haemorrhage of the foetus in the second stage is extremely common, and this is increased in frequency by an extremely difficult delivery with the forceps. Birth traumas are frequent, and post-natal death-rate is inordinately high. The author considers that the inertias of labour constitute in Canada the most common cause of difficult exhausting labours, with the highest percentage of maternal and foetal morbidity and death.

The treatment I suggest does not present anything which is new. During the hours of labour the patient's strength should be maintained by encouraging her to take food frequently and in small quantities. Should vomiting prevent this, dehydration and acidosis should be prevented by the judicious use of intravenous injections of 10 per cent glucose saline in quantities not greater than one injection of 500 c.c., and not repeated more frequently than once daily. Rest and uterine action should be alternated by the judicious use of sedatives and stimulation, and the vagina should be kept sterile by posture and the use of mercurochrome injected intravaginally, under some pressure, so as to force it into all parts of the birth canal.

Manual dilatation of the cervix in the late first stage may be employed and delivery may be effected by any one of the prevalent means. As a rule version and extraction are not often at our disposal, owing to the close co-aptation of the uterus to the child in the many hours of interval between the rupture of the bag of waters and intervention, and the same applies, to a lesser degree, to the Pomeroy and other manoeuvres which require a degree of uterine relaxation which often cannot be obtained in these cases, even under deep surgical anaesthesia, owing to the firm uterine retraction. Manual rotation in mid-pelvis or a double application of the forceps will probably be found to the most common mode of delivery.