UTERINE ACTION IN LABOUR AND A STUDY OF CERTAIN FACTORS INFLUENCING IT

Presented by

Robinson M. Millen, M.B.,Ch.B.(Commendation) Glasgow, 1934

House Surgeon, Kilmarnock Infirmary;
Junior Assistant Medical Officer,
Assistant Medical Officer and
Resident Obstetric Officer,
Stepping Hill Hospital, Hazel Grove, near Stockport,
Assistant Obstetrician and Gynaecologist to the
County Borough of Croydon.

P A R T I

1. Introduction.
2. Arrangement of the Sections.

I INTRODUCTORY

In choosing the above title as the subject of a Thesis, I was impressed in my clinical work by the relative infrequency of severe degrees of uterine inertia, and also, when such a condition did arise, by the catastrophic sequelae to mother and child often found in its train.

Many of the abnormal conditions occurring in labour can be surmounted if good uterine action is maintained, and it is true to say that the majority of catastrophes and undesirable results obtained in obstetrics are due to inefficient uterine action. A contracted pelvis of mild degree presents no bar to spontaneous delivery if the uterine forces are maintained good; a placenta praevia can be effectually controlled by efficient pains, whereas a similar condition not in labour presents one of the most serious emergencies found in obstetrics. And so on, wherever one turns one becomes impressed by the complete helplessness of the accoucheur, and the increased risk to the mother and the child, in the absence of efficient uterine action. It is no exaggeration to say that a normally functioning uterus

is of the most vital importance, and any variation from it must be quickly diagnosed and treated to avoid the many disasters which follow in its wake. Hence a study of uterine action in detail with a study of the many factors having a bearing on its efficient function is of the first importance to anyone embarking on a study of Midwifery.

The Object of the Thesis

The aim of this Thesis is to make a clinical investigation into the action of the uterus in labour and to assess the importance of the various factors which seem to influence uterine action in labour; and in particular to ascertain the cause of uterine inertia when it does arise.

The great majority of the cases have been personally supervised during their antenatal period, so that the cases have been followed throughout their pregnancy and ensuing labour. In this way, it has been possible to ascertain whether or not expected, or possible, aberrations from the normal course of labour have occurred; e.g. in cases of hydramnios, plural pregnancy, fibroid uteri, and so on. During the antenatal period also, it is possible for the clinician to assess such factors as malnutrition, debility, anaemia, psychological habit, and so on, and to endeavour to counteract and improve these conditions in preparation for labour.

The maternity work of this hospital, which is a Municipal Hospital having a maternity unit of 50 beds, is divided into two sections according to the financial means of the patient. One section comprises the "ordinary" patient, of whom a large number, but by no means all, are Public Assistance patients whose home conditions are such that delivery in hospital, away from home environment, is desirable on hygienic grounds; or they may express the desire to have their confinement in hospital, rather than at home - the majority of the latter being mainly primiparæ who are probably apprehensive of the ordeal, and the dangers, of labour.

The other section is designated "private" maternity section. This consists of women drawn mainly from the middle class, whose financial conditions are sufficiently good, and who can afford to pay a moderate fee for confinement in hospital. This section is at present slightly the larger of the two; it comprises patients who would normally be confined at home under quite congenial conditions, but under the present tendency for "hospitalisation" of maternity cases, these patients have come into hospital.

These two sections constitute the "booked" cases. In addition the hospital receives "emergency" cases from the district: the hospital area includes a population of 300,000,

but it verges on the area drained by a large city's (Manchester) maternity hospitals, and outlying cases of emergency frequently go to the nearest maternity unit, irrespective of the district. These emergency cases include the usual obstetrical catastrophes which may or may not have had antenatal supervision. The incidence of such conditions as uterine inertia in "booked" and "emergency" cases can be studied.

Accordingly, the clinical material here affords a good opportunity of comparing the "ordinary" with the "private" cases in labour. The sociological and economical factor can be assessed, with special relation to the condition of nutrition of the mother. The general maternal state of fitness for undergoing labour is an important factor in connection with subsequent uterine action.

To a certain extent physique is partly dependent on the social and economic factor. By physique is implied the state of muscular development and fitness: the stature is taken into account in estimating the value of this factor. The small wiry woman of the poorer classes is not necessarily inferior, as regards physical fitness for undergoing labour, to her more fortunate sister of the middle classes.

In particular the condition of the auxiliary forces of labour viz. the abdominal muscles, has been noted. This is

important in relation to the second stage of labour, and to a much lesser extent the third stage of labour; and although, strictly speaking, one cannot truly say that the state of the muscles of the abdominal wall has any direct relation to the action of the uterus in labour, nevertheless it has a very important bearing on the condition of maternal exhaustion.

Again, the psychological make-up of the woman in labour is of great importance, certain investigators, e.g. Bourne, claiming that the psychological behaviour of the woman in labour has a marked influence on the course of labour, fear not necessarily being manifest but more probably suppressed, the latter being the more serious of the two, as it is more difficult to diagnose and treat. Where any definite psychological condition has arisen its cause and effect have been noted: it includes such mental states as emotion, fear, general nervousness, hysteria, illegitimacy, and so on.

Co-existing maternal disease, whether directly due to pregnancy (e.g. toxaemias, pyelitis of pregnancy, etc.) or merely aggravated by, and not due to, pregnancy (e.g. cardiac or respiratory disease, diabetes, nervous disease etc.) has been studied and its incidence on labour noted. In relation to albuminurias of pregnancy, some special observations have been made on the influence of diet on the duration and nature

of labour.

The age and the parity of the patient have been considered in relation to these general factors, and wherever possible an attempt has been made to compare all these factors in the light of the woman's social and economic status.

The effect on labour of various drugs where such have been used in, or immediately preceding, labour has been observed. In medicinal induction of labour such drugs as quinine, pituitrin, thymophysin, etc. have been used, and their effect on the duration and the nature of the ensuing labour duly recorded, Calcium therapy, too, has been used in the treatment of toxaemias of pregnancy.

The total duration of labour forms a good clinical index of the manner in which the uterus has been acting. Accordingly, in this series of 752 cases the number of hours during which the patient was actually in labour has been recorded as accurately as is clinically possible, without subjecting the patient to unnecessary, and dangerous, interference. The record of labour has been drawn up in the form of a table which will be found at the end of this section, together with a table of general factors under consideration.

II. THE ARRANGEMENT OF THE SECTIONS

Part I: This section is introductory mainly. In addition, attention has been drawn to the comparison which can be made between so-called "private" and "ordinary" patients. The conditions under which the material was collected have been briefly referred to. Finally, the arrangement of the various sections of the thesis is found in Part I.

Part II: Anatomy and Physiology of Uterine Muscle.

(a) Anatomy. i. The anatomy of the uterus has been limited to the arrangement of the muscle layers, with a comparison between the conditions found in the pregnant and in the non-pregnant uterus. The anatomy of the isthmus has been considered in minute detail owing to the important bearing this part of the uterus plays in the mechanism of labour, and in the differentiation into upper and lower uterine segments.

ii. The Nerve Supply of the Uterus. An attempt has been made to elucidate the nerve supply, both extrinsic and intrinsic, of the uterus. The literature on this subject is at present very controversial.

(b) Physiology of Uterine Muscle.

This has involved a study of the properties of smooth muscle in general, and of uterine muscle in particular.

The influence of certain mineral substances e.g. Calcium, Magnesium, Potassium, on uterine contractility has been noted, together with the action of Ergot and its alkaloids, quinine and its salts, acetyl choline, etc. etc.

The relation of the Endocrine Glands to Uterine muscular activity has been investigated in great detail, owing to the recent research which has been, and is still being, carried on in this field. The influence of the various hormones on uterine muscular activity and, where applicable, on the parturient uterus has been discussed and the merits of the numerous endocrine secretions assessed in terms of their practical application.

Part III: A clinical study of the uterus in labour comprises this section. The normally functioning uterus has been studied, and certain independent observations have been made on the degree of pain experienced by different women when in labour, and an explanation has been sought to account for this variation in the painfulness of labour. The state of the uterus in the puerperium does not enter into this investigation.

Part IV: The manner in which the material was collected, and the methods used in investigating the cases in labour, have been given at the beginning of this section, which is in effect an introduction to the case records. A specimen of the form of investigation on which the necessary data was collected, is appended.

The Results of the investigation follow, in tabulated form as far as possible, or else discussed in short paragraphs. Owing to the number of subsections under investigation it has been found more convenient to arrange that the Discussion of Results should follow, instead of leaving the discussion to another separate section. A summary of each factor under discussion is made at the end of each separate discussion of results.

Part V: The conclusions arrived at are found in this part. It is more convenient to arrange the conclusion under separate headings, e.g. the conclusions regarding Age, Parity, Nutrition, Physique, and so on. The Thesis concludes with a list of references used.

P A R T II

Anatomy and Physiology of Uterine Muscle

ANATOMY AND PHYSIOLOGY OF UTERINE MUSCLE

a) Anatomy of Uterine Muscle.

b) Nerve Supply of Uterus.

c) Physiology of Uterine Muscle.

i. Physiology of contraction of uterine muscle.

ii. Influence of certain minerals on uterine contractility.

iii. Action of Ergot and its alkaloids on uterine muscle activity.

iv. Action of quinine and acetyl choline on uterus.

v. Relation of endocrine glands to uterine muscle activity.

(a) ANATOMY OF UTERINE MUSCLE.

Any study of the action of the uterus in labour must necessarily begin with a consideration of how the parturient uterus differs in structure from that of the non-pregnant uterus.

The normal nulliparous uterus is a pear-shaped, muscular organ measuring 7 cms. long by 4 cms. broad, by 2.5 cms. thick. It weighs 42 gms. In multiparae, the uterus is slightly larger from 7.5 to 8 cms. long, 5 cms. broad, and 3 cms. in

thickness, weighing 65 gms. It consists of hard unstriped muscle covered in part by peritoneum, and moored to the pelvis by eight so-called ligaments.

During the first half of pregnancy, the uterus enlarges mainly as a result of hyperplasia of the muscle cells. Hypertrophy of the muscle cells also occurs in the first, but mainly in the latter, half of pregnancy: the stretching of the muscle is no doubt in itself a stimulus to hypertrophy. At term the uterine wall varies from 4 to 7 mm. in thickness; the uterus is 32 cms. in length, 24 cms. in width, and 22 cms. in thickness. Krause estimated that its capacity is increased 519 times. At full term the uterus weighs about 1,000 grammes (about 2.2 lbs).

The muscle fibre of the non-pregnant uterus is spindle-shaped and measures 0.005 mm. in width by 0.05 mm. in length. The muscle fibre of the full term uterus is much larger, being about 2 to 7 times the width and 7 to 11 times the length of the non-pregnant muscle fibre. The muscle fibre in the full term uterus presents in addition a fine longitudinal fibrillation.

The muscle fibres of the cervix undergo hypertrophy to a much lesser extent than those of the fundus. Rarely cross-striated muscle fibres are found in the uterus, probably representing a metaplasia.

With the increase in the number and size of the muscle fibres is associated a marked development of mesenchymal tissue between the muscle bands, particularly in the external muscle layer, which resembles embryonic connective tissue. At the same time there is also an increase in the amount of elastic tissue. The fundus is less rich in elastic tissue than the cervix and it is found especially in the outer layer and around the blood vessels. The greater amount of fibro-elastic tissue in the cervical region is very important when one remembers the stretching and distension which the lower uterine segment has to contend with in labour.

The enlargement of the uterus is not symmetrical but is most marked in the fundal region. This can readily be seen by observing the relative positions of the insertions of the tubes and ovarian ligaments, which in the early months are only a little below the level of the fundus but in the later months are slightly above the middle of the organ.

The Arrangement of the Muscle Fibres.

It is to the work of Hélie, Bayer, and Ruge that we owe the description of the arrangement of the musculature of the uterus. Hélie devoted twelve years to his investigations whilst Bayer worked on the subject for almost forty years.

According to Hélie (Paris, 1864), there are three main layers, each layer having several subdivisions.

1. The outer layer. This is composed of two longitudinal layers between which lies a transverse layer of muscle. It extends from the fundus down to where the peritoneum is attached to the uterus. This longitudinal layer is continuous with the external longitudinal layer of the Fallopian tubes. It is hoodlike, and it arches over the fundus to extend into the various ligaments. It does not cover the uterus at the sides where it is deficient, and where the blood vessels enter. These fibres may, and do, interlace in the middle (representing fusion of the two Mullerian ducts) and probably cross over to the opposite side.

An analogy has been drawn between a layer of muscle found in this external longitudinal coat and the bundle of His - the "pace-maker" of the heart. Purkinjé (1839-1840) demonstrated special systems of muscle fibres in the non-gravid uterus as well as in the heart. His work has been recently confirmed by Hofbauer (1929).(1)

This special system is mainly subserous, a long thin longitudinal coat being distinguished in the upper two-thirds of the uterine body. Loose fibres of this system are found interspersed between ordinary muscle strands on the surface of the uterus. These muscle cells are broader than the ordinary muscle cells and in especially close relation to nerve fibrils. Hofbauer(1) considers that this special layer of muscle cells represents a "conducting system" and that these cells give a

better response to infundibulin than those of the ordinary musculature of the uterus. He suggests that this bundle may act as "pace-maker" to the uterine contractions.

2. The inner layer. The inner layer is also longitudinal, and is continuous with the inner longitudinal layer of the tubes. It lies directly under the endometrium, and is composed of two triangular portions running along the anterior and posterior inner walls of the uterus, and connected by an archiform layer at the fundus. It forms looped bundles of muscle around the tubal ostia and internal os, probably sphincter-like in action.

3. The middle layer. This is much the thickest of all the layers. It is continuous with the circular muscle layer of the Fallopian tubes and the radiating fibres of the utero-sacral, round, and ovarian ligaments. This middle layer consists of a dense net-work of muscle fibres perforated in all directions by blood vessels. Each fibre comprising this layer has a double curve, so that the interlacement of any two gives approximately the form of the figure 8. As a result of such an arrangement it happens that when the fibres contract after delivery they constrict the vessels and thus act as living ligatures.

Ruge (Stuttgart 1880) showed that the muscle fibres overlap each other, like the shingles on a roof, especially in the

lower portion of the uterus, one end of each fibre arising beneath the peritoneal covering of the uterus and extending obliquely downward and inward to be inserted into the decidua, thus giving rise to a large number of muscular lamellae. These muscular lamellae are connected to each other by short muscular processes.

THE CERVIX

The importance of this structure, and the changes it undergoes in the stage of dilatation, merit a close investigation of its anatomical changes preparatory to labour.

Recently Stieve has shown (Leipzig 1927) that the cervical mucosa undergoes such marked proliferation that at the end of pregnancy it occupies approximately one half of the bulk of the cervix, instead of a mere thin layer as normally. Moreover, the muscle fibres, instead of increasing in number like those of the uterus, actually diminish in number, although those which remain increase in size.

Externally the cervix shows a layer of longitudinal and oblique fibres which spread out into the bladder through the utero-vesical ligaments, and into the bases of the broad ligaments. The portio vaginalis has (1) an inner submucous circular layer, derived from the vagina; (2) an outer longitudinal layer, derived also from the vagina; and (3) a middle layer, vascular, derived from the similar middle layer of the body of the uterus, continuous with the ligaments entering the cervix.

The Ligaments of the Uterus.

The ligaments of the uterus are eight in number, four to each half of the uterus, and occurring in pairs. They are -

i. The Round ligaments of the uterus (two).
ii. The Broad ligaments of the uterus (two).
iii. The Utero-sacral ligaments (two).
iv. The Utero-vesical ligaments (two).

i. The Round ligaments extend on either side from the anterior and lateral portion of the uterus, just below the insertion of the tubes, to the upper part of the labia majora, traversing the inguinal canal en route. They are composed of unstriped muscle which is directly continuous with that of the uterine wall, and a certain amount of connective tissue. These ligaments hypertrophy with pregnancy.

ii. The Broad ligaments of the uterus extend from the lateral wall of the uterus to the pelvic walls, and are a double fold of peritoneum. Certain muscular and connective tissue bands pass from the uterus into the broad ligament, but the amount of muscular tissue is negligible.

iii. The Utero-sacral ligaments extend from the upper posterior margin of the cervix, encircle the rectum, and are inserted into the fascia over sacral two and three. They are also composed of connective and muscular tissue, although covered with peritoneum. Their function is to retain the uterus in its normal position by traction on the cervix.

iv. The Utero-vesical ligaments extend from the cervix forwards in the peritoneal folds between the cervix and bladder, and also contain muscular tissue from the cervix.

The Isthmus Uteri.

In view of the important relation between the isthmus uteri and the formation of the lower uterine segment during labour, it is instructive to note that certain anatomists do not recognize the existence of a part of the uterus known as the isthmus, e.g. Gray's Anatomy 1930. In modern times Aschoff (1906) has contributed greatly towards the delineation of the isthmus uteri: he named the upper limit of the isthmus (between body and isthmus) "orificium uteri internum anatomicum", and the lower limit (between isthmus and cervix) "orificium uteri internum histologicum". The upper anatomical limit corresponds more or less accurately with the point of attachment of the peritoneum and the point of entry of the uterine artery: the lower histological limit depends on essential histological differences - according to Aschoff the limit of the mucous membrane of the cervix lies 8-10 mm. deeper than the internal os (orificium internum uteri).

Frankl,[2] Professor of Obstetrics in Vienna, claims to be able to recognize the isthmus macroscopically. It manifests itself sometimes as a low fold, sometimes as a shallow furrow. The changes in the mucous membrane in the pre-menstrual non-

pregnant, and in the pregnant, uteri enable the difference to be easily seen by the naked eye. The mucous membrane of the isthmus is thinner, and less vascular: there is no obvious decidual reaction in the isthmus. Microscopically the main features of distinction are (1) the thinness of the mucous membrane; in the lower uterine segment in later months - no decidua compacta; (2) the greatly diminished number of glands, which in the pregnant uterus, are poor in glycogen, as compared with the glands of the body; (3) the characteristic arrangement of the glands which have a general direction downwards towards the os, as compared with the glands of the body which are either vertical or have a direction upwards towards the fundus.

With pregnancy and its accompanying decidual reaction, the isthmus becomes easily demarcated: there is no obvious decidual reaction in the isthmus-only a decidua spongiosum. From the second month onwards the upper limit of the isthmus begins to disappear, becoming no longer recognizable in the third month. The whole isthmic portion of the womb is drawn into the ovum chamber. Because of the growth of the uterus this originally narrow portion of the uterus becomes extended - only so far as the orificium internum histologicum of Aschoff, i.e. the internal orifice of the gravid uterus. The so-called lower uterine segment develops from the isthmus. The so-called

contraction ring (probably more accurately described as the retraction ring) of the gravid uterus coincides with the orificium isthmi anatomicum of Aschoff; and is also termed "Braune's ring" because he first described it: also Bandl's ring, because he first showed its clinical importance in dystocia. It represents the division between the thick muscular layer of the body of the uterus and the thin muscular layer of the lower uterine segment, and can be demonstrated during lower segment Caesarean section as a thick transverse ridge of muscle.

(b) THE NERVE SUPPLY OF THE UTERUS.

The nerve supply to the uterus is very abundant and hence very complicated. Physiologically, the function of the nerve supply to the uterus would seem to be regulatory rather than primary, since in experimental animals labour can progress normally after all the nervous connections have been severed. The human uterus will contract rhythmically and strongly for many hours after excision if immersed in warm Locke's solution, and plentifully supplied with oxygen.

It is convenient to study the nerve supply under the headings of -

I. Extrinsic nerve supply, which includes

(a) autonomic nervous system (sympathetic)
(b) cerebrospinal nervous system (parasympathetic)

and II. Intrinsic nerve supply.

The autonomic nervous system forms the main nerve supply to the uterus. Whitehouse[3] has shown that the sympathetic nerves stimulate the circular muscle fibres of the fundus and cervix to contract, and inhibit the longitudinal muscle fibres. The cerebrospinal nervous system, via the pelvic nerves, has the opposite effect.

I. Extrinsic Nerve Supply.

(a) Central connections. Unknown or imperfectly understood.
(b) Peripheral connections.
    (i) Abdominal root - sympathetic supply.
    (ii) Pelvic root - parasympathetic supply.
    (iii) Fusion of i and ii in pelvic (hypogastric) plexus.

(i) The abdominal autonomic system supplies the pelvic (hypogastic) plexus via the solar plexus, the renal plexus, the intermesenteric plexus (superior and inferior mesenteric plexuses) and the superior, middle, and inferior hypogastric plexuses, in that order from above downwards. The superior hypogastric plexus is also known, rather unfortunately, as the "pre-sacral nerve" - it is more often a plexus, or right and left intercommunicating plexuses, than a single nerve (10% only), and it is pre-lumbar (lumbar 4th and 5th) rather than pre-sacral in situation.

The superior hypogastric plexus, or pre-sacral nerve, merges below into the middle hypogastric plexus, lying in front or just below the front of the sacrum. The inferior hypogastric plexuses (hypogastric nerves of Latarjet) arise from the

extremities of the lower border of the middle hypogastric plexus, or, in the absence of the latter, as the direct continuation of the bifurcated pre-sacral nerve or plexus. Each inferior hypogastric plexus, or hypogastric nerve, runs downwards and outwards to enter the corresponding utero-sacral fold, and passing in the fold lateral to the rectum, finally enters the postero-superior aspect of the pelvic plexus. This forms the abdominal sympathetic root of the pelvic plexus (ganglion of Frankenhauser).

(ii) Pelvic Roots consist of -

(1) Nervi Erigentes.
(2) Twigs from sacral sympathetic ganglion.

(1) Nervi Erigentes arise from cerebrospinal system, vary in number from two to four and arise from the anterior primary divisions of the intermediate sacral nerves. They (nervi erigentes) pass into the utero-sacral folds and then run forwards and laterally to enter also the pelvic plexus on the medial aspect.

(2) These fine twigs from the sacral sympathetic ganglia pass to the posterior border of the pelvic plexus.

The pelvic root (or pelvic nerve) is parasympathetic in action.

The Pelvic Plexus, also known as the lateral cervical plexus, utero-vaginal plexus, cervical ganglion, or plexus of

Frankenhauser, lies on the lateral aspect of the ampulla of the rectum, which is clasped, as it were, between the pelvic plexuses on either side of it. The nerves to the uterus leave the anterior border of the pelvic plexus along with those to the bladder and vagina, which latter they soon leave, to pass as a thick bundle within the utero-sacral folds to the cervix, where they lie medial to the crossing of the uterine artery and ureter. The nerves then enter the uterine musculature in which they terminate.

The "lateral nerve of the uterus" arises from the inferior hypogastric plexus directly, without passing through the pelvic plexus and passes horizontally forwards to the uterus.

The "para-cervical ganglia" described by Luschka, Koch, Heale and recently by Gemmell[4] represent offshoots from the main pelvic plexus.

Branches from the pelvic plexuses supply the uterus, bladder, and upper part of vagina. Some terminate by free endings between the muscle fibres, while others make their way towards the free surface of the endometrium.

(a) The Central Connections. The centres for the afferent pathway probably lie in the posterior root ganglia. The centres for the efferent pathway lie in the intermedio-lateral tract in the lower dorsal and lumbosacral regions (sympathetic) and the medioventral columns in the sacral

region (parasympathetic). The exact anatomy is still very obscure.

II. The Intrinsic Innervation of the Uterus.

This must necessarily depend on the histological study of the uterus. In 1840 Remak first demonstrated nerve fibres in the uterine substance. Frankenhauser in 1867 found both myelinated nerves along the blood vessels, and non-myelinated fibres passing to the smooth muscle and to the cells of the mucous membrane. Herlitzka on the other hand, in 1897, showed three types of nerve in the wall of the uterus (1) Non-myelinated nerves to the blood vessels; (2) myelinated nerves (sympathetic) to the muscles; and (3) cerebrospinal nerves with Ranvier's nodes ending intracellularly.

Intrinsic Nerve Supply. Dahl[5] more recently has demonstrated the rich intrinsic nerve supply, both medullated and non-medullated, among the muscle fibres and along the blood vessels. The cervical musculature shared in this general richness. The muscular nerve endings were cone-shaped or button-shaped. Fibrils were traced to the mucosa, though not into the cells, and there was no important change in the nerves in the uterus with pregnancy.

As to the occurrence of "ganglion cells" in the uterine musculature, recent work by Davis[6] tends to discount their existence, except in the subepithelial region of the vaginal portion of the cervix, which is supplied by the cerebrospinal

system and associated with sensation: elsewhere they are not present in any degree of constancy.

Although no authentic ganglion cells have been demonstrated, physiological experiments tend to show that the uterus does have some intrinsic ganglionic system, or else that uterine muscle has inherent power of contraction analogous to cardiac muscle. It has been shown by Sun and Flury[7] that excised strips of pregnant and non-pregnant human uteri will contract spontaneously and rhythmically for many hours in Locke's solution: these strips have no nervous connections. In addition others have shown that labour may terminate uneventfully in experimental animals in whom all nerves to and from the uterus had been severed.

From a consideration of these facts it would appear that the function of the nervous connections of the uterus must be regulatory rather than primary.

(c) PHYSIOLOGY OF UTERINE MUSCLE.

Most unstriped muscles under suitable conditions show spontaneous rhythmic contractions which vary in frequency from one (in spleen) to twelve (small intestine) per minute. It is fair, in the present state of knowledge, to assert that nobody knows how these tonic and rhythmic activities in plain muscle are brought about. A distinctive feature of all

unstriped muscle is the extreme sluggishness with which it responds to mechanical excitation as compared with the quick movements of skeletal muscles.

The excitability of smooth muscle varies according to the nature of the stimulus. Most agents which excite skeletal muscle are also effective with smooth muscle, but the latter is much less sensitive to electrical stimulation. On the other hand unstriped muscle is much more sensitive to chemical stimulation: this contributes to the performance of the normal slow, purposeful, functions of visceral muscle, which can be controlled in part by substances circulating in minute amounts in the blood: skeletal muscle, which requires to be quick in its effect, is practically unaffected by low concentrations of such chemical substances.

In regard to the uterine muscle, it is developmentally plain muscle which has acquired specially highly developed functions in the higher mammals: in the lower mammals, with tubal uteri, the uterine muscle approximates more to what one understands by plain visceral muscle: uterine muscle therefore differs fundamentally in no way from ordinary visceral muscle, and is subject to the same laws of plain muscle contraction as the plain muscle, say, of the intestine.

The relation between chemical substances in the blood, and their effect on plain muscle contraction is very well

exemplified in the case of the uterus. In recent years uterine muscle has been subjected experimentally to various alterations in environment, and its reactions studied. Especially so is this the case in regard to the hormones secreted by the pituitary and ovarian glands of internal secretion, the experimental findings of which will be found in another section (Endocrines). Again, the action of salts of calcium, potassium, etc., on uterine activity shows the important part played by the blood constituents in modifying the normal function of the muscle.

"Tension" in relation to production of pain.

A further function which uterine muscle has in common with other visceral muscle, is its great capacity to alter its length - it may shorten to a quarter and less of its extended length, while its capacity for developing tension is very small. If smooth muscle in general is induced by an abnormal stimulus to develop any considerable tension it is accompanied by the intense pain of "spasm" e.g. colic in intestine. Here then, one may find a part explanation of the difference between pregnancy and labour uterine contractions: in labour the stimulus (probably hormonal but supplemented by local nervous reflex) is very much more powerful, and corresponds to an abnormal stimulus, say, in the small intestine. The result of this powerful stimulus acting on the smooth muscle of the

uterus is to produce extreme "tension", and the subjective result is intense pain. Again, if the uterus is subjected to extreme distension, with a corresponding increase in tension, the result is severe pain: this is found clinically in concealed accidental haemorrhage. This relation has been attributed to internal frictional resistance in the muscle analagous to viscosity.

It is an axiom of experimental physiologists that the more quickly a stimulated muscle is allowed to shorten i.e. to contract, the less is the tension it develops: conversely the more slowly a stimulated muscle contracts, the greater the tension. It has been stated previously that the contractions of smooth, and especially uterine, muscle are slow and deliberate - and hence it follows that there is a greater production of tension developed in the muscle. Luckily, smooth muscle has less capacity for producing tension than striped skeletal muscle, and this tension is only developed under response to exaggerated, abnormally powerful, stimuli such as are found in labour contractions: otherwise, were tension to be produced in smooth muscle functioning under normal stimuli, we would be uncomfortably aware of intestinal peristalsis. As it is, abdominal colic is only produced in response to powerfully abnormal stimuli with resultant increase in tension.

Hence in regard to the causation of uterine pains may be

advanced the theory that they may be in part due to extreme tension produced in the muscle cells contracting rhythmically in response to powerful stimuli. The contractions of pregnancy are not appreciated as pain, just as normal intestinal peristalsis is not appreciated as pain, because the tension produced by these contractions is sub-liminal, i.e. insufficient to give rise to painful impressions. Undoubtedly, the compression by uterine contraction of the terminal nerve twigs in and around the muscle bundles also takes part in the production of the "pains"; but from a study of the physiology of muscle contraction the above theory is tenable, and may contribute in great measure towards the production of "labour pains". I have not been able to find any support for this theory in any of the standard textbooks of obstetrics, and merely advance it as an application of accepted physiological principles of smooth muscle contraction in general to uterine muscle action in particular.

Muscular Tone.

"Tonus" has been defined in its simplest form[8] as "that disproportion between tension and length", which is a relatively permanent characteristic, and may be regarded as a sustained sub-maximal contraction. The meaning of muscular tone has been extended to include that small permanent state of tension which enables skeletal muscles, for example, to maintain the

postural relations of the body (provided the nervous paths are intact). The longer the muscle, in proportion to its sustained tension, the lower is its tone: hence overstretching of muscular fibres in general leads to an increase in length without corresponding increase in tension and subsequent lowering of tone.

(ii) The Influence of Certain Minerals on Uterine Contractility

Calcium. It has long been known that the presence of Calcium salts is essential to the maintenance of the tone and contractility of involuntary muscle. During pregnancy a hypocalcaemia is present (7 mgs/100 c.c.s or less) as shown by Heller and Holtz[9] and others. The former experimenters also found that the isolated uterus of the guinea pig contracted better, and showed more response to pituitrin, when there was a reduction in the calcium-potassium ratio in the bathing fluid. This finding runs counter to such authorities as Blair Bell,[10] who has suggested that a reduction of the calcium content of the blood below the optimum may be a factor in the causation of primary inertia. Later work by Blair-Bell[11] tends to show that uterine muscle activity is diminished by the withdrawal of calcium from the solution bathing the muscle in vitro: the tone of the muscle and response to infundibulin are similarly depressed. In addition he showed that increase or decrease of the potassium content, within reasonable limits, produced no consistent alteration in uterine movement. On

investigating the problem on living animals, some of which received intravenous injections of 5 per cent $CaCl_2$ and others the same proportion of potassium chloride, discordant results were obtained, and in the same animal one injection was found to inhibit and another to stimulate contractions.

From these equivocal results one hopefully turns to other conditions for help on the problem. Undoubtedly calcium ions are essential for the maintenance of muscular tone and contractility: there is an optimum value when the muscle will give of its best. Increased muscular tone is found in tetany due to parathyroid deficiency e.g. after total thyroidectomy, or in parathyroidectomised animals: the blood calcium is then critically low. Also in rickets - there is a hypocalcaemia and there results the muscular spasms. Lastly in pre-eclamptic conditions the calcium content of the blood serum is said to be critically low, the muscular tone is greatly increased so that a subminimal stimulus will evoke a maximum response, and when this critical level is passed the patient has eclamptic seizures. In all these conditions, then, the blood calcium is lowered and the muscular tone greatly increased: but these other clinical conditions involve skeletal muscle, not smooth muscle. Is uterine muscle different in action from skeletal muscle in relation to calcium content? The literature on the subject is rather confused at the moment. It seems logical to conclude that increased muscular tone of tetany and eclampsia

depends on a hypo- rather than a hyper-calcaemia, although one cannot overlook the fact that the relatively high value of the potassium salts (vide infra) may in part account for the increase in excitability, since excess of potassium increases contractions.

Potassium. Blair-Bell claimed that alteration of the potassium content produced no consistent alteration in uterine movements: but in muscle activity in general it has been found that excess of potassium salts induces contraction of most muscles other than the heart. It is found as a constituent in such physiological muscle bathing solutions as Ringer's, Locke's, and Tyrode's solutions; these solutions were only adopted after trial and error, potassium (in the form of potassium chloride) being a desirable but not essential constituent.

Potassium-Calcium Ratio. Salts of potassium and calcium would seem to be interdependent on each other; they must be present in the correct ratio (though their absolute amounts may vary considerably) to give optimum muscular activity. The proportion of potassium to calcium in the blood plasma is $\frac{20}{9}$. If the calcium is dangerously reduced, the relative value of the salts of potassium is correspondingly increased: the increased muscular tone may be due in part to the relatively high potassium value.

Magnesium. Magnesium salts (e.g. Mag.Sulph.) are known

to control the convulsions of eclampsia: it has also been advocated as an analgesic in labour. Blair-Bell recently confirmed the work of Fontes[12] who showed that the addition of magnesium to the isolated uterus inhibited all movement.

Sodium. Sodium salts must be present in much larger numbers than any others for effective muscle contraction. It is present mainly as chloride, but also as carbonate and phosphate.

The Hydrogen Ion Concentration must of course be slightly on the alkaline side. Any increase in acidity causes "slowing of muscular contraction and relaxation of the tone of most unstriped muscles" (Winton and Bayliss[13]).

(iii) The Action of Ergot and its Alkaloids on Uterine muscle activity.

Ergot was the first authentic oxytocic drug introduced into obstetrics, and the medicinal properties of this fungus, which attacks rye, and the consumption of which resulted in widespread epidemics of "ergotism", was known to European midwives. Its introduction into obstetrics is ascribed by American writers to John Stearns of Saratoga, who popularised its use in obstetrics in 1807.

Mode of Action of Ergot. Ergot preparations were said to act on the uterus by causing prolonged firm contractions of the uterine muscle. Recent detailed study of mechanically recorded

tracings of uterine activity show that in the dosage used in clinical work this does not accurately describe the mode of action. The essential fact is the production of a rapid series of contractions which, after sufficient dosage, follow each other so closely that the uterus as a whole has no time to relax, and a state of spasm therefore results. Great muscular activity continues throughout this stage. As this initial, almost tetanic, stage passes off, the contractions again become spaced out and the uterus relaxes completely between successive contractions. This second stage persists for 3 to 4 hours, the initial stage of spasm lasting only 5 to 6 minutes.

The Alkaloids of Ergot. Of the many alkaloids of ergot described only three have survived investigation. In 1875 Tauret isolated ergotinine from crude ergot, but this alkaloid proved to have no oxytocic properties. In 1906 Barger and Carr isolated ergotoxine, and in 1918 Stoll prepared ergotamine. Two other alkaloids are described viz. sensibamine and ergoclavine, but little authentic work has been done on the latter drugs. Thus, until recently, only two of the alkaloids of ergot were known to have oxytocic properties (a) Ergotoxine and (b) Ergotamine.

In 1932 Moir and co-workers began work on isolating an alkaloid of Ergot which was isolated by Dudley in 1935[14] and

to which the name ergometrine has been given, although American workers later isolated the same alkaloid and gave to it the name "ergonovine".

Ergometrine differs from ergotoxine and ergotamine in that, although they all have the same basic physiological action on the uterus, ergometrine is much quicker in action and more effective, being probably the main oxytocic alkaloid of ergot. Given by mouth its effect will appear in seven and a half minutes, after intramuscular injection in three and a half minutes, and after intravenous injection in 45 to 60 seconds. The other alkaloids - ergotoxine and ergotamine - have no action if given by mouth, and are unreliable when given parenterally.

Owing to its physiological action producing an initial state of tetany of the whole musculature, the alkaloids of ergot have no place in labour till the third stage is completed, owing to the great danger of foetal asphyxia. It has the advantage then that the dose can be safely repeated, where pituitrin in repeated dosage has been known to cause a condition analogous to shock and possibly due to constriction of the coronary vessels in the heart muscle.

Ergometrine given before labour, in clinical dosage, has no effect on uterine contractions and only acts when labour has begun; nor has it any action on the non-pregnant uterus.

It has therefore no place in the medicinal induction of labour.

(iv) (a) The Action of Quinine on Uterine Muscle.

Quinine, an alkaloid of cinchona bark, has maintained a wide clinical popularity. It has been used to augment weak, ineffective labour pains, but more commonly in conjunction with castor oil in the induction of labour. Its oxytocic action is very mild and unreliable, and in very large doses may even be harmful to the foetus. Quinine sensitizes the pregnant uterus at or near term so that it becomes more responsive to the more potent oxytocics.

Ganner[15] found that the antenatal administration of quinine accelerated the first and second stages of labour by strengthening uterine action: premature labour was less likely, but inertia was not entirely abolished, though lessened. The dose was 1 to 2 grains daily from the 36th week onwards. Other investigators[16] claim that quinine given antenatally in small daily dosage renders the first stage quick, easy, and often painless and imperceptible.

Quinine would therefore appear to act as a tonic to uterine muscle, reinforcing the basic tone of the muscle cell. This improvement in tone results in more efficient and strong contractions in labour. Its 'oxytocic' action is probably negligible, and its action mainly to improve the tone of uterine muscle. Most observers are agreed that quinine in

large doses (over 5 grains) paralyses uterine muscle, and in small doses (one to two grains) renders reaction more sensitive and powerful.

(b) The Action of Acetyl Choline on Uterus in Labour.

It has long been known that acetyl choline is concerned with parasympathetic stimulation. Dale[17] found acetyl choline in most organs of the body and later suggested that it was carried in the blood stream in the red blood corpuscles in an "inactivating and protective complex", being only given up at the call of the parasympathetic. To support this, Dale found that acetyl choline could be isolated from red blood corpuscles in minute traces.

Recently it has been shown that acetyl choline is a constant constituent of the placenta (Walker and Henderson[18]) and these workers claim that in those patients having a short labour the acetyl choline is higher than in those having a long labour.

Certain derivatives of acetyl choline are therapeutically more active than acetyl choline itself.[19] Of these Mecholyl - acetyl-beta-methyl-choline - has been used with success, having a more prolonged and constant effect, and recently uterine inertia has been treated with this substance with, it is claimed, definitely good results.[20]

(v) The Relation of the Endocrine Glands to Uterine Muscular Activity.

Ovarian Internal Secretions.

Two hormones are now recognized to be produced in the ovary:

i. Oestrin, or follicular hormone, found in the fluid of the ripening follicle.

ii. Progestin, or corpus luteum hormone, found in the corpus luteum of the ovary.

i. Action of Oestrin on Uterine Muscle.

a) Oestrin has been shown to lead to hyperplasia and hypertrophy of the uterine muscle cells, with a consequent increase in size and muscularity of the uterus. The ability of oestrin to bring about rapid growth of the mature or immature uterus is now an axiom of endocrinology.(21) During pregnancy in the human subject an increase in the actual number of muscle cells occurs only in the first three or four months; in the later months the uterine enlargement is due to hypertrophy of the individual muscle fibres along with some stretching of muscle, which aids hypertrophy.

b) Oestrin sensitizes the uterus to the action of posterior pituitary hormone - oxytocin; it improves uterine tone.

The uterus which has been first subjected to oestrin injections is capable of the response to oxytocin to the extent required to cause abortion.(22) This capacity to respond is

in marked contrast to the insensitivity of the normal pregnant uterus to oxytocin. It is possible therefore that oestrin acts on the muscle fibres, or on the nerve elements in connection with them, in such a way as to enhance response to stimulation by oxytocin. Oestrin, then, prepares the uterine muscle for the forceful, regular contractions of parturition by sensitizing the muscle to posterior pituitary hormone. The fact that the oestrin content of the blood and its excretion in the urine gradually increase during pregnancy, reach a maximum at parturition, and thereafter rapidly decrease following expulsion of the placenta provides proof of the parallelism between the reactivity of the uterine muscle and sensitization by oestrin.

That this sensitization is due to oestrin is shown by the fact that the administration of oestrin to oophorectomised animals (rabbits) leads to a degree of uterine muscle reactivity to oxytocin similar to that observed at parturition.[23] Oxytocin by itself will not induce abortion, nor will oestrin alone: a combination of the two seems to be necessary for the establishment of the uterine muscle changes preparatory to parturition. Recently, however, Robinson, Datnow and Jeffcoate[24] claim that, in humans, the administration of oestrin is an excellent method of inducing labour in cases of missed abortion and intra-uterine foetal death, and that it is

successful in 80% of such cases. In other cases it is unsuitable for immediate induction of labour, an onset of 7 to 8 days elapsing between the onset of treatment and the commencement of normal expulsive contractions. Moreover the expense of this method of induction is at present prohibitive, the average dose found necessary being 2,000,000 units (international).

Unlike the inhibitory hormones of the corpus luteum and of the gonadotropic group, oestrogenic substances in moderate dosage do not influence uterine contractions until 6 to 12 hours have elapsed, and the maximum effect is not apparent until at least 20 to 24 hours after an injection. The effect of the inhibitory hormones is obtained in a few hours, and even within 15 to 30 minutes when large doses are used.

The American workers Falls, Lackner, and Krohn,[25] however, by using very large doses (40,000 units) of oestrin - progynon-B Schering - have obtained a response in the human uterus within 1 hour. The method of Chassair Moir, using the intra-uterine hydrostatic bag, was used and the patients were seventh day lying-in women. Using Progynon-B (Schering) they found that 10,000 rat units intramuscularly led to no change; 20,000 units caused a moderate increase in the intensity and frequency of the contractions; 40,000 units precipitated a tetany one hour after administration which lasted for 5-10

minutes. This dosage is considerably less than that found necessary to produce an effect by the Liverpool school of workers.

Still more recently, Jeffcoate of Liverpool[26] has used oestrin in labour itself for the treatment of primary inertia, as well as for the induction of labour in certain cases (missed abortion, intra-uterine foetal death etc.); and he claims a successful result of 50% of cases of true primary inertia. The dosage is very high - 20,000 international benzoate units 4-hourly for a total of ten injections - 200,000 international benzoate units, or 1,000,000 international units. The therapeutic uses of oestrin in labour are still on trial, however, although it appears to merit a definite place in treatment of primary inertia, in its prevention as well as its cure, by improving uterine tone.

c) During pregnancy, oestrin is neutralised or inactivated by some as yet unknown factor. This is obviously desirable for the prolongation of gestation, otherwise the stimulating action of oestrin on uterine muscle activity would lead to abortion, miscarriage, or premature labour.

In the early months the corpus luteum has been shown to inhibit uterine activity so that the uterus is rendered insensitive to oxytocin stimulation; but in the later months the corpus luteum has degenerated and its secretion become

inactive. Recently Cohen, Marrian, and Watson[27] have proved that 99% of oestrin excreted in pregnancy urine is in a physiologically inactive form; and not until a few days before labour sets in do oestriol and oestrone (chemically pure crystalline oestrin) appear in the urine and replace the inactive esters. What, then, renders oestrin inactive during pregnancy?

Some writers have it that oestrin during pregnancy is neutralised by prolan.[28] The secretion of prolan in pregnancy is also high; its concentration in the later months increases pari passu with oestrin content. Dahlberg has found it possible to prevent the Zondek-Aschheim test of pregnancy (which depends on the presence of active prolan B) by adding follicular fluid (containing oestrin) to urine from women who are known to be pregnant. If the follicular hormone does not directly neutralise prolan B it is difficult to understand why ovulation does not take place during pregnancy. This evidence has been adduced to substantiate the claim that prolan B and oestrin are antagonistic during pregnancy. In the early months, prolan (probably prolan B) assists the inhibitory action of the corpus luteum on the uterine muscle. With the degeneration of the corpus luteum in the later months, prolan takes up the inhibitory action and antagonizes the stimulating action of oestrin by possibly rendering it inactive, or directly neutralising it.

Still more recently it has been suggested that the placenta may produce progestin-like substances, but experimental proof of this is still lacking. If they are proved to exist they will undoubtedly exert their antagonistic action on the oestrin content of the blood.

ii. Action of Progestin on Uterine Muscle.

As far back as 1912, Ancel and Bouin suggested that the corpus luteum inhibited uterine activity. In 1927 Knaus[29] confirmed this conclusively in the rabbit, using posterior pituitary extract to excite uterine activity. Other workers since then have verified the inhibitory action of the corpus luteum hormone on uterine activity in the human as well as in experimental animals; Robson[30] in using progesterone in the treatment of albuminurias, quotes a case which, after a preliminary course of progesterone injections, had labour induced by rupture of the membranes, the patient not being in labour even after an interval of 5 days - and then only after the exhibition of pituitrin.

The hormone secreted by the corpus luteum, to which the name progestin has been given, produces specific changes on (1) endometrium, (2) uterine muscle. It is suggested by Robson that this is due to two different hormones, the first being "secretory" and leading to the proliferative changes in the endometrium (pre-menstrual changes in the human, pseudo-pregnancy changes in lower animals); the second is "inhibitory"

to uterine activity.

i. Progestin normally antagonizes the action of oestrin. T.N. Morgan[31] has shown that in immature rabbits injections of oestrin caused increased uterine activity, oestrous changes in the endometrium and increase in the size of the uterus. Injections of corpus luteum extract caused decrease and finally quiescence of uterine activity and pseudo-pregnancy proliferation of endometrium. Cessation of corpus luteum injections was followed by a return of uterine activity and regression of endometrial changes. Identical results were obtained in sexually immature rabbits from which the ovaries had been removed, and also on sexually mature oophorectomised rabbits. On the other hand these results were not obtained in sexually mature rabbits with intact ovaries, corpus luteum extract injections even in large doses not being shown to produce quiescence of uterine activity etc. These results are due to the presence, in the mature rabbits with intact ovaries, of a supply of endogenous follicular hormone which has to be counteracted before the inhibitor effect of the corpus luteum hormone becomes apparent. This antagonism between oestrin and progestin is important in therapeutics and probably accounts for the failures in the treatment of threatened abortion etc. by corpus luteum extracts, the endogenous store of oestrin being high.

ii. In addition to this antagonism, there exists a

synergism between oestrin and progestin. Morgan has proved that progestin is unable to produce its effect on rabbits in the absence of oestrin. Other observers have confirmed this synergistic relation.[32] Robson also maintains that a certain amount of oestrin is necessary for the corpus luteum secretory and inhibitory changes to occur. He (Robson) holds that the amount of oestrin necessary for the effective action of the inhibitory hormone is greater than that required for the endometrial effects of the proliferative hormone.

The corpus luteum hormone probably takes no part in determining the onset and the character of labour, since its effect decreases from mid-term onwards. Its action seems to be limited to the early months of pregnancy, although its use therapeutically near the onset of labour has the effect of delaying the onset of uterine contractions (Robson[30]). The corpus luteum hormone is therefore antagonistic to the action of the oxytocic hormones - pituitrin (oxytocin) with its sensitizing oestrin.

The inhibitory action of the hormone of the corpus luteum on the human uterus has also been demonstrated by Falls, Lackner and Krohn[25] in America. In the same series of experiments on lying-in women they found that very small doses - 1 rat unit of progestin (Corner, not European, units) inhibited uterine activity in the great majority of their

cases. The effect was rapid, beginning in 5 to 10 minutes and lasting for 2 to 3 hours.

Others have used progestin in the treatment of "after pains";[26] one rabbit unit acting within 15 minutes in nearly 90% of cases but "it is extremely doubtful whether it is wise to allay muscle contractions whose purpose is to expel blood clot and necrotic shreds of decidua and membrane from the uterine cavity" (Jeffcoate).

In threatened abortion good results are claimed by the use of progestin: and also in habitual death of the foetus.

Like oestrin, the use of progestin in pregnancy has definite indications; but, unlike oestrin, it is extremely doubtful if it plays any part in the physiology of normal labour.

The Action of the Pituitary Hormones on the Uterine Muscle.

The pituitary gland is now recognized to be the "leader of the endocrine orchestra". It has now been shown that a normally functioning pituitary gland is absolutely essential to a normally functioning ovary and uterus. It has been claimed by Morgan[31] that the pituitary effect is dependent on the integrity of the ovary, pituitary oxytocic factor having no effect on the oophorectomised animals. How far this is true of humans, however, is extremely doubtful, as labour can be completed successfully and normally in the absence of both

ovaries (e.g. where the ovaries have been removed by operation during pregnancy); but it is probable that the placenta is the source of the oestrin present in the blood, and not the ovary, during pregnancy. Hence it would appear that the ovaries are not essential for parturition to take place, but the pituitary gland is essential to normal parturition. Hypophysectomy, it may be noted, is followed (according to Cushing and others) by atrophy of the uterus and ovary in mature animals.

The effect of the pituitary on the uterus varies according to the source of the hormone, i.e. whether anterior or posterior lobe hormones are being considered.

1. Anterior Lobe (Gonadotropic) Hormones.

Certain of the hormones of the anterior lobe are gonadotropic in action. Two gonadotropic hormones are described:

i. Prolan A: an acid extract of anterior lobe, which induces maturation of the follicles and ovulation, i.e. it stimulates the formation of oestrin.

ii. Prolan B: an alkaline extract of anterior lobe which stimulates luteinisation of the follicles, without necessarily previous rupture and ovulation.

According to Morgan, these two hormones are the same in essence and only differ in dosage: i.e. they are qualitatively the same and differ only in their quantitative effect.

Recently Evans in California,[33] and Hamburger in Copenhagen, have described a third gonadotropic hormone found

in the serum of the pregnant mare; it has been called "antex". This "antex" powerfully stimulates the ovary and is more powerful than pituitary or urinary prolan. It differs from the hypophyseal hormone in that its injection into animals is not followed by its secretion in the urine.

Hain,[34] as far back as 1932 showed that the gestation period in rats may be prolonged from four to ten days by extracts of urine from pregnant women and of alkaline pituitary extract (prolan B). He suggested that pregnancy was not maintained solely by the corpus luteum, and that the inauguration of parturition was not alone due to the degeneration of that body. The relation of prolan to oestrin and progestin has been discussed elsewhere (see section on Oestrin and Progestin).

It has to be remembered, in dealing with comparative physiology, that the gonadotropic extracts in therapeutic use are obtained not from the pituitary gland, but from the placenta and pregnancy urine - the active principle of the latter being chorionic in origin. Although it is similar to the type that can be prepared from chemical treatment of the pituitary, there is an important difference in their physiological action. The one obtained from the pituitary can obtain both maturation of the ovarian follicles and subsequent increased uterine activity, followed by luteinisation

in an animal deprived of its own pituitary; while the chorionic preparation cannot do either under the same conditions (Collip[35]). This is not in entire agreement with the earlier work of Morgan who also had investigated the action of gonadotropic hormones on uterine activity. Small doses - 16 rat units - of gonadotropic preparations (Antuitrin "S", Pregnyl, and saline extracts of anterior pituitary) were all found to stimulate follicular enlargement; larger doses - 60 rat units - to produce ovulation and subsequent luteinisation leading to quiescent uterine activity eventually, and changes of pseudo-pregnancy. No such changes in the uterus occurred in oophorectomised animals, indicating that gonadotropic extracts of pregnancy urine have no effect on the motor activity of the uterus of castrated animals and only exert their influence through the intact ovary.

Hence it would seem that gonadotropic hormones, to exert their influence on uterine muscle activity, must have (a) intact ovaries, and (b) intact pituitary gland. Given these essentials, gonadotropic hormones (anterior pituitary and pregnancy urine extracts):

i. Cause uterine muscle quiescence in moderately large dosage, probably through secondary corpus luteal influence.

ii. Cause uterine muscle activity in very small dosage, probably through oestrin production (without necessary ovulation).

The gonadotropic effect on uterine muscle is therefore obtained indirectly.

2. The Posterior Lobe of the Pituitary.

The extract of the posterior lobe of the pituitary gland (known variously as pituitrin, infundibulin, oxytocin) has long been known to have a very powerful oxytocic effect on the uterus, being first discovered and investigated by H.H.Dale in 1908, and later investigated by Blair-Bell in 1909.(36)

Physiologically, pituitrin causes an increase in tone of the uterine muscle and stimulates the uterus to contract and retract more forcibly, and with better rhythm. In 1918 Cow(37) observed that rings of muscle in the lower (vaginal) end of the uterus often relaxed under the influence of infundibulin. More recently Robson(38) has shown that the effect of this hormone on the lower uterine segment of the human uterus is very much less than it is on the fundal portion. In 1925, Clark and Knaus(39) showed that infundibulin improves the conduction and regulates contractions in the uterus of the rat.

Hence, pituitrin (posterior pituitary oxytocic factor) -

i. Is a powerful stimulant of unstriped muscle, with specially developed, selective, action on the contraction and retraction of uterine muscle.

ii. Increases conductivity and polarity in the parturient uterus, giving the contractions their rhythmic and expulsive character.

iii. Acts best when the uterus has been sensitized to its influence by oestrin (see before).

iv. Is normally antagonised by progestin in the early months; and later by the gonadotropic hormones (prolan B) and anterior pituitary-like hormones of the placenta in the later months (see before).

The extract of the posterior lobe of the pituitary has been shown to consist of two components viz. (i) oxytocin, which exerts its influence on uterine muscle, and (ii) vasopressin which exerts its influence mainly on the cardiovascular system.

Bourne and Burn[40] found that pitressin (95% vasopressor factor) has no action on the human pregnant uterus; whilst David and Vareed[41] claim that the oxytocic effect of pitocin (95% oxytocin factor) is almost the same as that of pituitrin. Blair Bell and Robson have found that pituitrin has a greater tonic effect than either of its components, but that both pitressin and pitocin can cause contractions of the rabbit's uterus, but not so in the guinea pig.

In 1933 Robson[38] showed that vasopressin produced a uterine response at an earlier stage of pregnancy than oxytocin did. Using strips of muscle excised from uteri at different stages of pregnancy it was found that at two months the muscle either did not respond to stimulation by oxytocin, or only after large doses were injected; whereas vasopressin caused a contraction. With advance in the stage of gestation, smaller and smaller injections of oxytocin caused contractions till at

term the muscle strip reacted with the minimal dose of oxytocin, parturition marking the apex in the reactivity curve to oxytocin stimulation.

The Relationship between the Diencephalon and the Pituitary Gland.

The diencephalon - that part of the base of the mid-brain adjacent to the pituitary gland - and particularly the hypothalamus, has been conclusively shown (Cushing[42]) to be the nervous structure concerned with the emotions. The pituitary gland is anatomically and physiologically related to the hypothalamus, which has been called the head ganglion of the sympathetic nervous system, and is influenced by emotion in its various shades and degrees. The importance of this in relation to uterine muscle action is that emotion and other psychological states have an inhibitory effect on uterine contractions, and emotional states in fact cause an inhibition of the secretion of pituitrin. It is not therefore illogical to postulate that inhibition of uterine contractions due to emotion, etc., may be caused by the action of the sympathetic nervous system on the diencephalon with a subsequent reaction in the adjacent pituitary gland, resulting in an inhibition of pituitary secretion - particularly of pituitrin.

A familiar example of the inhibition of pituitrin secretion through emotional disturbance is provided by observation on cows that refuse to yield their milk, either

because the calf has been taken away, or because they object to a particular dairyman. If the cow hears the bleating of her calf she may yield her milk, but either cause of her refusal can be overcome by an injection of pituitrin. The disturbance of her diencephalon inhibited the chemical or hormonal stimulus to the expulsion of milk but the injection of that stimulant cut under the nervous inhibition, with the resultant secretion of milk.

In the same way t he indirect result of emotional stimuli may cause a diminution in the secretion of pituitrin with as a result a diminution of the oxytocic factor concentration of the blood. The liberation of adrenalin, which inhibits uterine contractions cannot however be excluded (see adrenal glands section).

As already mentioned, the uterus during pregnancy is relatively insensible to pituitrin, being antagonised by progestin and prolan. Near term, however, owing to sensitization of the uterine muscle by oestrin, pituitrin more easily elicits uterine contractions. The frequent failure of pituitrin to induce labour therapeutically unless near term is established; possibly a course of oestrin injections, to simulate as nearly as possible the conditions found near term, may in the future contribute towards a greater percentage of successful medicinal inductions of labour.

The use of pituitrin in labour is a very effective, though dangerous, drug. In view of the very powerful contractions which may result from the injection of pituitrin in the first stage of labour (e.g. in primary inertia), it is a dangerous practice to use this drug in order to expedite the first stage of labour, owing to the risk of tetany of the uterine muscle with asphyxia of the foetus. The sedative form of treatment of primary inertia is always preferable.

During the second stage of labour it is permissible to give the extract of posterior pituitary for simple delay due to weak pains, always provided that (1) there is no disproportion, (2) the head is low down in the pelvic cavity near the outlet and (3) preferably in multiparae, owing to the risk of severe perineal laceration in primiparae.

The chief and, some would hold, the only use for pituitrin is to control haemorrhage after the expulsion of the placenta. Its effect is then most dramatic. In emergency it has been injected directly into the uterine muscle e.g. during Caesarean section. In America it is often given as a routine after the completion of the second stage of labour (De Lee's Midwifery), and it is claimed that there is no increased risk of retained placenta from the contraction ring which is said to result from the exhibition of pituitrin before the end of the third stage.[43]

Thymophysin.

In 1918, Müller demonstrated that a muscle working under

the influence of thymus gland takes longer to become fatigued than a muscle working normally. Under the influence of pituitary (posterior lobe) extract the fatiguing of a muscle takes place in a shorter time than without this influence.

Starting from the observations of Müller, Temesvary studied the influence of various endocrine glands on the guinea pig uterus. He was able to establish that thymus extract produced a rise of tonus and a rhythmization of the uterine contractions; and that it had a fatigue-inhibiting influence (Temesvary). In contradistinction to pituitrin, whose effect is more violent and short-lived, thymophysin induces well controlled rhythmic and prolonged labour pains with no danger of tetany of uterine muscle.

It has further been claimed that thymophysin lessens the painfulness of the first stage of labour, owing to its regulating and tonic effect on uterine contractions. Its chief use is in the stage of dilatation, and in primary and secondary inertia; in the latter condition, however, thymophysin has no effect on the over-fatigued uterus.

In the induction of labour, opinions differ. It is used in induction of labour at Queen Charlotte's Hospital.[44] Ham[45] holds that it is of no value in induction of labour, but that it will differentiate true from false labour pains, by establishing rhythmic contractions in the former, but not in the latter.

In the second stage of labour, the use of forceps has been greatly diminished in certain clinics by as much as 50% (Diasio[46]). As a result the risk of infection in the puerperium is considerably lessened.

To summarise it would appear that thymophysin

(1) Is of doubtful value in the induction of labour, being milder in effect than pituitrin, which itself frequently fails unless at or near term;

(2) Is of value in the treatment of primary inertia;

(3) May be used in secondary inertia, provided the uterus is not over-fatigued before its exhibition. It is safer in inertia conditions than pituitrin;

(4) Its use lessens the incidence of forceps deliveries;

(5) Its use lessens the duration of labour, and is said to render labour less painful.

The Thymus Gland.

The only work done on the relation of the thymus gland to muscle activity has been that of Muller, Del Campo, and Temesvary. Temesvary (1926) described experiments on isolated uterine segments, in which he found that thymus extract alone, in relatively large doses, increased uterine contractions slightly, and that this action was much augmented by addition of posterior pituitary extract. Muller and Del Campo had previously (1917-1918) shown that, when muscular fatigue was induced by electrical stimulation, thymus extract

inhibited fatigue and increased muscular contractions under prolonged stimulation, provided that the muscle was not too greatly fatigued before the thymus extract was used. This observation probably best explains its modifying effect on the action of pituitary extract on uterine muscle. The action of posterior pituitary extract on uterine muscle is a specific one; that of thymus extract a non-specific action, being true of all types of muscle.

Adrenal Glands.

The adrenal glands undergo slight hypertrophy during pregnancy, but little work has been done in special relation to uterine muscular activity. What work has been done tends to be contradictory and confusing. In 1927, Bourne and Burn[47] found that the hypodermic administration of adrenalin inhibited all contractions for 12 minutes, and concluded that adrenalin in moderate doses was inhibitory to uterine contractions. To supplement this, Whitridge Williams in 1930 advises the injection of 5 minims of adrenalin hydrochloride 1/1000 solution to limit the violent contractions of the uterus in labour, or after the maladministration of pituitrin, as in the first stage of labour. Hence, clinically adrenalin may be said to inhibit uterine contractions in moderate dosage.

On the other hand, Blair-Bell[48] found that, in vitro, and in non-pregnant uteri of guinea pigs, addition of

adrenalin hydrochloride 1/1000 solution eventually caused a contraction of the muscle. Here, however, the experiments were performed in vitro, the muscle was from the non-pregnant uterus, and the other endocrine factors (which obviously play an important part normally), were not exerting their influence.

Miller and others[49] conducted experiments on muscle strips removed from human uteri. Using 0.1 c.c. of 1/1000 adrenalin hydrochloride and kymographic recording apparatus, they found that adrenalin contracted 88% (500 strips were tested), had no effect on 2%, and relaxed 10%. The small dose given should be noted. Incidentally, they also found that acetyl choline caused contraction in all cases although the response varied in intensity. Probably the action of adrenalin is to cause contraction in very small minute doses, and inhibition in moderate clinical doses: this follows the old pharmacological principle that small doses often stimulate, larger doses inhibit.

The relation of the sympathetic nervous system to the adrenal glands cannot be ignored. It is well known that the secretion of adrenalin will result from the stimulation of emotional states - fear, anger, hysteria, and so on: and it is also well known that these emotional states - the psychical factor - in labour will inhibit or at least modify uterine contractions. It is therefore logical to conclude that the

inhibition or perversion of uterine contractions which is a feature of these emotional states may be the direct result of outpouring of adrenalin, owing to over-action of the sympathetic nervous system. Moreover the sympathetic is thought to be the supply to the circular (sphincter) muscle of the uterus and cervix, the parasympathetic to the longitudinal (extrusor) muscle: in emotional states the condition of cervical spasm is probably due to over-action of the sympathetic nervous system, with stimulation to contraction of the circular muscle of the uterus and subsequent interference with polarity: dilatation is slow.

More recently the influence of the diencephalon - that part of the base of the mid-brain (hypothalamus) in relation anatomically with the pituitary gland - has been investigated. It is the head centre of the sympathetic nerve paths: certain writers postulate the theory that impulses from the diencephalon affect the adjacent pituitary body with inhibition of pituitrin secretion and cessation of uterine contractions. Here then, are two schools of thought: on the one hand inhibition of pituitrin caused by the influence of the diencephalon on the adjacent pituitary; and on the other hand over-production of adrenalin from the suprarenal medulla. Both appear to be closely connected with the sympathetic nervous system: the result in each case is inhibition of uterine activity.

Adrenalin is sympathomimetic, pituitrin parasympathomimetic, in action: the effect on the uterine muscle is therefore the result of hormonal factors, a direct neutralisation of the one by the other being probable.

The Thyroid and Parathyroid Glands.

There appears to be no relation between the thyroid gland and uterine muscle activity.

The parathyroids do not directly influence uterine muscle activity specially. The effect of the parathyroids on muscle contractility in general is due to its influence on calcium metabolism, a hypercalcaemia not clinically having any influence on muscle contractility, whereas a hypocalcaemia results in the painful, prolonged spasms of tetany. This hypocalcaemia is the result of parathyroid insufficiency, being characterised by an increase in the blood inorganic phosphorus content in addition to the diminution in serum calcium.

It may therefore be stated that a normally functioning parathyroid system is essential for normal calcium metabolism and hence for normal muscle cell metabolism, calcium being a most important inorganic constituent influencing muscle contraction (see Calcium in Physiology of Labour).

The Pancreas.

The internal secretion of the pancreas also influences muscle contraction indirectly through its regulation of carbohydrate metabolism. Glucose is a most essential substance,

being the chief source of muscular energy. By the oxidation of glucose (as glycogen) muscle work is performed, the end products being lactic acid, carbon dioxide and water. A store of glucose (as glycogen) in the body is accordingly of prime importance for the supply of muscular energy and hence for a normal labour. The fact that glycogen is being used in excess during labour is shown clinically by the traces of acetone and diacetic acid which will normally be found in the course of a normal labour: the glycogen has been drawn upon, it may be to the point of depletion, the fats are incompletely combusted in the absence of the necessary glycogen and the bi-products of incomplete fat combustion are found in the varying traces of acetone and diacetic acid in the urine during labour. The longer the labour the more glycogen is combusted, the greater the depletion of the carbohydrate stores, and the greater the need of supplying the parturient mother with a diet rich in glucose, to avoid the development of an acidosis.

Insulin, therefore, in normal amounts is absolutely essential for the supply of glycogen to the uterine, and other, muscle in a form suitable for utilisation. If carbohydrate metabolism is impaired, as in diabetes mellitus, the storage of glycogen during pregnancy is inadequate to meet the demand for glycogen during parturition. A normally functioning muscle cell is hence dependent on a normally functioning pancreas for its supply of energy in the form of glycogen, in

which form glucose is stored under the influence of insulin, and supplied to the muscle cell as required. An inadequate supply of glucose to the uterine muscle will predispose to states of uterine exhaustion.

P A R T III

A Clinical Study of the Uterus in Labour

A CLINICAL STUDY OF THE UTERUS IN LABOUR

1. The Prodromal Stage of Labour.
2. Labour - First Stage.
   - i. Nature of labour contractions.
   - ii. Retraction.
   - iii. Polarity.
   - iv. Differentiation into Upper and Lower Segments.
3. Labour - muscle action in the Second Stage.
4. Labour - muscle action in the Third Stage.

NORMAL UTERINE ACTION IN LABOUR.

Normal labour has been defined as "a labour in which the child presents by the vertex and which terminates without artificial aid, or injury to mother or child."(50) Some authorities attempt to define a normal labour according to its duration and limit a normal labour to 24 hours in duration (Johnson Ch.12, p.116). While this is convenient as an arbitrary division, it is very difficult to define a normal labour according to its duration, for this varies with first and subsequent labours and from individual to individual.

1. THE PRODROMAL STAGE OF LABOUR.

This prodromal stage is a very necessary and essential factor in preparing the uterine muscle for its participation in labour proper. During pregnancy the uterus is passive, but towards the end of pregnancy, notably in the last three to four weeks, signs of awakening activity can be detected. The intermittent uterine contractions of pregnancy become more easily elicited on manual stimulation: the uterine muscle becomes more sensitive to stimulation - mechanical and physio-chemical, probably due to hormonal influences.

In primigravidae, especially, the head sinks lower into the pelvic excavation with subsequent relief of pressure on the upper abdomen and chest - to this phenomenon the term "lightening" is applied. The mother often feels much better generally than at any time during her pregnancy. In addition there is an increased discharge of mucus, signifying awakening activity as well in the mucous glands of the vagina and cervix.

"False pains" may make their appearance, due to the increasing frequency of the rhythmic intermittent pregnancy contractions: the early differentiation into the upper active and the lower passive uterine segments has begun. Increased pressure and stretching of the soft parts leads to backache of varying degree, and some clinicians would have it that severe backache in this prodromal stage is the herald of a pathological labour (Kreis[51]). If examined near term the cervix

often shows some degree of so-called dilatation: in primiparae it will usually admit one finger, in multiparae two fingers: in some cases the canal may be completely obliterated and represented only by the external os, evidence in itself of the activity of the uterine muscle and early differentiation into upper and lower segments in this important prodromal period.

2. LABOUR.

The transition into labour from this prodromal stage is often very gradual and labour is not said to be properly begun until the contractions become sensible to the patient whether as actual pains, or merely as severe backache. Whatever the patient's appreciation of the nature of the pain, labour has not set in until the uterine contractions become effective in "taking up" the cervix.

The action of the uterine muscle varies clinically according to the three stages of labour.

THE FIRST STAGE OF LABOUR:

From the onset of proper labour contractions to full dilatation and effacement of the cervix. Certain features of muscular action come into prominence. These are -

i. The Nature of Labour Contractions.
ii. Muscle Retraction.
iii. Polarity of the Uterine Muscle.
iv. Differentiation into upper and lower uterine segments.

i. The Nature of Labour Contractions.

(a) The contractions are involuntary. The musculature of the uterus has been accredited with having an intrinsic muscular action analagous to that of the heart, i.e. the uterine muscle will contract in vitro after all its nervous connections have been severed. Ganglionic nerve cells have been described in the uterus analagous to the Purkinjé fibres in heart muscle; but unfortunately no nerve connections have been successfully and unequivocally traced to their ganglionic nerve cells. Davis[6] describes ganglion cells in the cervical region which connect with nerve endings from the plexus in the muscularis coat; they are very sparse, being more numerous outside the muscle, beneath the peritoneal coat. "With the exception of the tiny ganglion cells in the probably subepithelial plexus of the cervix ganglion cells do not occur with any degree of constancy within the substance of the uterus."

The uterine contractions are also under the control of the cerebrospinal nervous system. The effect of fear, emotion, and mental trauma on the nature of the uterine contractions is evidenced by the inhibition or stimulation of contractions which may result from these stimuli.

That the contractions are involuntary is amply demonstrated by the fact that labour may occur and be brought to a natural successful end in case of paraplegia due to myelitis of the cord etc., while uterine contractions will continue, luckily,

under the deepest of anaesthesia.

(b) The action of the uterus is not peristaltic to the degree that is found in the lower animals. In lower animals with tubal uteri the labour contractions are more peristaltic in nature, and it is probable that polarity of uterine muscle has arisen and developed from peristalsis, both peristalsis and polarity being in essence the same, i.e. contraction of one segment with relaxation of the muscle of the neighbouring segment.

Chassar Moir[52] has adapted a kymographic recording apparatus to record the contractions of the fundal and cervical part of the uterus at the same time. He found that in some, not all, of the contractions, the fundal and cervical contractions were coordinated, the cervix starting to contract just after the fundal contraction had passed its peak: in point of time, the cervix lagged 17 seconds behind the fundus in contracting. From this he infers that a wave of contraction passes over the uterus from fundus to cervix. Ivy, Hartman, and Koff[53] found similar results on the mode of action of the monkey's uterus during labour.

If one studies the morphology of the uterus it is seen that the human uterus expels its contents in a manner comparable with that seen in the lower mammals, in whom typical peristalsis of the uterine horns takes place. In the human, however, the purely peristaltic nature of the contractions has become more

highly evolved, with the addition of retraction to control the progressive nature of the expulsion, which is necessarily slower in the human.

(c) The Uterine Contractions are Intermittent and Rhythmic.

The "pains" recur at regular intervals. In the first stage of labour they will recur at intervals of perhaps 30 minutes: as dilatation proceeds the interval becomes shorter until near full dilatation of the cervix they are separated by only a few minutes. During the second stage of labour, when the contractions become expulsive in character they recur in favourable cases every 2-3 minutes, until, when the head is about to be born one strong contraction succeeds another. With the delivery of the child the contractions, though more infrequent, are nevertheless intermittent although now the interval is from 4 to 5 minutes.

This intermittency and rhythmicity of the contractions is necessary, since

(1) It aids the circulation in the uterus. The blood in the large maternal sinuses of the uterus would be stagnant were it not for the uterine contractions, which may again be likened to the systole of the heart muscle.

(2) It prevents foetal injury from pressure. Prolonged contraction would be fatal to the foetus, e.g. tetanic contraction from any cause - dystocia, or mal-administration of an oxytocic drug - will cause foetal death.

(3) It prevents foetal asphyxia from strangulation of the circulation in the placenta.

(4) It provides necessary rest for the muscle and the removal of waste.

The uterine action is a good example of applied power: the intermittency and rhythmicity is beautifully controlled and regulated.

(d) The Labour Contractions are accompanied by "Pains".

The contractions of the parturient uterus are manifest clinically as "pains". The amount of pain felt varies with the psychological factor, and is therefore most acute in highly strung, nervous, patients who call out for an anaesthetic at the slightest pretext, and generally do their best to render their labour pathological. Some labours have been described as being truly painless; the influence of diet on the character of the pains is probably very important, a salt-free, protein-free diet being accredited with rendering a labour comparatively painless.

Social factors are important in the registering of pain, the poor class being supposed to have less painful labours (clinically, not necessarily subjectively). Racial factors are also reputed to influence the severity of the pains, but accounts of labours in uncivilised races tend to disprove the claim that their labour is painless e.g. in Engelman's "Labour Among Primitive Peoples". Probably their apparently easy

painless labours are due in no small part to a combination of the psychological and the physical factors, being mentally enured to suffering and physically very fit to undergo the ordeal of labour.

The Causation of Labour "Pains".

It has to be remembered that labour "pains" are referred in character. MacKenzie developed the conception of referred pain, and postulated a viscero-sensory and viscero-motor reflex. (J. MacKenzie, Symptoms and their Interpretation, 4th Edition, 1920.) More recently, Morley (Abdominal pain, 1931) has challenged MacKenzie's theory; in his (Morley's) opinion "referred pain only arises from irritation of nerves which are sensitive to those stimuli that produce pain when applied to the surface of the body." Morley therefore introduced the terms "peritoneo-cutaneous radiation" and "peritoneo-muscular rigidity". Modern physiological opinion rejects the possibility of accurately localized abdominal pain being caused by irritation of autonomic nerves.

Developing MacKenzie's theory of viscero-sensory reflex, which has held the support of physiologists, it has been suggested by Theobald[54] that "the pains of labour, with the exception of the stretching of the perinaeum during delivery, are largely, if not entirely referred along cutaneous nerves." The abdominal pain may be completely abolished by infiltrating the ilio-hypogastric and ilio-inguinal nerves, and the sacral

pain can be markedly diminished, if not completely relieved, by infiltrating the pudendal nerves. From these observations, if substantiated, it is probable that labour pains are referred in character, being really associated somatic pains felt in the body wall supplied by the cerebrospinal nerves of the same segment of the cord. Labour "pain" is therefore an example of a viscero-sensory reflex, the sensation arising in the stimulation of sensory nerve twigs in the pelvic viscera and connective tissue and muscular planes by the descent of the foetus following uterine contraction. The painlessness or otherwise of labour would therefore appear in part to depend upon variations in those sensory stimuli; the degree of resistance of the pelvic floor and pelvic soft tissues in general will directly influence the magnitude of the sensory impressions, or in other words, the amount of referred pain felt by the patient will vary directly with the amount of resistance offered by the pelvic structures to the descent of the foetus. Similarly, in regard to uterine contractions, it may be that the degree of tension developed in the contracting muscle cell sets up a corresponding viscero sensory-reflex which is felt subjectively as pain. The spinal segment involved is the first lumbar segment, which explains why low spinal anaesthesia does not abolish completely the abdominal pains of labour.

Labour pains are therefore due to referred pain, which

arises as a result of -

(1) Contractions of uterine muscle.
(2) Stretching of the soft tissues of the pelvis,
or (3) To a combination of both (1) and (2).

Uterine muscle is unstriped or involuntary. As such it is not unreasonable to assume that its contractions must be subject to the same laws that govern smooth muscle action elsewhere, e.g. in the alimentary tract, the bladder, and the heart; although the latter differs from other plain muscles, in that it has an intrinsic, inherent, mechanism of contraction, which has not yet been proved to exist in the case of the uterus, although it is a possibility which cannot be excluded.

The normal physiological contractions of unstriped muscle are painless: the intermittent contractions of the pregnant, not the parturient, uterus are painless - these contractions are physiological. In labour the contractions vary in intensity - they become progressively stronger; but the clinician will note often that the labour contractions in the first stage of labour are not necessarily any stronger, nor last any longer, than the contractions of pregnancy. During antenatal examinations one has often to wait for from 45-60 seconds or more for a contraction to subside before continuing with the abdominal examination: clinically this contraction may be as strong as that occurring in the first stage of labour: and yet the former is painless.

The explanation may lie partly in the degree of tension developed in the contracting muscle cell. (This point is discussed in detail in the section on the Physiology of muscle contraction.) The greater the tension developed in contracting smooth muscle, the greater the pain experienced: under the powerful stimuli of labour the tension will become progressively greater. On the other hand many women go through labour without appearing to suffer any great degree of pain: other factors are therefore in action.

Turning to the second point, i.e. stretching of the soft tissues by the descent of the foetus through the birth canal, further light is thrown on the point at issue, viz. the cause of the "pains".

By the soft tissues is meant all the structures of the pelvic floor and the birth canal - from the pelvic peritoneum to the skin overlying the perinaeal body: cellular tissues, muscles, blood vessels, nerves, vagina, and so on.

In labour, certain new features make their appearance in the mechanism of the uterine muscular action, viz. retraction and polarity. As a result of a combination of these the foetus may be likened to a foreign body which is being expelled from a viscus: the mechanism is merely modified so that the foreign body i.e. the foetus, may escape injury during its expulsion. The foetus is hence driven strongly downwards, pressing out and stretching the soft tissues of the pelvis in

its progress. This bursting, stretching, and tearing inevitably give rise to severe pain. The pain will vary with the intensity of the contraction. It will also vary with the degree of resistance of the tissues. It will synchronize with the acme of the contraction, for only during a contraction will further pressure be exerted on, especially, the nerve filaments found in the pelvic and other soft tissues. Between the contractions the pressure will be, to a great extent, relieved, although a certain amount of pressure will be kept up by uterine retraction. During the muscular contraction - and only those muscular contractions which are strong enough to open up the soft tissues - the stretching and possibly tearing of the soft structures gives rise to acute pain; between the contractions, retraction maintains the progress gained by the preceding contraction, the pressure is relieved somewhat, and dull aching, especially backache, succeeds the acute pain.

To advance certain points in favour of this causation of the "pains":

(1) The rhythmic pregnancy contractions are not felt as pains, because there is no polarity, no retraction, no progress downwards and hence no stretching of the soft tissues. The contraction achieves no alteration in the position of the foetus: it is exerted equally in all directions on the body of the foetus: no differentiation into upper or lower segment

action has begun.

(2) In a varying percentage of cases in their first stage of labour, severe backache may be the only complaint, although the uterus is contracting normally and strongly. The patient may not complain of any "pains" till near the end of the first stage when the pains become expulsive in character. The new factors superadded then are (i) increase in the intensity, frequency and duration of the contractions, supplemented by the auxiliary forces of the mother, and hence (ii) greatly increased stretching of the soft tissues. The intensity of the second stage pains is not sufficiently increased to account for the gradual transition from a "painless" (if backache is excluded) to a "painful" labour: to a certain extent tension in the muscle cell is increased, with a corresponding appearance of "pain", but this cannot account for all the pain felt. The bearing down forces of the mother are now in evidence and these obviously supplement the amount of pressure being exerted on the pelvic and perinaeal soft tissues, including the sensory nerves and ganglia..

Moreover, the parturient mother will describe the agonizing pains just as the head is being crowned, or passing over the perinaeum, as "bursting", "tearing", or "rending", in character. Here the pains are most intense, and here also the stretching of the soft tissues - in this case the fibro-elastic perinaeum - is greatest.

(3) After the end of the third stage of labour, the uterus is fully retracted: during a contraction it becomes as hard as a cricket ball: and no pain is felt. There is a strong contraction, with full retraction, of uterine muscle, yet the act is painless, due to the fact that there is now no pressure being exerted on the soft tissues of the pelvic floor. It has been stated that uterine pains are due to compression of the terminal nerve twigs lying between the muscle lamellae: but the compression of these nerve twigs after the third stage of labour must be very powerful indeed during a contraction: this explanation is hence inadequate. The contractions of the uterus have been recorded experimentally as measured by tracings of uterine contractions, using a pressure bag connected up to a tampon and recording lever etc. It has been found that a third stage contraction can be as strong as a second stage contraction: yet no pain is felt in the latter. It is obvious that uterine contractions per se do not cause "pains".(55)

(4) In "precipitate labour" the uterus is acting strongly and efficiently, yet the mother may be caught unawares, and may merely feel the urge to go to stool. The great majority of these cases occur in multiparae with already stretched and relaxed muscles and soft tissues. The uterine muscle is acting normally here, the actual muscle contractions are painless, the soft structures are not being severely stretched

being already lax and atonic, and labour proceeds painlessly. Very rarely painless, and hence often an apparently precipitate, labour may occur in a primipara.(56)

(5) In early rupture of the membranes with, as a result, more direct pressure of the hard foetal head on the pelvic tissues labour is said to be more painful - the pains are said to be more severe and stronger in intensity, with a subsequent diminution in the duration of the first stage of labour. No doubt the reflex stimulation from the head pressing on the cervical nerve ganglion is responsible for the increased frequency and intensity of the contractions often found in these cases.

(6) The frequent exhibition of warm hip baths at or near term is accredited with having a relaxing effect on the soft tissues of the pelvis, by rendering them more hyperaemic and causing a greater degree of softening of the soft tissues.

(7) In albuminurias of pregnancy there is a slight percentage of painless or comparatively painless, labours. The outstanding feature of most of these cases is severe backache, although clinically they are having very good, strong, regular, labour contractions. Multiparae and primiparae are both affected thus. Albuminurias have a varying amount of oedema of all the tissues: the pelvic soft tissues, and possibly the interstitial tissue of the myometrium,

probably participate in this oedematous infiltration, and they are therefore more soft and less resistant: as a result they are more easily, and hence more painlessly, stretched. This theory is advanced in an attempt to explain the comparatively painless, speedy, labours found to occur in albuminurias in this investigation.

In recent years Sir Thomas Lewis has shown that the pain found in angina pectoris and intermittent claudication has its origin in an ischaemia of the musculature. The blood supply is temporarily interrupted, the muscle striped and unstriped, is deprived of oxygen, waste products accumulate and the pain of angina or intermittent claudication results. It is interesting to apply this ischaemic theory to uterine muscle, for the powerful contractions of the uterus can, and must, produce a temporary ischaemia of the musculature: however, the blood supply of the uterus is so rich that it is difficult to attempt to attribute "pains" to an ischaemia of the muscularis.

In concealed accidental haemorrhage, the pain is continuous, of much greater severity, and accompanied by shock. The cause of the pain is still controversial. Some would have it due to the haemorrhages into and between the muscle lamellae, together with the ballooning and over-distension of the uterine walls: here the pain is due to over-stretching of the nerve filaments in the uterine wall, together with the varying degree of ploughing up of the uterine muscle and nerves by the

interfascicular haemorrhages. Others would have it that the intense pain is really due to a tetanic state of the uterine muscle. This type of pain is pathological, however, not physiological, whatever its nature and causation.

To summarize therefore labour pains are due to sensory impressions arising in

(1) The contracting uterine muscle;
(2) Trauma due to stretching and dilating of the soft structures of the pelvic floor.

In the contracting uterus, several components are found each probably contributing in varying degree to the degree of pain experienced.

a) The tension within the muscle cell increases with the intensity of the contraction. Increase in tension leads to the production of painful impressions.

b) The terminal nerve twigs lying between the muscle lamellae are compressed. This is probably of minor importance in the causation of pain.

c) Ischaemia of muscle during contraction may, theoretically, give rise to pain. It is interesting to note that the greater the tension and the slower the contraction, the greater will be the degree of ischaemia developed.

These visceral impressions become referred along certain cutaneous nerves. Alteration in the magnitude of the contraction of the muscle cell will cause an alteration in the strength of the afferent stimuli; and, similarly,

alteration in the resistance of the pelvic and perinaeal structures e.g. through multiparity, oedema, heat, and degree of development of the pelvic floor etc., will cause a proportionate alteration in the nature of the afferent stimuli. The weaker the afferent stimuli the less painful the labour.

ii. Muscle Retraction.

Retraction is the name applied to that progressive and permanent diminution in length of a muscle which accompanies and succeeds contraction of that muscle. It may, however, occur independently of a contraction, i.e. it may occur in the absence of a contraction. It is possible for retraction to disappear, and hence it is not, strictly speaking, permanent - e.g. in atonic post-partum haemorrhage the uterus may be completely relaxed. The function of retraction is to maintain the advantage gained by the preceding contraction.

Retraction is not a feature reserved for application to uterine muscle action: it is found also in the mechanism of bladder and bowel action. Clinically it is observed by the hand on the abdomen noticing how the level of the fundus descends as the head descends lower in the pelvis. Retraction is merely specially developed in the case of the uterine muscle.

In the bladder retraction occurs, but it is possible to again re-distend a retracted and empty bladder. Not so in

the uterus - it is permanent, and only disappears when muscular tone is abolished. The degree of retraction present is therefore a good clinical index of the tone of the uterine muscle; where tone is normal, retraction is normal: when tone is altered, as in general or local tonic contraction seen in some cases of dystocia, retraction is also influenced, and the result is injurious both to mother and foetus.

In the first stage of labour, retraction maintains whatever progress has been gained by the preceding contractions, by preventing the foetus from recoiling, as it were, at the end of a contraction, it helps to dilate and stretch the soft parts, to open up the birth canal and to aid in the descent of the foetus. It gives to labour its progressive character. At the same time this retraction is non-injurious to the foetus, exerting only steady pressure on the upper pole of the uterus, not compressing the uterine contents as in a contraction: there is therefore no interference with the placental and uterine circulation during retraction, in the first stage at least. In the third stage of labour, retraction becomes complete and it is definitely the best haemostatic devisable.

Certain authors would have it (Fairbairn[57]) that the cause of uterine pains is to be found in retraction. The pregnancy contractions are not felt because there is no retraction present: in labour, retraction appears as a new feature of the uterine mechanism and the "pains" follow:

moreover, the "pains" increase in severity as retraction progresses and the head descends through the birth canal. Unfortunately, this suggested cause of "pains" does not account for the absence of pain when the uterus is fully retracted, as after the third stage of labour; nor those cases which have good progress, and retraction, in the absence of "pains". The absence of "pains" after the third stage of labour has been ascribed to the fact that retraction is then complete: no further retraction is possible, and hence there are no pains.

iii. Polarity.

Polarity is not a new feature which develops during labour; it is found in the uterus during pregnancy. Nor is polarity of muscular action confined to uterine muscular action - it is found also in the contracting bladder and rectum. By the term polarity, in relation to the uterus, is meant contraction of the upper active segment of the uterus, with co-ordinated relaxation of the lower passive segment of the uterus, or vice versa. During pregnancy the reverse holds good, the upper segment (body) of the uterus being in a state of relaxation, the lower segment (isthmus and cervix) in a state of contraction. This is obviously essential to the continuance of gestation.

Polarity must therefore be co-ordinated, co-ordination being dependent on the integrity of the nervous connections.

Where this nervous co-ordination is interfered with, or perverted, labour comes to an impasse and no progress is possible: such a condition is found in spasm of the cervix, where the muscle in relation to the cervix and lower uterine segment contracts instead of relaxing. Polarity is analogous to the physiology of other muscular organs in which contractions of the muscles of the body of a viscus is attended by relaxation of the muscles guarding its exit. The reverse also holds good e.g. stretching the anus of an infant with the little finger stimulates evacuation of the bowel.

The analogy with peristalsis is even greater. The analogy is so close - contraction of one segment with associated relaxation of the segment above or below it - that uterine polarity may have originated in, and developed from, peristalsis. In the tubal uteri of the lower mammals the contractions are peristaltic in character, but this peristaltic action is lost as the tubal form of uterus develops into that found in the higher mammals.

iv. Formation of the Upper and Lower Segments of Uterus.

As a result of these phenomena of polarity, contraction and retraction, the uterine muscle becomes differentiated into two segments with completely different physiological actions. The muscle fibres of the upper pole of the uterus - in what corresponds to the fundus and body of the uterus (excluding

the isthmus) - are in this case the upper, active, contractile part of the uterus; contraction with its accompanying retraction results in the muscle fibres of the upper segment being progressively and permanently shortened and thickened. The muscle of the lower pole of the uterus, and especially that part of the uterus which originates from the growth and development of the isthmus, is relaxed. The result is that this lower uterine relaxed segment of muscle becomes progressively more thinned and stretched by the active contraction and retraction of the upper segment. The action of the longitudinal and, to a lesser extent, the oblique muscle fibres is to progressively pull up the longitudinal muscle fibres of the lower segment, which offer no resistance to this upward pull. Hence the cervix becomes first effaced; the cervical canal as such disappears, the internal os is obliterated. The cervical canal becomes first cone-shaped with the base uppermost at the internal os: the canal becomes progressively shorter till only the external os is left to represent the cervix. This in turn becomes greatly thinned out, forming, as it were, a diaphragm stretching across the vault of the vagina, with the external os as an aperture in this diaphragm. As the pulling out-and-up process proceeds this aperture becomes gradually larger until the cervix ultimately disappears. The vagina and the cavity of the uterus are then continuous, and the birth passage has been canalized.

In the later months of pregnancy, the lower uterine segment is being gradually prepared for its passive part in labour, but it does not become fully developed till labour has set in. In examining multiparae and primiparae near term it will often be found that the cervical canal has been effaced before labour, the cervix being represented merely by the external os. The less the cervical canal is obliterated at the beginning of labour the greater is the resistance during the contractions of labour. Labour cannot be judged according to the amplitude of the contractions alone, without at the same time finding what relation that bears to the general condition of tension and the resistance of the cervix in each case. Kreis(51) maintains that severe backache near term is a pathological prelude to labour and indicates a considerable resistance of the cervix, which he attributes to spasm of the cervical muscle, i.e. to perverted polarity, resulting from interference with the normal reflex nervous mechanism.

3. THE SECOND STAGE OF LABOUR.

The action of the uterus in the second stage of labour differs mainly in degree. No new features are found; the contractions increase slightly in intensity, in frequency, and in duration, but otherwise the nature of the contractions is unaltered. The "pains" increase in severity in the great majority of normal cases, probably due to the increase in the

tension developed in the muscle cell with the increase in strength of the contraction, and to the greater pressure now being exerted on the soft parts as the presenting part descends. According as the uterus expels the foetus and the uterine cavity is thereby diminished in capacity, so does retraction increase and the fundus follow down the upper pole of the descending foetus. The differentiation into upper and lower segments is now greatly in evidence, the line of demarcation between the two segments being now manifest clinically as a transverse ridge normally situated about 2 inches above the symphysis pubis. It is known as a "retraction ring", and the variation in the level of the retraction ring is a valuable clinical index of the state of the uterine musculature in cases of obstructed labour, where it gradually ascends in the abdomen till it may be found as high as the umbilicus in advanced dystocia. To this ring, in pathological labours, the name "Bandl's ring" has been given, although it was first described by Braune.

As the second stage progresses, the upper segment becomes gradually thicker, therefore, and the lower segment thinner and more distended.

A new feature is found in the second stage of labour, but it is really an extrinsic factor and has nothing to do with the intrinsic action of the uterus, viz. the appearance

of a bearing down action on the part of the mother. It is in great measure due to this bearing down action by the "auxiliary forces" - the abdominal muscles - that the contractions become expulsive in character. The mother involuntarily begins to expel the foetus along the birth canal. The importance of the bearing down efforts of the mother cannot be exaggerated: without them, labour may be terminated normally, but the process is greatly slowed up and the life of the child thereby jeopardised.

The mother's prime object now is to expel the foetus, and near the end of the second stage she becomes apparently powerless to control the uterine contractions, although by making a strong mental effort she should be able to curb her bearing down action. The contractions now succeed one another in rapid succession until when the head is passing over the perinaeum she feels as if she is being torn asunder. The pain at this stage may be so severe that she may faint, and has even been known to go temporarily insane.

4. THE THIRD STAGE OF LABOUR.

With the expulsion of the foetus and the end of the second stage, the uterus enters on a stage of complete physiological rest. Retraction is now complete and maintains haemostasis. After from five to ten minutes rest, the uterus begins to contract again but the contractions are now painless

in character, and the mother may merely be aware of a slight 'tightening' feeling in the lower abdomen. The effect of full retraction aided by the contractions, which recur about every four minutes, is to slide the placenta off the uterine wall into the lower uterine segment. The fully retracted and contracted uterus is felt to ride on top of the placenta in the lower uterine segment, until finally parturition is completed by the placenta being expelled either by a further bearing down on the part of the mother, or more often, by being expressed by the accoucheur.

Scientifically controlled experiments[55] have shown that the contractions of the uterus after the third stage may be as powerful dynamically as second stage contractions: it is to a great extent the aid given by the auxiliary forces which tend to give the impression that the second stage contractions of the uterus are much more powerful than any other.

The importance of the phenomenon of retraction during the three stages of labour cannot be exaggerated. In the first stage, retraction is not greatly in evidence until near full dilatation: but nevertheless it is important in maintaining what progress has been made by the preceding contraction, and but for the shortening in length which occurs during retraction, dilatation of the cervix would be impossible, and likewise the differentiation of the uterine muscle into upper and lower segments. In addition, the steady firm pressure

which is exerted on the foetus aids moulding of the head to occur: but for the presence of retraction the head might tend to ascend again, with the elastic recoil of the pelvic floor, between the contractions. Following rupture of the membranes, with the escape of the liquor and the consequent diminution in the capacity of the uterus, retraction becomes more marked and the uterus now embraces the foetus more firmly and hence the uterine contractions exert their applied power more directly, and hence more forcefully.

During the second stage this action is continued and the uterus follows down the foetus, always being in close contact with the descending upper pole: retraction gives to labour its progressive character, and without it, labour would be a more prolonged and spasmodic process - in fact, spontaneous delivery would be impossible.

With the expulsion of the foetus retraction is at its acme; it aids in separating the placenta from the uterine wall; and, most important, it acts as nature's method of producing efficient haemostasis, by compressing the large uterine sinuses between living ligatures of uterine muscle, the "figure-of-eight" arrangement of the muscle being a very important factor in contributing to this perfect haemostasis.

P A R T IV

Case Records

CASE RECORDS

1. Introduction to Case Records.
2. Results of Investigation into Uterine Action in a series of 752 Consecutive Cases of Labour.
3. Discussion of Results.

1. INTRODUCTION TO CASE RECORDS.

The case records have been briefly summarized in the forms of investigation shown at the end of this section. The forms are self-explanatory. As far as possible the results have been tabulated: in some instances graphs have been drawn.

A certain amount of the details of the investigation were obtained by the sisters-in-charge of the Obstetrical wards in the course of their routine enquiries when a woman was admitted to the ward. The record of progress of the cases in labour, too, has largely been made from the official hospital case reports, many of the details of which are entered by the nursing staff. It has happened also that a normal case may

have been admitted in labour and delivered soon after admission without having been seen by the medical supervisor, e.g. in cases admitted during the night. Such cases have been assessed and classified as faithfully as possible: a great deal of reliance has hence been placed on the co-operation and support given by the nursing staff in general.

Those cases which were abnormal in any degree were all personally supervised and recorded. In certain cases, where possible, cases are cited in more detail to illustrate some particular point; otherwise the results have been collected together and tabulated according to the subject under consideration, individual recording of 752 cases within the thesis being impracticable as well as undesirable.

The results are arranged in sections under individual headings according to the factor under investigation e.g. "Age", "Parity", "Nutrition" and so on. It has been found to be less confusing to discuss the results in the same section as the results are tabulated or recorded, rather than to give a bare statement of results and defer the discussion to a subsequent section.

The sections in which the results are arranged and discussed are headed as follows:

a) Age.
b) Parity.

c) Co-existing Maternal Disease -

i. Not due to pregnancy

ii. Due to pregnancy.

d) Social and Economic Factor.

e) Nutrition.

f) Physique.

g) Psychological Factor.

h) Relation of Rupture of the Membranes to Uterine Action.

i) Relation of Induction of Labour to Uterine Action.

j) Uterine Inertia.

SPECIMEN OF FORM OF INVESTIGATION

USED TO COLLECT DATA FOR RESULTS

Name of Patient	511 Jane Shields	512 Nellie Malone	513 + Ada Duff	514 Mary Gibson	515 Morfydd Roberts	516 + Bertha Taylor	517 Dora Hall	518 Lily Dickens
Age	31	24	22	21	32	29	27	25
Parity	I	I	I	I	I	I	I	I
Co-existing Disease:-								
a) Not due to Pregnancy	-	-	-	-	-	-	-	-
b) Due to Pregnancy	-	-	-	-	Mod.toxaemia	-	Mod.toxaemia	-
Economic State	P.P.	P.P.	P.P.	P.P.	P.P.	P.P.	P.P.	P.P.
Nutritional State	Good	Good	Good	V.Good	Good but poor tone	Good	Good	Good
Physique (Abdominal Wall, etc.)	Good	Good	Small,slim	Plump, good	Trifle flabby	Good	V.Good	Good
Psychical Factor	Good	Good	V.Good	Good	Poorish	Poor	V.Good	V.Good
Notes:-			Anencephalic with hydramnios		Surg.Induction 1 week's A.N. treatment. High B.P. No alb.	Vying with each other. Making noise with pains. Slow progress.	Vying with each other. Both in labour & ward simul. Bad effect on each other.	Forceps: delay at outlet: poor bearing down. No rigidity.
Prem. Rupture, cause	-	-	-	-	Surg.Induction	-	Drug induction	-
Faults of Passages:-								
a) Bony Parts - Contraction	-	-	-	-	(Died at 3 months *from toxaemia* with breast abscess)	Primary inertia	-	-
b) Soft Parts	-	-	-	-		-	-	-
Faults of Passenger:-								
a) Presentation	R.O.A.	L.O.A.	L.O.P.	L.O.A.	R.O.A.	L.O.A.	-	R.O.A.
b) Plurality							b) i R.O.P.6.5oz. ii L.O.A.5.10 oz.	
c) Disproportion i.e. size, etc.	6.8 ozs	7.5ozs	2.4 ozs	8.6 ozs	8.8 ozs	7.6 ozs		7 lbs
d) Abnormality								
Forces:-								
a) Sedatives Drugs					Thymophysin 6 units (3x2)		Thymophysin 0.3c.c½-hrly 6x (1.8 c.c.s)	
b) Over Distension			Hydramnios		b) Liquor ++ Large foetus		b) Plural preg.	
c) Psychical Inhibition					c) Present. Calcium lactate	c) Present inertia.		Secondary inertia.
d) Aliter								

Name of Patient	Jane Shields	Nellie Malone	+ Ada Duff	Mary Gibson	+ Morfydd Roberts	+ Bertha Taylor	+ Dora Hall	+ Lily Dickens
Pains Commenced	7.3.37 6 p.m.	9.3.37 8 a.m.	10.3.37 9 a.m.	11.3.37 10 a.m.	12.3.37 2 p.m.	12.3.37 4 a.m.	12.3.37 6 p.m.	15.3.37 3 a.m.
Pains, Character i Weak ii Moderate iii Strong	Mod.	Mod.	Mod.	Mod.	Mod.	Weak and niggling. Stronger after p.v. examination.	Feeble, niggling inertia type of pains.	Mod.
Pains, Progress i Slow ii Moderate iii Quick	Slowish	Mod.quick	Mod.	Mod.	Mod.	and stretching of cervix 5/-	V. Slow	Mod.
Duration of First Stage	29.50 mins	10 hrs 30 mins	7 hrs	13 hrs	9 hrs 30 mins +	62 hours	45 hours	16 hrs 15 mins
Membranes, Ruptured	8/3/37 11.50 p.m.	6.55 p.m.	4 p.m.	10.20 p.m.	11.3.37 6 p.m. L.P. of 20 hrs Surg.induction	14.3.37 2.20 p.m. (5/- d) during p.v.	12.3.37 2.10 p.m. Prem.rupt.(drugs)	8 p.m.
Pains in Second Stage — Character i Weak ii Moderate iii Strong	Mod.	Mod.	Mod.	Mod.	Mod.	Mod.	Fair becoming exhausted	Mod.
Pains in Second Stage — Progress i Slow ii Moderate iii Quick	Mod.slow	Mod.	Mod.	Mod.	Mod.	Mod.	Slow: no progress (posterior)	Slow. Delay on perinaeum; no progress for 1 hr.
(Breech) Head Showing							Head in cavity. Manual rotation and (1) Forceps: (2) Normal	Forceps
Termination	Normal	Normal	Normal	Normal	Normal	Normal	(1) 2 hrs 5 mins (2) 2 hrs 55 mins	3 hrs
Duration of Second Stage	2 hrs 55 mins	2 hrs	2 hrs	2 hrs 15 mins	1 hr 55 mins	2 hrs 30 mins	15 mins	15 mins
Duration of Third Stage	20 mins	20 mins	20 mins	10 mins	20 mins	20 mins	Normal	Normal
Termination: Condition of Uterus: Loss	Normal	Normal	Normal	Normal	Normal	Normal	48 hrs 10 mins Mild inertia Prem.Rupt.	19 hrs 30 mins Forceps
Total Duration of Labour	33 hrs 5 mins Slowish	12 hrs 50 mins	9 hrs 20 mins Hydramnios	15 hrs 25 mins	11 hrs 45 mins Toxaemia Poor gen.condn. Died 3 months breast infection	64 hrs 50 mins Inertia		

RELATION OF AGE TO UTERINE ACTION IN LABOUR

Five age groups have been made. These are:

(1) Under 20 years of age;
(2) From 21 to 25 years of age, inclusive;
(3) From 26 to 30 years of age, inclusive;
(4) From 31 to 35 years of age, inclusive;
(5) Above 35 years of age.

The total number of cases in each age group is shown in a separate column, irrespective of parity. It is impossible, however, to consider Age per se in regard to uterine action, since the Parity may be of the greatest importance in assessing a case, e.g. in the "above 35 years" group it is of infinite value to know whether one is dealing with a primigravida or a thirteenth para - the whole assessment is altered according to the Parity. Hence Tables II and III show the primigravidae and the multiparae separately.

"Inertia" All degrees of inertia are included in Table I. A rough classification of inertia into mild (primary or secondary) and genuine (primary and secondary) has been made in Tables II and III. A more detailed classification and tabulation of Inertia will be found in the "Inertia" Section (Section J).

"Good Uterine Action" The main factor in determining this factor is the duration of the labour and its character, e.g. if comparatively painless. In primigravidae a labour is "good" if its duration is less than nine hours. In multiparae, if its duration is less than three and a half hours (see Discussion). The average duration of labour in a primigravida has been taken as 12 to 18 hours; in a multipara 6 to 8 hours.

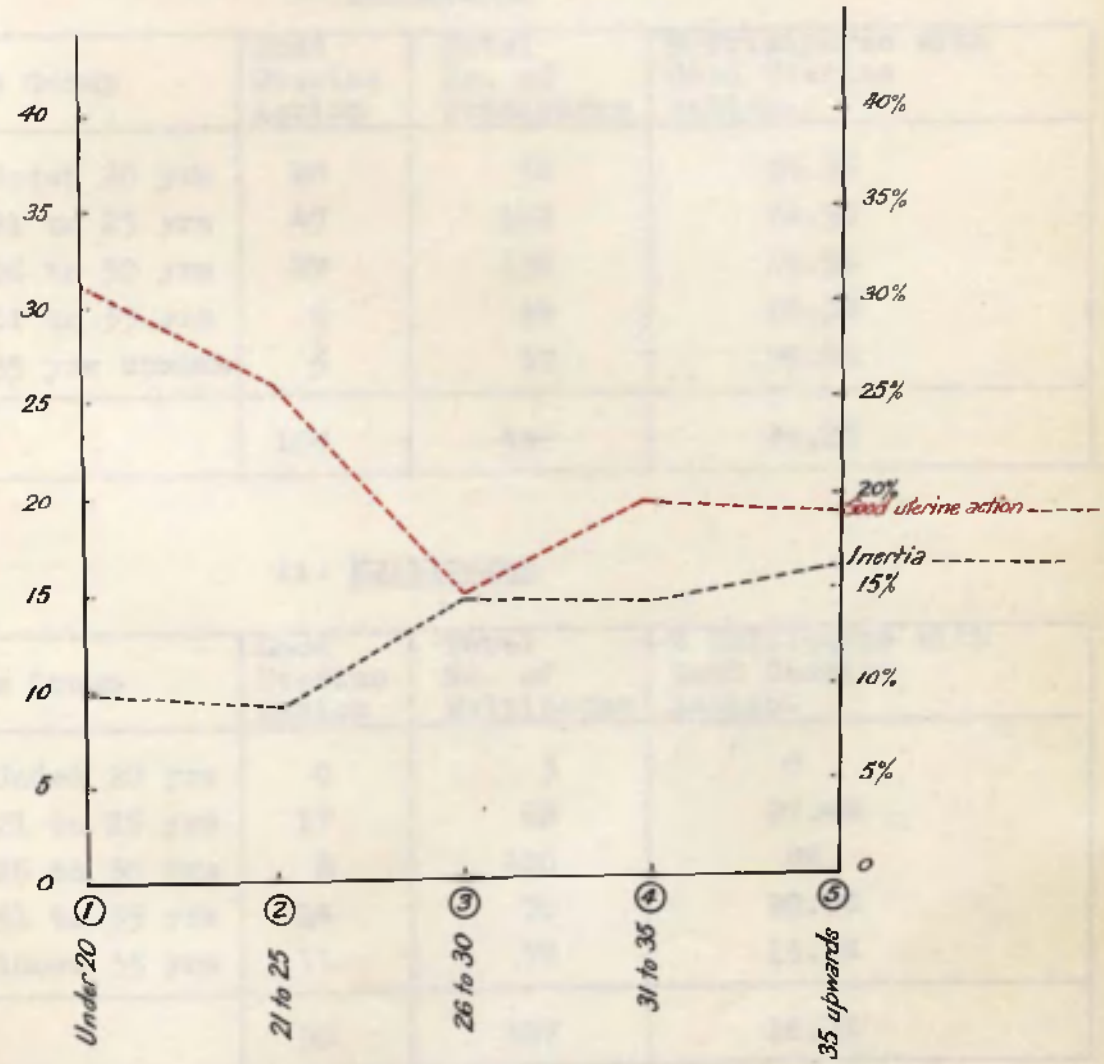
TABLE I

AGE (Primigravidae and Multiparae together)

Age Group	Inertia (all degrees)	Good Uterine Action	Total No. in Age Group	Percentages	
				Inertia	Good Uterine Action
(1) Under 20 yrs	6	20	61	9.8%	32.8%
(2) 21 to 25 yrs	21	64	243	8.9%	26.3%
(3) 26 to 30 yrs	35	35	238	14.9%	14.9%
(4) 31 to 35 yrs	17	23	119	14.2%	19.3%
(5) Above 35 yrs	15	16	91	16.4%	17.5%
	94	158	752	12.5%	21%

GRAPH I

Age (Irrespective of Parity)



Age Groups

TABLE II

AGE AND PARITY IN RELATION TO UTERINE ACTION

Good Uterine Action

i. Primiparae

Age Group	Good Uterine Action	Total No. of Primiparae	% Primiparae with Good Uterine Action.
(1) Under 20 yrs	20	58	34.5%
(2) 21 to 25 yrs	47	181	24.5%
(3) 26 to 30 yrs	27	138	19.5%
(4) 31 to 35 yrs	9	49	18.3%
(5) 35 yrs upwards	5	19	26.3%
	108	445	24.2%

ii. Multiparae

Age Group	Good Uterine Action	Total No. of Multiparae	% Multiparae with Good Uterine Action.
(1) Under 20 yrs	0	3	0
(2) 21 to 25 yrs	17	62	27.4%
(3) 26 to 30 yrs	8	100	8%
(4) 31 to 35 yrs	14	70	20.0%
(5) Above 35 yrs	11	72	15.2%
	50	307	16.2%

GRAPH II

Age and Parity in Relation to Uterine Action

Good Uterine Action

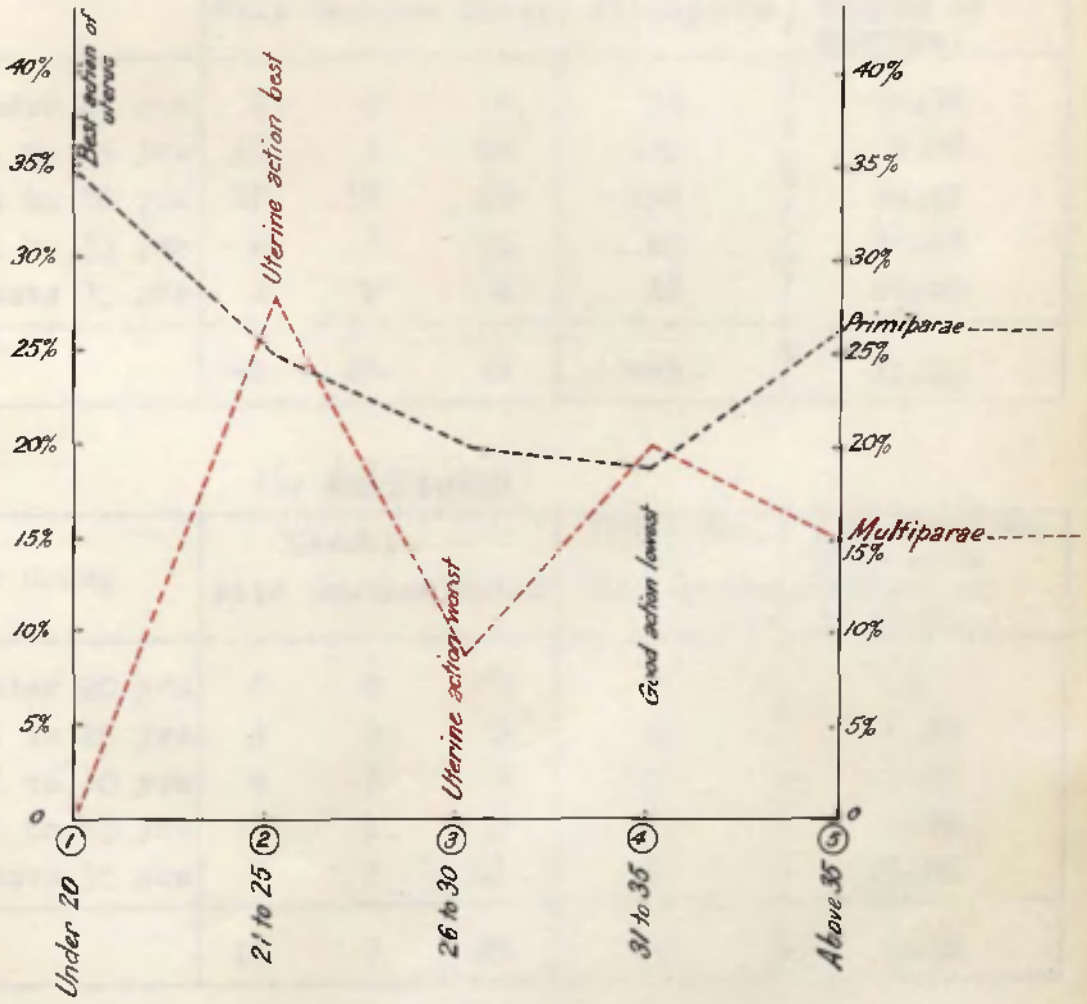


TABLE III

AGE AND PARITY IN RELATION TO UTERINE ACTION

Inertia

i. Primiparae

Age Group	Inertia			Total No. of Primiparae	% Primiparae with some degree of Inertia.
	Mild	Genuine	Total		
(1) Under 20 yrs	4	2	6	58	10.3%
(2) 21 to 25 yrs	15	3	18	181	9.9%
(3) 26 to 30 yrs	12	14	26	138	18.9%
(4) 31 to 35 yrs	8	7	15	49	30.6%
(5) Above 35 yrs	2	2	4	19	21.4%
	41	28	69	445	15.5%

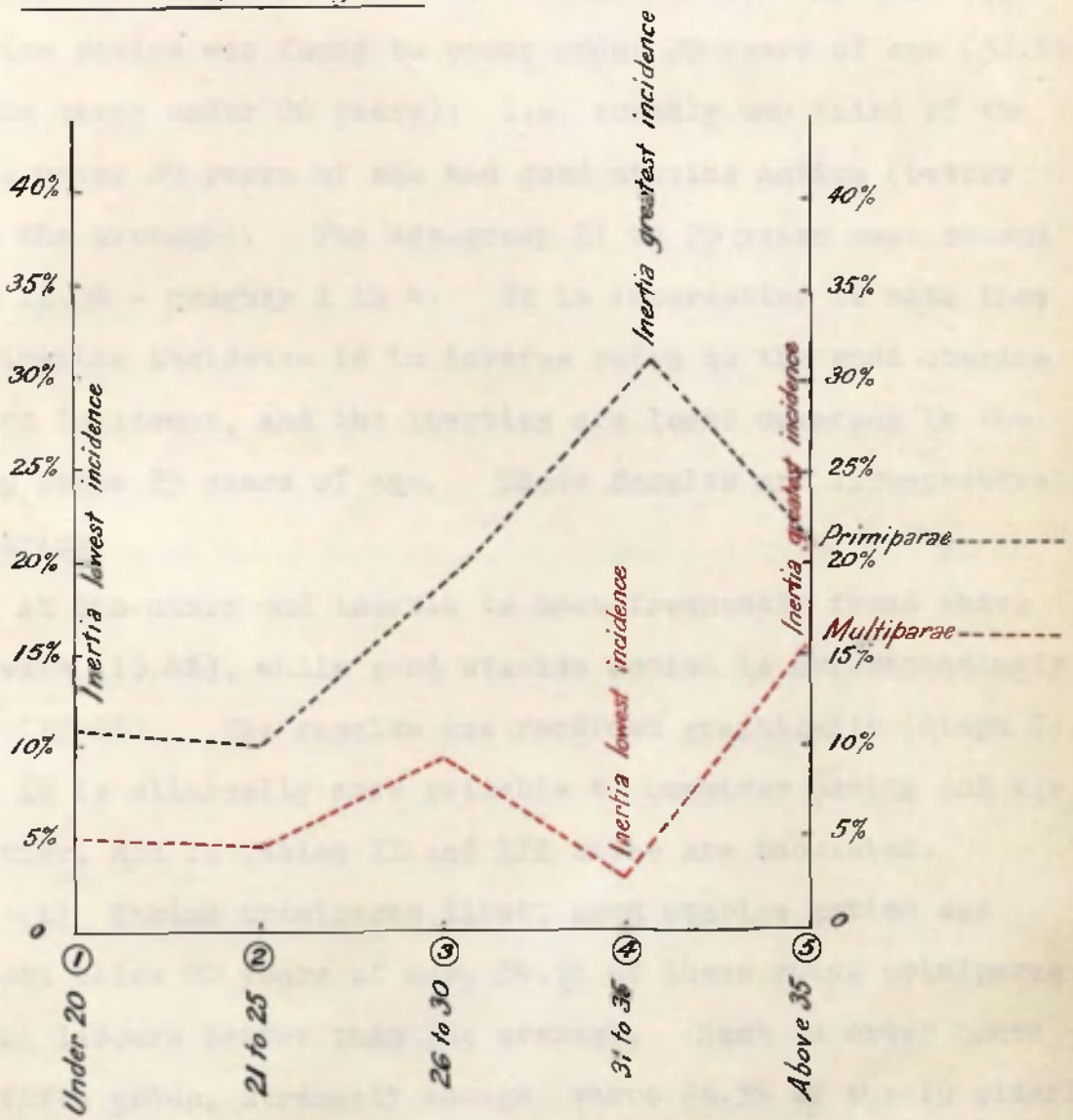
ii. Multiparae

Age Group	Inertia			Total No. of Multiparae	% Multiparae with some degree of Inertia.
	Mild	Genuine	Total		
(1) Under 20 yrs	0	0	0	3	0
(2) 21 to 25 yrs	3	0	3	62	4.8%
(3) 26 to 30 yrs	6	3	9	100	9.0%
(4) 31 to 35 yrs	1	1	2	70	2.8%
(5) Above 35 yrs	8	3	11	72	15.2%
	18	7	25	307	8.1%

GRAPH III

Age and Parity in Relation to Uterine Action

Inertia (all degrees)



Age Group

Discussion

(a) Age:

If one considers Table I, it will be seen that the best uterine action was found to occur under 20 years of age (32.8% of the cases under 20 years): i.e. roughly one third of the cases under 20 years of age had good uterine action (better than the average). The age-group 21 to 25 years came second with 26.3% - roughly 1 in 4. It is interesting to note that the inertia incidence is in inverse ratio to the good uterine action incidence, and the inertias are least numerous in the group below 25 years of age. These results are irrespective of parity.

At the other end inertia is most frequently found above 35 years (16.4%), while good uterine action is correspondingly poor (17.5%). The results are recorded graphically (Graph I).

It is clinically more reliable to consider parity and age together, and in Tables II and III these are tabulated.

(i) Taking primiparae first, good uterine action was highest below 20 years of age, 34.5% of these young primiparae having labours better than the average. Next in order comes the fifth group, strangely enough, where 26.3% of the 19 elderly primiparae had a labour better than the average i.e. less than

9 hours; next, the second group (21 to 25 years), then the third group, and lastly the fourth group. Hence the best group in primiparae is the below 20 group, the worst group in primiparae is the 31 to 35 years group. The elderly primipara in this series (above 35 years) is a very good second.

In the Inertia tables (all degrees of inertia), in Table III, the highest incidence of inertia in primiparae is found in the fourth age group - from 31 to 35 years of age. This corroborates the previous findings viz. that, in these cases, the worst age in primiparae is from 31 to 35 years. Next comes the fifth age group - above 35 years - where 4 of the 19 elderly primiparae developed some degree of inertia. The lowest incidence of inertia is found at the younger ages, the second age group (21 to 25 years) being lowest (9.9%) with the first age group close on its heels (10.3%)

Hence in primiparae, uterine action has been shown to be best below 20 years of age, uterine inertia least common below 25 years of age; and uterine inertia consistently most common in the age group 31 to 35 years.

(ii) Multiparae and Age-Groups (Tables II and III): In multiparae, uterine action is seen to be best (Table II) in the age-group 21 to 25 years (27.4%); next, the fourth age-group with 20%, then the fifth age-group with 15.2%, and lastly the third age-group with 8%. (The first age-group has been left out owing to the small number of cases, viz. 3 out of 307 multiparae).

hence the ages 26 to 30 years in multiparae represent the lowest group with good uterine action.

As regards Inertia (Table III) the highest incidence is found from 35 years upwards in multiparae (15.2%). Next in order comes the third age-group - 26 to 30 years - with 9%; followed by the second age-group 4.8%, the fourth age-group 2.8%, and lastly by the first age-group with no inertias below 20 years of age (only 3 cases out of 307). Hence, some degree of inertia is most common in elderly multiparae (above 35 years) and least common below 20 years in young multiparae. The next most favourable age in multiparae is from 31 to 35 years of age.

In comparing Tables II and III, therefore, it will be seen that multiparae above 35 years of age have a poor uterine action, below 25 years of age and (in Table III) between 31 and 35 years of age have a very good uterine action. In both tables the uterine action in the age-group 26 to 30 is indifferent.

To summarize:- (1) Uterine action is best in primiparae below 20 years of age, in multiparae below 25 years and between 31 and 35 years of age. (2) Uterine action is worst in primiparae between 31 and 35 years of age, and in multiparae above 35 years of age.

This is in accordance with accepted views. The uterus in the young woman, primipara or multipara, is in a fresh and

physiologically good condition. Its tone is good, fatigue is more easily combated and less likely to appear, and there is no weakening of its musculature by the appearance of fibrous and yellow elastic tissue found in elderly multiparae, in whom the proportion of muscular tissue is less owing to the admixture of involution deposits. Moreover, the nutrition of the uterine muscle is certainly better at the younger ages.

(b) Parity:

The parity has already been considered in relation to uterine action in conjunction with age-groups. Of the 752 cases in this series 445 are primiparae, and 307 are multiparae. Of the primiparae 108 out of 445 had good uterine action, i.e. 24% of primiparae had a labour lasting less than the average time; and of the multiparae 50 out of 307 had good uterine action i.e. 16.2%. Hence good uterine action is found, in this series, more often in primiparae in the proportion of roughly 1 in 4, as compared with 1 in 6 in multiparae (Table II).

Considering all degrees of inertia, 69 out of 445 primiparae developed some degree of inertia i.e. 15.5%; and only 25 out of 307 multiparae developed some degree of inertia i.e. 8.1%. Hence uterine inertia (all degrees, no matter how small) is found with greater frequency in primiparae than in multiparae: the proportion being 15.5% to 8.1%, or roughly 2 to 1

respectively. In this series, uterine inertia is twice as frequent in primiparae as in multiparae in all degrees of impaired uterine action. In genuine uterine inertia the proportion is almost 6 primiparae to 1 multiparae (vide Part IV "Uterine Inertia").

To summarize:- Primiparae are more prone to develop a uterine action less efficient than normal, and also (paradoxical as it may seem) to have a uterine action better than the normal: the two extremes of uterine action, very good and bad, are found more often in primiparae. The multiparae tend to steer a more uniformly normal course through labour.

(c) CO-EXISTING MATERNAL DISEASE

i. Not attributable to pregnancy.

(1) Cardiac Disease

TABLE IV

Nature of Lesion	Decompensation		Uterine Action			Result	
	i Present ii Absent		Good	Average	Bad	Mother	Child
Mitral Stenosis	i 5 ii 4	9	1 2	3 2	1 0	Normal Normal	Alive Alive
Mitral Regurgitation	i 0 ii 2	2	0 0	0 1	0 1	Normal Normal	Alive Alive
Cardiac Debility	i 2 ii 0	2	1 0	1 0	0 0	Normal Normal	Alive Alive
Total	13		4	7	2	Mortality and Morbidity nil.	

(2) Pulmonary Disease

TABLE V

Nature of Disease	Uterine Action		
	Good	Average	Bad
Pneumonia	0	1	0
Pulmonary Tuberculosis	0	1	0
Chronic Bronchitis	1	0	0
Bronchiectasis	1	0	0
	2	2	0

(3) Nervous Disease

TABLE VI

Nature of Disease	Uterine Action		
	Good	Average	Bad
Cerebellar Tumour	0	1	0
Epilepsy i.Major	1	0	0
ii.Minor	0	1	0
Cerebral Embolism	1	0	0
Disseminated Sclerosis	1	0	0
Deaf Mutism	1	0	0
Ante-Partum Insanity	2	0	0
	6	2	0

Discussion

i. Not attributable to pregnancy

(1) Cardiac Disease.

There are 13 cases in all suffering from Cardiac Disease. The commonest lesion is mitral stenosis, 9 cases, with 2 cases each of mitral regurgitation and cardiac debility. By cardiac debility is meant a general myocardial weakness not attributable to any valvular disease of the heart primarily, in which insidious signs of decompensation have appeared during pregnancy: the fatty heart, and the easily fatigued myocardium of the elderly multipara are examples of cardiac debility.

Only 2 out of the 13 developed inertia, in both cases mild, while 4 had uterine action better than the average, the remaining 7 having a normal labour. Seven of the cases developed clinical signs of decompensation, of whom only 1 developed inertia (mild), 2 were very good, and 4 average in labour.

Only 3 of the 13 cases were induced near term, the others going into labour normally about the expected date.

Hence cardiac disease would not appear to be a factor in the causation of uterine inertia. The cardiac patient as a rule has a fairly easy labour, and one is often surprised at the easy labours which these cases, in the main, enjoy. No

doubt a contributory factor is the fact that the babies are on the small side, though none of the cases in this section were more than a few weeks premature: consequently, delivery is usually normal or expedited by low forceps according to the patient's condition in the second stage. In addition, the majority of these cases with obvious cardiac lesions are rested for a few weeks before term, and put on a light diet, rich in glucose. The glycogen capacity of the muscle cell may safely be said to be very good. A further contributory factor, in addition to a carbohydrate rich diet, which all these cases had, is the exhibition of salts of calcium, all the cases receiving Calcium Lactate gr.x t.d.s. during their treatment in hospital; the tone of the muscle cell consequently benefits. The end result is a muscle cell which is probably replete with stores of glycogen and calcium salts, in addition to being possibly more irritable, as evidenced by the tendency for the cardiac subject to go into premature labour.

(2) Pulmonary Disease.

Only 4 cases of the series were clinically suffering from pulmonary disease of such severity as to warrant treatment in hospital. None of these cases developed inertia, 2 being average, and 2 better than the average. No conclusions can properly be drawn from such a small number of cases.

(3) Nervous Disease.

Some very interesting nervous conditions are found in this section. The case with cerebellar tumour had been in hospital 2 years previously, when a diffuse, probably gliomatous, tumour of the cerebellum was diagnosed by the neurological surgeon; she was blind, and intractable vomiting was present till mid-term. Labour ensued and terminated normally, apart from severe headache during the second stage, probably due to increased intracranial pressure.

The patients with major and minor epilepsy were kept under luminal and labour was normal in the latter, very good in the former.

The cerebral embolism mother was unmarried, and only 22 years of age. A complete left-sided hemiplegia supervened before admission to hospital, and a few days later the patient, who was unable to speak, went into labour. When discovered to be in labour the patient was near the end of the first stage; normal delivery ensued the child being at full term, and alive. No cause for the cerebral lesion could be found apart from a probable embolism. The labour was apparently easy, although the mother's condition was one of stupor throughout, though conscious.

The disseminated sclerosis mother was multiparous, with early spastic paraplegia. Delivery was very quick and speedy.

The deaf mute mother had been given a full drug induction at term, her friends being rather apprehensive of keeping her at home till term. Labour was very quick and better than the average, though the mother became very frightened if the nurse happened to leave the labour ward.

Ante-partum Insanity supervened in one case near term, in a primipara. Following two unsuccessful drug inductions, the membranes were ruptured. Labour was very short in duration, though anaesthesia à la reine was necessary during the second stage. The second case was in a third para at 32 weeks, the foetus being macerated. The labour was quicker than normal, lasting 2 hrs 30 mins in all. This patient was melancholic and depressed rather than exhilarated.

These cases offer a wide variety of nervous lesions, yet not one case of inertia or poor uterine action supervened. In the cases of deaf mutism and ante-partum insanity there was present fear and exhilaration respectively, with no adverse effect on the stage of dilatation. In the case with cerebellar tumour the patient was blind: her sensory appreciation was highly developed, fear was obviously present in suppressed form; and yet labour was uneventful. The epileptic cases were under maintenance doses of luminal; the nervous system in both can be assumed to be highly sensitive to unusual stimuli, and excitable; no adverse effect on labour was present. In

the patient with cerebral embolism the condition was one of mental hebetude rather than excitement, as a contrast to the above cases; and again delivery was normal. The disseminated sclerosis mother was highly nervous, though, being parous, fear was not present.

These cases serve to demonstrate the fact that labour, once initiated, will progress steadily and will terminate normally, other things being equal, no matter what coincident nervous disease is present. Both functional and organic nervous diseases are illustrated in this series, the organic diseases predominating. It would appear, then, that provided the uterine nervous connections are intact, labour will supervene uneventfully: none of the above cases had a lesion interfering with the normal reflex mechanism, which carried out its function without any variation from the normal.

The majority of the above cases are characterised by a highly excitable nervous system, which has been claimed to inhibit the progress of labour. The higher nerve centres are accredited with a purely inhibitory action over the lower, spinal, nerve centres, release of which central connections leads to an exaggeration of the reflex responses; but as this only applies to the voluntary nervous system there is no direct application of the principle in the case of the uterus. The

action of the higher centres is indirect, in that such psychological influences as fear, emotion, etc., may inhibit the normal reflex mechanism. It is not improbable that this inhibition is due to the liberation of adrenalin into the circulation; adrenalin is sympathetico-mimetic in action, and as such it inhibits the contraction of plain muscle. No such effect was noted in the above cases, indeed the reverse has been true, viz. that the hyper-excitability of the nervous system has led to quicker labours and stimulation of the reflex mechanism, rather than inhibition.

The local myogenic contraction is probably the chief factor at work, the local nerve connections being regulatory in action and helping in the co-ordination of uterine muscle action; external stimuli exert their effect in an uncertain manner and merely modify the contractions, but do not materially affect the outcome. The degree of inhibition which these external stimuli may give rise to is probably proportionate to the amount of adrenalin liberated into the circulation, a factor which is inconstant and subject to hormonic control in addition.

(4) Chronic Nephritis

TABLE VII

Uterine Action in Labour		
Good	Average	Inertia
1	2	1

(5) Uterine Fibroids

TABLE VIII

Case No.	Age/Parity	Duration of Labour	Uterine Action in Labour		
			Good	Average	Inertia
246	31/1	35 h. 10 m.			+
251	28/2	11 h.		+	
427	37/2	8 h. 5 m.		+	
509	26/2	3 h. 20 m.	+		
520	39/1	50 h. 15 m.			+
580	30/1	28 h. 10 m.			+
			1	2	3

Discussion

(4) Chronic Nephritis.

There are 5 cases of chronic nephritis in the series, of which only one had a labour quicker than normal, two were normal, and one case developed a very mild inertia. That with mild inertia occurred in a third para, the labour lasting 14 hours 20 mins, delivery of the child (premature) being normal. Dieting had been rigid for 8 weeks prior to onset of labour. Dieting was a factor in all five cases. The number of cases is too small to draw conclusions from.

(5) Uterine Fibroids.

Of the 6 cases of uterine fibroids which were diagnosed, 3 were in primiparae, 3 in multiparae. All the primiparae were above 30 years of age, the average age being 33, and all the primiparae developed inertia in some degree. In two of these primiparae, the inertia was no more than a moderate delay in the first stage with a forceps (low) in one of these due to severe secondary inertia; in the third case - an elderly primipara of 39 years of age - primary inertia was present, delivery being effected by forceps. In all primiparae the third stage was uncomplicated.

Of the multiparae, two were normal while the third was quicker than the average, no reason for the quick labour being

manifest apart from the comparative youth and good condition of the mother.

From the above results, though few, it is concluded that elderly primiparae with fibroid uteri are prone to develop uterine action which is not so good as one normally expects, and are liable to have a primary inertia. In multiparae, fibroid uteri are less liable to lead to interference with the mechanism of the uterus.

In considering the intricate arrangement of the uterine muscle fibres (see Part II Anatomy of Uterine Muscle) it is obvious that the interposition of a tumour will break up the continuity of the muscle layers, and will lead to inco-ordination of the waves of contraction, breaking them up and causing them to become irregular in time and weaker in force. The rhythmic nature of the contractions is partly lost, and the powerful wave of contraction dissipated and converted into smaller, less powerful, contractions. Moreover there is interference with the myogenic control, which depends upon the continuity of the muscle fibres.

(6) Anaemia.

TABLE IX

Case No.	Age/Parity	Duration of Labour	Uterine Action in Labour		
			Good	Average	Inertia
452	21/1	14 h. 45 m.		+	
499*	34/2	5 h. 30 m.		+	
552	25/1	7 h. 20 m.	+		
554	29/3	46 h. 45 m.			+
595	24/2	5 h. 5 m.		+	
744*	27/2	6 h. 10 m.		+	
			1	4	1

(7) Constitutional Disease

TABLE X

Nature of Disease	Uterine Action		
	Good	Average	Inertia
Syphilis		3	1
Diabetes			1
Extreme Obesity			1

Discussion

(6) Anaemia.

Of the 6 cases which were suffering from anaemia of such severity as to necessitate hospital investigation and treatment, 2 were of the macrocytic hyperchromic type - pernicious anaemia of pregnancy. Only one case out of the 6 developed inertia of a primary nature, a third para, delivery being normal, and the labour lasting 46 hrs 45 mins. The anaemia was secondary in type - a microcytic hypochromic anaemia due to malnutrition. One case had a very good labour - a primipara with a breech delivery; the four others were normal. There was no complication in the third stage in the way of undue loss of blood.

Again the series of cases of severe anaemia is too small to draw any conclusions, but according to the tabulated scanty data the majority of the cases had an uncomplicated labour. The blood supply of the uterus is so rich and free that it is difficult to conceive that anaemia per se will have any local detrimental action on the uterus in labour; rather will it exert its effect on the various systems, especially the cardiac system.

In relation to the blood supply to the uterine muscle it is permissible to discuss the role of ischaemia of the uterine muscle, and its relation, if any, to the cause of uterine pain.

Lewis showed many years ago, when carrying out work on

the cause of angina pectoris and intermittent claudication, that pain developed when unstriped and striped muscle was made to do work in the absence of a sufficient blood supply. This be ascribed to the liberation of some toxic factor elaborated in the metabolism of muscle working under a deficient blood supply, this factor not being produced when the muscle, striped or unstriped, was working under normal conditions, i.e. with its full blood supply.

It is well known that a uterine contraction causes a temporary blanching of the uterus, e.g. when seen to contract during Caesarean operation: in fact, every contraction of the uterus implies a temporary ischaemia of the uterine muscle. In the course of making investigations into the cause of menstrual pain, Moir[52] has shown that the intra-uterine pressure, as recorded by kymographic tracings, may exceed the systolic blood pressure taken simultaneously at the brachial artery; the uterine arteries, indeed, were shown to cease their pulsation temporarily during a pain (menstrual). The uterus, therefore, is rendered ischaemic temporarily during a "pain", or contraction, and the "pain" is not experienced by the patient till near the summit of the contraction, i.e. when the intra-uterine pressure is greater than the systolic blood pressure. Reasoning from this fact Moir suggests that the "pain" may, in part, arise as a result of prolonged ischaemia

of the uterine muscle during the contraction phase of its activity.

(7) Constitutional Disease.

Of the 6 cases of constitutional disease, four were due to syphilis, one to diabetes, and one to a case of extreme obesity, possibly due to endocrine dysfunction. None of the cases of syphilis had any paralytic symptoms; three were normal in labour; the fourth developed inertia, the case being an emergency sent in as "failed forceps". Delivery of this last case was by high forceps, a large child (10 lbs 8 ozs) being delivered stillborn through a slightly contracted pelvis.

Syphilis is not recognized as being a cause of uterine inertia; it is usually associated with abortion or premature labour, but these are due to associated vascular degeneration in the placenta.

Diabetes, on the other hand, may frequently cause uterine inertia, as in this one case. The reason is probably two-fold: (1) glucose storage in the muscle cells is impaired, and the store of energy for muscle work is rapidly exhausted; (2) insulin is accredited with the property of rendering muscle atonic. The former is probably the more important, though insulin deficiency may in some way upset the endocrine balance in the body.

Extreme obesity, due to any cause, will lead to poor uterine action, in that fatty infiltration and degeneration of all the tissues is inevitable, with as a result a lowering of the output of work from the organ concerned (in this case the uterus). In this case the patient, a primipara, weighed 19 stones; the distribution of the hair was of a masculine type, the adiposity was generalized and gross, the genitalia were normally developed and there was no alteration of voice. X-ray revealed no bony changes in the sella tursica, nor were any signs of organic nervous disease elicited. Menstruation was normal. A familial tendency to obesity was present, her mother weighing 21 stones at one time!

Following a week's rest in hospital owing to breathlessness and general health reasons, the patient went into labour spontaneously 12 days from term, rupture of membranes being premature. A mild primary inertia ensued; delivery was normal of a small child - 5 lbs 15 ozs, but secondary inertia developed in the third stage, the placenta being retained, necessitating manual removal owing to severe haemorrhage. The puerperium was normal.

This case illustrates the type of labour one expects to find in cases of gross obesity, and also in cases of endocrine dysfunction characterized probably by hypo-pituitarism. The mild primary and the secondary inertia in the third stage give support to the theory that the pituitrin concentration in the blood was sub-normal in this case; such a statement can only

be proved biochemically and merely inferred clinically. The gonadotropic group of hormones may probably be involved as well as the posterior pituitary hormone, in that deficiency of production of prolan A would lead to a subnormal production of oestrin with the result that the uterus would not be fully sensitized to the action of pituitrin.

The number of cases of constitutional disease is insufficient to merit any conclusions being made.

(c) CO-EXISTING MATERNAL DISEASE

ii. Attributable to Pregnancy

(1) Albuminuria of Pregnancy

TABLE XI

Case No.	Age/ Parity	Degree of Album. Mild	Mod.	Severe	Uterine Action Good	Average	Inertia	Notes
2	22/2	+				+		Diet
17	26/2		+				+	(Diet Malnu- (trition ++
20	35/7	+				+		Diet
67	23/1		+				+	(No diet in (hosp. Slight (at home
69	19/1	+				+		No diet
102	35/1	+				+		(R.O.P →R.O.A (diet
104	26/1	+				+		(Malnutrition (diet
109	26/1		+			+		Diet
111*	30/2			+		+		(No diet (Accidental Haem.
116	39/3	+				+		No diet
118*	42/10			+		+		No diet
130*	40/9			+		+		No diet
131	28/2	+			+			Hydramnios Diet
136	25/1	+				+		Diet
140	34/1			+		+		(No diet (A.P.Haem.
141	32/2			+		+		No diet
146	32/1	+				+		Diet
151	27/2	+				+		Diet
159	32/1	+				+		No diet

Case No.	Age/ Parity	Degree of Album. Mild	Mod.	Severe	Uterine Action Good	Average	Inertia	Notes
189	23/1		+		+			Diet
192	28/1			+	+			Diet
215	19/1	+			+			Diet
229*	24/1			+		+		Diet
231*	33/1	+				+		Diet
237	42/13			++			+	Pre-eclamptic No dieting
239*	39/2			++		+		Diet
247	29/1			+	+			(Painless (strict diet
248	32/1	+					+	R.O.P.
253*	34/1			+		+		(Quick 1st (stage. Diet
255*	23/1			+		+		Diet
252	32/2			+	+			(Diet (Painless (1st stage
260	25/1			+		+		A.P.Haem. No diet
264	24/1	+			+			Diet
269	37/3	+				+		Diet
272	31/3			+	+			(Diet (Painless (1st stage
275	36/5		+		+			Diet
281	22/1			+	+			(Premature (No diet
303*	27/2	+				+		Diet
328	39/3		+			+		Diet
353	24/1		+		+			Diet
375*	26/4			+		+		A.P.Haem. No diet
386*	31/1	+				+		Diet
390	25/3		+			+		Premature Diet
401	30/2			+++	+			No diet Eclampsia

Case No.	Age/ Parity	Degree of Album. Mild	Mod.	Severe	Uterine Action Good	Average	Inertia	Notes
420	37/2		+		+			(Diet (Painless (1st stage
432	27/1			+		+		Diet
424	33/2		+		+			Diet
446	25/1	+			+			(Hydramnios (No diet
456	25/1	+			+			(No diet (Premature
457*	24/2	+				+		Diet
462	20/1			+			+	Diet
466	34/2	+				+		Diet
468*	251		+			+		Diet
482	22/1			+		+		No diet
486	23/1			+	+			Diet
491	24/1			+		+		Diet
[502	28/1	+					+	(Justo Minor (pelvis (diet]
506	30/3			+		+		(Diet (Macerated
515	32/1		+			+		Diet
537	19/1	+			+			No diet
538	31/3	+			+			Diet
542	20/1	+			+			No diet
554	29/3	+					+	(Malnutrition (No diet
555	30/1	+				+		(Diet (Premature
558	24/1			+	+			(Diet (Premature
566	30/6	+				+		(Premature (Diet (Macerated
567	23/1			+	+			P.P.H. No diet
570*	29/1		+			+		Diet
573	30/2			+	+			(Diet (Premature

Case No.	Age/ Parity	Degree of Album. Mild	Mod.	Severe	Uterine Action Good	Average	Inertia	Notes
586	39/13			++			+	No dieting
588	22/2			+			+	A.P.H. No diet
596	37/3		+			+		(Easy 1st stage. Diet
612	23/1			+	+			(Diet (Premature
620*	25/1			+		+		Diet
635	35/1			+	+			(No diet (Premature
642	31/1		+			+		Diet
659	28/2			+++			+	(No diet (Eclampsia (Flat small (pelvis
676	26/1			+	+			(Diet. Twins (Premature
689	41/6		+			+		Dieting
695*	37/2		+			+		(Painless 1st (stage. Diet
709*	29/2		+			+		Diet
734*	30/1		+			+		Diet
740	37/6	+				+		Diet
697	24/3	+				+		Diet
702	27/1	+			+			Diet
713*	25/1	+				+		Diet
638*	37/3	+				+		(Diet (Premature
254	41/3		+		+			(Diet (Macerated
232	25/1	+					+	(Hydramnios (No diet
89		35	20	34	28	50	11	

20*

Albuminurias

TABLE XII

Total No. of Albs.	Type of Albuminuria			Uterine Action			
	Mild	Mod.	Severe	Very good	Good	Aver.	Inertia
89	35 (39.3%)	20 (22.4%)	34 (39.2%)	28 (31.4%)	20 (22.4%)	30 (33.7%)	11 (12.3%)

TABLE XIII

Cause of Delay in "Inertia" Cases

		Diet
Hydramnios	1.	No diet
Pre-eclampsia and Eclampsia	2	No diet
Ante-Partum Haemorrhage (Accidental). . .	1	No diet
Elderly multipara (39/13)	1	No diet
Malnutrition	2	No diet
Generally Contracted Pelvis	1	Diet
Generally Contracted Flat Pelvis	1	No diet
Malpositions (R.O.P)	1	Diet
Unknown	1	Diet
Total	11	

ii. Attributable to Pregnancy

(1) Albuminuria of Pregnancy

The albuminurias have been divided into three columns, viz. "mild", "moderate", and "severe". A "mild" case is reserved for those with less than 1 part Esbach, a trace of, or no, oedema, and a blood pressure not above 140 mms. Hg. systolic.

A "moderate" case is one in which the albumin and/or the oedema are more severe - up to 8 pts. Esbach, a blood pressure up to 180 mms. of Hg. systolic, with moderate oedema.

A case is considered to be severe when the Esbach reading exceeds 8 pts. Esbach, the blood pressure is above 180 mms. of mercury systolic and visual disturbances and severe vomiting are present, with probably severe oedema. Two signs (++) indicate a pre-eclamptic condition; three signs (+++) indicate eclampsia.

It is impossible to group all the albuminurias into their true category according to the above criteria, but a rough clinical classification has been made, e.g. a case with no oedema, 3 gms. Esbach per 100 c.c.s urine, and a B.P. of

190 mm. Hg. is considered to be pre-eclamptic for clinical purposes (excluding chronic nephritis) whereas a case with moderately severe oedema, 5 parts Esbach, and a B.P. of, say, 140 mm. Hg. systolic, is considered to be only of moderate severity. A certain amount of overlapping must be encountered in any attempt to classify albuminurias according to the above criteria, but the deciding factor in a doubtful case is the investigator's weighing up of the patient's condition clinically. Hence the accompanying results may be considered fairly accurate in their classification.

The criteria adopted for good, bad, and average uterine action are as follows:

(1) "Good" : a labour lasting less than 9 hours in a primipara, 3 hrs 30 mins in a multipara.

(2) "Average": labour lasting 12 to 18 hours in a primipara, 6 to 8 hours in a multipara.

(3) "Inertia": labour lasting more than 30 hours in a primipara, 14 hours in a multipara. All degrees of inertia are considered whether mild, moderate, or severe.

In certain cases the time factor is less important than the clinical estimation of the progress of labour, e.g. a multipara may have a quick first stage of, say, 4 hours, with a subsequent secondary inertia; the labour may have to be terminated by an instrumental delivery; but the total duration of the labour,

according to the time factor, may be within normal limits. Again the investigator must use his clinical judgment in assessing the action of the uterus in labour.

Cases marked with an asterisk (*) indicate lesser degrees of good uterine action, which according to the above criteria, are included in the "average" group. They are really better than the average, and are reserved for (1) multiparae with a labour lasting less than 6 hours (but more than 3½ hours) and (2) primiparae with a labour lasting less than 12 hours (but more than 9 hours). For purposes of discussion the "good" class will be considered as "very good", and the intermediate class as "good".

It is recognized that the term "inertia" is used in a very broad sense here: under the heading "inertia" are included all labours which have lasted longer than the usually accepted duration. Some are no more than a simple delay, others a very mild inertia, while a small few are severe primary inertias as usually understood by that term. The heading "inertia" therefore is one of convenience in classifying the results and simply means "uterine action less good than the average".

Discussion

Albuminuria of Pregnancy

89 cases out of the series of 752 cases suffered from albuminuria, by far the most frequent disorder of pregnancy; almost 12% therefore, or 1 in 8 developed albuminuria. Of these 89 cases, 35 were of very mild severity (39.3%); 20 were of moderate severity (22.4%); and 34 were severe in degree (39.2%).

The effect of albuminuria on uterine action is striking, for 48 cases had a labour better than the average (53.8%), of which 28 were "very good" (31.4%), i.e. less than 9 hours or 3½ hours for primiparae and multiparae respectively, and 20 were "good" (22.4%), i.e. between 9 and 12 hours, or 3½ and 6 hours in primiparae and multiparae respectively. 30 cases were of "average" duration (33.7%), while only 11 cases come into the "inertia" class (12.3%).

On further analysing the "inertia" class (all degrees of poor uterine action) the result is still more striking, for only 3 out of the 11 are purely albuminurias - one pre-eclampsia, one eclampsia, and one accidental haemorrhage; in all the other 7 there is some other factor at work which might in itself be a cause of delay. In one case the cause is unknown.

If this unknown cause of inertia can be included, a total of 4 out of 89 cases of albuminuria developed inertia (4.5%). Of the 11 inertia cases 5 occurred in severe, 2 in moderate, and 4 in mild cases of albuminuria.

From the above results it is concluded that in albuminuria of pregnancy:

(1) Uterine action is better than the average in more than half the cases (53.8%).

(2) Inertia in any form is the exception, and when it does occur can usually be ascribed to some other complicating factor.

(3) Inertia, when it does occur, bears no relation to the severity of the albuminuria.

The cause of these easy labours in albuminuria of pregnancy is difficult to find. Undoubtedly the smaller size, and often the prematurity of the children contribute towards making labour quicker; but this applies mainly to the second stage of labour - the stage of expulsion. Obviously the size of the child should not affect markedly the stage of dilatation, which is appreciably shortened.

(1) Endocrine factor.

The endocrine factor has been invoked to account for albuminuria of pregnancy. At the present day it is accepted that the endocrine factor is becoming more important in the causation of albuminuria. It is known that pituitrin, if

given in tolerably large doses will produce a clinical picture not unlike that found in albuminuria - disorders of vision, vomiting, oedema, and even fits of an eclamptic nature. The connection between an excess of posterior pituitary hormone and the production of toxaemic signs and symptoms is too striking to be neglected. It is possible, then, that in albuminuria of pregnancy there is present some hormonic imbalance as a result of which posterior pituitary hormone is present in excess in the blood stream. The effect of this will be to increase the supply of the oxytocic factor to the uterine muscle, whereby its tone is increased; its expulsive power is increased, the contractions are stronger and more efficient than normally, and the stages of dilatation and expulsion are expedited.

The myometrium-inhibiting action of progestin and the motility-stimulating action of oestrin are contributory factors which must be considered along with the main oxytocic factor, pituitrin, but their rôle (progestin and oestrin) is secondary to that of the pituitary; oestrin probably preparing the myometrium for the subsequent action of pituitrin.

(2) Hypocalcaemia.

It has been claimed that there is a hypocalcaemia in albuminuria of pregnancy.(58) Here, in a series of only 10 cases of varying degrees of albuminuria, the blood calcium was determined by skilled independent laboratory investigation,

and in none of the cases was a hypocalcaemia demonstrated beyond the slight fall in blood calcium level normally found in pregnancy; one of the specimens was taken from an eclamptic patient, but no hypocalcaemia was found.

If a critical hypocalcaemia does exist in albuminuria - and the literature is in a very confused state on this subject - then there will result an increased neuromuscular excitability such as is found in the hypocalcaemia of tetany, of parathyroid or rickets origin, with similar tetanic fits.

The ratio of calcium to potassium content is dealt with in Part II of this Thesis (Physiology of Uterine Muscle), a constant ratio being necessary for optimum muscular activity.

If the increased neuromuscular excitability is at an optimum, then the increased tone and excitability of the muscular and nervous tissues would lead to more frequent and strong contractions, with a speedier labour ensuing. This would explain, in conjunction with the increased amount of posterior pituitary factor in the blood stream, the great tendency for albuminurias to go into premature labour: the same causal agent is at work. If on the other hand the increased neuromuscular excitability is at a maximum, then tetanic spasms would result. This state could theoretically be produced if the blood calcium were critically low, the critical value being estimated by some to be 2:5 mgm. of

calcium per 100 c.c.s serum. Indirect evidence in support of a hypocalcaemia being contributory in albuminuria towards premature labour or quick labour, has been supplied by Nahmmacher[59] who has shown that by feeding experimental animals on a diet rich in calcium (given with Vitamin D) they went over term by eight to ten days, i.e. he produced post-maturity by a calcium excess in the diet.

(3) Diet.

The influence of dieting is discussed elsewhere (vide "Painless Labour" infra). The results do tend to show that light diet (salt poor) leads to a less prolonged and more easy labour. It has, furthermore, been noted that women who have been staying in hospital for some weeks prior to labour and living on a full and unrestricted diet, with little work to do, have a tendency to go beyond the expected date of delivery. Moreover, it will be noted in the section on Socio-Economic factor that more inertias occurred amongst the so-called "private patients" who were in a much better state of nutrition than the more unfortunate (financially) "general patients".

(4) Toxaemia.

If one knew the aetiology of albuminuria, one would be helped materially in explaining the quick easy labours usually found to occur in albuminurias of pregnancy. Obviously, from

the above results the degree of albuminuria does not alone seem to influence the duration of labour; the duration of labour is not proportional directly or inversely to the amount of albumin in the urine, nor to the severity of the toxaemia.

One of the signs common to all albuminuric cases is oedema - from a mere trace to gross oedema. If this oedema is generalized, as it is, it will be found in the tissues of the pelvic floor and the parametrial cellular tissue. The softening and lessened resistance of the tissues resulting will contribute to quicker opening up of the tissues of the pelvic floor, with less trauma and hence less pain: but this will apply mainly to the second stage of labour when the foetus is descending through the pelvis, although descent is progressive to a lesser degree during the first stage of labour.

It is interesting to note here that the administration of oestrin has been shown to lead to a marked vasodilatation and an accumulation of interstitial fluid in the endometrium and myometrium (of the rabbit), with subsequent oedema. This phase is temporary and is followed by increased muscular activity, the intermittent muscle contractions helping to prevent the congestion due to the vasodilatation, and removing the plasma proteins and tissue fluid by way of the lymphatic vessels.(60)

Should any other factor be at work, therefore, and inter-

fering with the tissue exchange of lymph, a condition of oedema of the interstitial tissue of the myometrium will be produced: and such a state of affairs is found in albuminuria of pregnancy.

The microscopic and macroscopic changes in the myometrium in severe toxaemia and pre-eclamptic and eclamptic conditions, viz. cloudy swelling, capillary haemorrhages etc., exclude the possibility of toxaemia increasing the efficiency of the musculature - rather the pathological changes have the reverse effect of impairing its efficiency. Only those factors which influence the efficiency of muscle contraction are at work, and of these posterior pituitary hormone and calcium acting on an oestrin-sensitized muscle cell are the only two factors with any claim to increasing muscle output.

The cause of good or bad uterine action must remain problematical until the greater problem of how labour is brought on is solved. It is probably under strictly hormonal control, such factors as calcium values and dietary restrictions being only contributory factors, and not in themselves of primary importance in the initiation of uterine muscle action.

To summarize, therefore:

(1) It has been shown that the greater percentage of cases of albuminuria in this series have a comparatively easy labour, with a minimum of obstetrical interference.

(2) Some contributory factors in bringing about this result are discussed, with particular reference

to the influence of a salt-poor diet and serum calcium values on the character of the labour.

Relation of Albuminuria to Painless Labour.

The incidence of "painless", or comparatively painless, labours has been noted: in all the albuminurias a note has been made as to whether or not the cases received dietetic treatment, thus a note has been made - "diet" or "no diet". No statement as to the degree of dieting will be found, but it may be assumed that if the case is one of mild albuminuria, the patient would be on a light diet in which only protein foods were excluded; if of moderate severity, the diet would consist of bland fluids, including milk, with possibly fish or chicken according to progress; if of extreme severity then only glucose and water would be given. It is obviously impracticable to enter the details of the dieting in the tabulated records, hence the degree of dieting will be assumed to correspond to the severity of the case.

None of the diets were "salt free"; a more exact phrase would be "salt poor", as the majority of the patients would not tolerate a salt free diet and became discontented, either adding salt themselves surreptitiously, or importuning the other patients to supply them with the commodity. Also, salt substitutes were a failure. Hence a more correct phrase is "salt poor diet".

Similarly it was found to be impossible to have a control

series of normal healthy pregnant mothers on a salt poor or salt free diet - these controls were free to move about the ward and it was found impossible to keep a strict eye on their dietary. (There are often a few healthy mothers awaiting full term in municipal hospitals who could be utilized as controls - these include illegitimate, or destitute women, or women who are domiciled well out in a country district and unable quickly to be transported into hospital when labour begins.)

Five truly painless first stages are recorded.

1. Case No.247.

Primigravida, 30 years of age. Admitted with 10 gms. Esbach, Blood-pressure 210/120, moderate oedema. Treated by glucose and water only for 4 days, with intravenous calcium laevulinate 10%. Spontaneous labour lasting 6 hours in all. Did not complain of any pains - merely slight backache - till second stage began. Duration 6 hours. Responding to treatment: Blood-pressure 155/95, Esbach 4 gms on morning of labour.

2. Case No.252.

Para 2, 32 years of age. Unwilling to enter hospital and being treated at home by own doctor: salt poor diet. At term, Blood-pressure 200/110, albumin 2 pts Esbach, slight trace oedema. Told to come in for induction. Intravenous calcium laevulinate given personally at 2 p.m. - not then apparently in labour. Asked for bed-pan, and membranes ruptured; found to be almost fully dilated. Child born at 4.40 p.m. weighing 8 lbs 1 oz. Pains only during brief second stage. Duration of

labour - about 2 hours approximately. Not responding to home dietetic treatment.

3. Case No.272.

Para 3, 31 years of age. Admitted with Blood-pressure 150/120, Esbach 6 pts, oedema moderate. Water and glucose only for 2 days, previous light diet at home. 10 days from term, spontaneous labour. No complaint of backache or pains, merely urge to go to stool at beginning of second stage. Child 5 lbs 4 ozs. Duration 1 hour 20 minutes. No response to treatment at home. Eclamptic seizures (three) following birth. Puerperium otherwise normal.

4. Case No.695.

Para 2, aged 37. 37 weeks pregnant. Blood-pressure 150/90, Esbach 2 parts, oedema a trace. Responding to dietetic treatment after 1 week in hospital. Premature spontaneous labour. No complaint of pains till second stage began. Duration of labour 5 hours 45 minutes. Premature child 4 lbs 8 ozs alive.

5. Case No.420.

Para 2, aged 37. 38 weeks pregnant. Blood-pressure 180/110, Esbach mere trace, oedema moderate. Previously treated in hospital for 4 weeks, but readmitted after 2 weeks' home dieting, for surgical induction. Dieting in all for 12 weeks prior to labour. Membranes ruptured (surgically) at 6 p.m.; delivery at 10 p.m.; no dilatation when membranes ruptured. Slight backache following induction followed by pains only when in or near second stage (30 mins). Child 6 lbs 8 ozs. Response to treatment while in hospital.

In considering the above 5 cases it will be seen that four were multiparae, only one being a primipara. Three of the cases responded to treatment in hospital, the other two were treated as out-patients, and it is probable that they were not having efficient treatment. None of the cases were considered to be severe albuminurias; they did not come under the pre-eclamptic class.

In 1933, Reeb and Israel[61] published their results, which included 10 primiparae and 10 multiparae, all albuminurias on salt poor - not salt free - diet. They stated that a salt free or a salt poor diet diminished the duration as well as the pain of labour, the salt free diet giving the better results.

In 1935 Karpati[62] carried out a similar investigation, and concluded that a salt free diet, in cases of albuminuria of pregnancy, resulted in a short and painless stage of dilatation. Those who improved clinically by medical treatment prior to labour did have shorter and more painless stages of dilatation; but those nephropathies which did not respond clinically had no difference in the character of labour. The more rapid and thorough the improvement the more noticeable the shortening of the period of dilatation and its painlessness. In primiparae the first stage was reduced to 3 to 4 hours, in multiparae to one to one and a half hours.

In this series of albuminurias, 28 had a very good uterine

action, i.e. a total duration of labour lasting less than 9 hours in primiparae, and 3½ hours in multiparae (giving approximately a first stage of 7 hours and 3 hours respectively) Of these 28, 20 had a salt poor diet, whilst 8 had no dieting; and of these latter with no dieting, 3 were premature labours, 1 was complicated with hydramnios, 1 was eclamptic, 1 was an accidental haemorrhage, and only 2 were straight forward "no diet" albuminurias. Hence, including the above complications, 71.4% of the very good uterine action column had a salt poor diet, while the remaining 28.6% of the very good labours had no diet.

Twenty cases had good uterine action, i.e. a labour lasting less than 12 hours, but more than 9 hours in primiparae, or less than 6 hours but more than 3½ hours in multiparae. Of these 16 had a salt poor diet, the remaining 4 being accidental haemorrhage cases with no diet, hence, if these 4 are excluded as pathological uterine action, 100% of the good labours had a salt poor diet.

Taken together, if the five cases of accidental haemorrhage are excluded, there were 43 cases of albuminuria with labours quicker than the normal, and of these 36 had a salt poor diet, i.e. a corrected figure of 83.7%.

Turning to a consideration of the dietary in the cases of inertia occurring in albuminuria, 6 cases out of the 11 cases

of inertia had no dieting. Excluding the 3 cases with disproportion and malpresentation, of the remaining 8 cases 7 had no dieting, i.e. 87.5% of the uterine inertias had no dietary restrictions.

To summarize:

1. Five "painless" labours are recorded. All the cases were on a salt poor diet, three of them definitely improving under hospital treatment, one improving under hospital treatment but relapsing under home treatment, and one being dieted, probably inefficiently, at home.
2. Of 43 cases of albuminuria with labours quicker than normal, 83.7% were on a salt poor diet (corrected figures).
3. 87.5% of the cases of uterine inertia occurring in albuminuria had no dietary restrictions.

From a consideration of the above results, it is seen that in albuminuria of pregnancy a salt poor diet contributes towards a labour quicker than the average. Albuminurias not so treated have a greater tendency to develop inertia. As regards painless labour, only five cases out of 89 cases of albuminuria were truly painless in their stage of dilatation; in albuminuria there is therefore a greater tendency towards painless dilatation than in normal pregnancy.

(2) Pyelitis of Pregnancy

TABLE XIV

Case No.	Age/Parity	Duration of Labour	Uterine Action in Labour		
			Good	Average	Inertia
541	21/1	26 hrs 50 m.		+	
743	19/1	6 hrs	+		
			1	1	0

(3) Pernicious Anaemia of Pregnancy

TABLE XV

Case No.	Age/Parity	Duration of Labour	Uterine Action in Labour		
			Good	Average	Inertia
499	34/2	5 hrs 30 m.		+	
744	27/2	6 hrs 10 m.		+	
			0	2	0

Discussion

(2) Pyelitis of Pregnancy

(3) Pernicious Anaemia of Pregnancy

Only 2 cases of pyelitis are recorded as having in-patient treatment: cases of mild severity were treated as out-patients, the signs not being such as to merit treatment in hospital. Accordingly, no conclusions can be drawn.

Similarly, two cases of macrocytic hyperchromic anaemia of pregnancy occurred in the series. In neither did inertia develop in any degree, both having labours of average duration. Both occurred in multiparae. Again, the numbers are too small to merit discussion.

(4) Ante-Partum Haemorrhage

TABLE XVI

Case No.	Age/ Parity	Ante-Partum Haemorrhage Accidental	Placenta Praevia	Uterine Action in Labour Good	Average	Inertia
81	40/3	+ C			+	
140	34/1	+ C			+	
141	32/2	+ R			+	
260	25/1	+ R			+	
375	26/4	+ C			+	
384*	37/4		+	+		
408	24/1	+ Traum.		+		
434	23/3		+	+		
482	22/1		+	+		
528	36/4		+	+		
529	26/1		+	+		
546	31/2		+		+	
567	23/1		+	+		
588	22/2	+ C				+
629	30/1		+		+	
671	32/2		+	+		
333	32/3		+		+	
17		7	10	8	8	1

C: Concealed Accidental Haemorrhage

R: Revealed

Traum: Traumatic Accidental Haemorrhage.

(4) Ante-Partum Haemorrhage.

Altogether there are 17 cases of ante-partum haemorrhage, of which 7 are of accidental haemorrhage and 10 of unavoidable haemorrhage.

The treatment of the cases of accidental haemorrhage is as follows:

Membranes ruptured	2
Membranes ruptured and pituitrin	1
Spontaneous labour	4

Good uterine action resulted in one case only: an unsuccessful external version at 36 weeks was followed by partial separation of the placenta.

Average uterine action was present in five cases, whilst a very mild inertia followed in one case which had been treated by rupture of the membranes and pituitrin (0.25 c.c.s of pituitrin given half-hourly till a total of 2 c.c.s given, or till labour contractions begin).

The treatment of the cases of placenta praevia is as follows:

Ruptured membranes and pituitrin	2
Bipolar podalic version	2
Internal podalic version	5
In spontaneous labour and allowed to proceed. .	1

Good uterine action resulted in 6 out of the 10 cases, viz. in one case where the membranes were ruptured and pituitrin given,

in one where bipolar podalic version, and in four where internal podalic version, was performed; and lastly, in one case where labour supervened spontaneously and was allowed to progress without further interference.

Average uterine action resulted in three cases, treated by rupture of membranes in one, bipolar podalic version in another, and internal podalic version in a third.

No degree of inertia occurred in any of the cases.

From a consideration of the above results it is seen that uterine action was much better in placenta praevia than in accidental haemorrhage. It was noted, also, that the degree of pain experienced during labour contractions was much less in placenta praevia than in accidental haemorrhage, one case of lateral placenta praevia being virtually "painless" - the estimated duration of pains being only 25 minutes (multipara). The reasons for this better uterine action are probably as under:

(1) Method of Treatment. It will be seen that in 9 out of the 10 cases of placenta praevia there was increased pressure exerted on the cervix and paracervical ganglia. In 7 of the cases a leg was pulled down to plug the lower uterine segment: and in the other two cases the membranes were ruptured and pituitrin given in cephalic presentations, the head being thereby pressed down more firmly on the cervix.

The reinforcement of the reflex arc by stimulation of the sensory component in the paracervical ganglia undoubtedly contributes to more efficient uterine action, the same result being seen in surgical induction by rupture of the membranes, insertion of hydrostatic bags and other artificial dilators into the lower uterine segment, and in stretching the rim of the cervix in sluggish labour by running the finger around the os.

(2) Increased vascularity of the lower uterine segment and pelvic floor structures. In placenta praevia the increased blood supply to the lower segment possibly leads to quicker dilatation, the uterine muscle and cervix being already rendered soft and boggy before the onset of labour contractions. There is less resistance offered to the dilating forces: and similarly the structures of the pelvic floor are more easily stretched and opened up by the descending part, owing to this increased "succulence" of the tissues. This increased vascularity and softening of the birth canal structures may account in no small measure for the more easy, less painful, labours which were found to ensue in cases of placenta praevia.

(3) In placenta praevia there is no interference with the uterine muscle efficiency, as occurs in accidental haemorrhage. In cases of accidental haemorrhage of the concealed variety, the muscle lamellae may be ploughed up by haemorrhages which

vary from rupture of small capillary vessels with extravasation of a few corpuscles, to rupture of larger vessels with rupture and disintegration of muscle continuity by blood clot. The efficiency to contract of the uterine muscle is seriously impaired: the increased intra-uterine tension which is found in this condition still further decreases the efficiency of the musculature by producing a condition almost of paralytic over-distension and loss of tone due to over-stretching. Of the 7 cases of accidental haemorrhage in the series 4 were concealed, 2 were revealed, and 1 was traumatic in nature: the good uterine action was found to occur in the case of partial separation of the placenta (revealed haemorrhage).

A factor which still further interferes with the muscle efficiency is the toxaemia which invariably is found in accidental haemorrhage. The muscle cell which is the seat of cloudy swelling, hyaline degeneration, and other toxic changes cannot compete with the healthy, well nourished, muscle cell of the uterine wall found in placenta praevia.

(4) Lastly, the type of subject is important. The patient suffering from accidental haemorrhage is usually parous, elderly, and it may be debilitated from the toxaemia. The placenta praevia subject is more often young, a primigravida and previously in generally good condition. The constitutional factor is much better in the latter.

To summarize:

(1) Of 17 cases of ante-partum haemorrhage, 10 were due to placenta praevia, 7 to accidental haemorrhage.

(2) Uterine action is more efficient in placenta praevia than in accidental haemorrhage, probably due to -

i. Method of treatment - measures which act by exerting pressure on the cervix lead to quicker and better labours.

ii. Increased vascularity of the lower segment, cervix, and pelvic floor in placenta praevia.

iii. The pathological changes in accidental haemorrhage reduce the efficiency of the musculature.

iv. The type of subject - placenta praevia subject is not debilitated by age, toxaemia, or parity, as a rule.

(d) Socio-Economic Factor.

It has been pointed out that the cases are divided into two categories: (1) "Private" i.e. patients who pay a certain fixed amount for which they receive certain privileges not enjoyed by the other class. These patients are in the main drawn from the middle classes and from those in moderately comfortable, though by no means wealthy, circumstances. (2) "General" patients, who pay according to their means: who may be impoverished - when they pay nothing, or working class women - who may pay anything up to "full costs". A certain amount of overlapping will occur between the good type of general patient and the "pseudo private patient" who can just barely afford the fees, but for clinical purposes, the distinction between private and general patient is a rough index of the socio-economic factor.

Results

TABLE XVII

Total number of cases in series 752

i. Private patients . . . 293 (i.e. 38.9%)

ii. General patients . . . 459 (i.e. 61.1%)

(a) Good	355	(47.3%)
(b) Fair	71	(9.4%)
(c) Poor	33	(4.4%)
Total. .	459	(i.e. 61.1%)

"Good" - socio-economic factor quite good.

"Fair" - socio-economic factor not so good - mainly intermediate between "good" and "poor".

"Poor" - socio-economic factor very bad - refers to those who are impoverished and almost destitute.

TABLE XVIII

A. Socio-economic Factor in Relation to Good Uterine Action

Total number of cases with good uterine action 158

(a) Total number of Private Patients with good uterine action 54 (34.1%)

Total number of Private Patients in series 293

% Private Patients with good uterine action 18.5%

(b) Total number of General Patients with good uterine action 104 (65.9%)

Total number of General Patients in series 459

% General Patients with good uterine action 24.8%

TABLE XIX

B. Socio-economic Factor in Relation to Inertia

Total number of cases of Inertia (all degrees) 94

(a) Total number of Private Patients with some degree of inertia 50

Total number of Private Patients in series 293

% Private Patients with some degree of inertia 17.1%

(b) Total number of General Patients with some degree of inertia 44

Total number of General Patients in series 459

% General Patients with some degree of inertia 9.5%

This figure - 9.5% - represents the average. On investigating in more detail, the results refer to the three types of General Patient - "good" "fair" and "poor".

TABLE XX

C.

	Total	Good	Fair	Poor
General Patients	459	335	71	33
Inertias	44	25	11	8
%	9.5%	7%	15.5%	24.2%

The tendency to inertia rises as the social factor falls.

Discussion

In considering the above results certain salient points are noted.

In the first place, the general class of patients, on the average, have better uterine action as shown both by the "good uterine action" and the "inertia" results. 18.5% of private patients had good uterine action (i.e. better than the average) as compared with 24.8% of general patients; and 17.1% of private patients developed some degree of inertia, as compared with 9.5% of general patients i.e. some degree of inertia is almost twice as common amongst better class women as in women of the ordinary working class.

Secondly, on further analysing the inertia incidence in

the general patients' section, it is shown that as the social factor deteriorates, the tendency to uterine inertia rises. Only 7% of the average working class women developed some degree of inertia whilst 24.2% of the poorest, destitute, class developed some degree of inertia.

Lastly, the best type of patient as regards uterine action is the working class woman of average means who, although her financial circumstances do not allow any luxuries (e.g. private maternity home fees), nevertheless has sufficient to sustain herself and her family in moderate comfort. This type of woman is intermediate in the social scale between her middle class and her lower class, it may be destitute, sister.

The reasons why this should be are probably general. Nutrition undoubtedly is important; the mother who is under-nourished has insufficient stores of energy to sustain her during a normal labour; the muscle cell becomes easily exhausted from lack of nourishment; muscle metabolism during labour calls for a plentiful supply of glucose, phosphagen, etc., which is drawn from the previous storage of the necessary energy providing factors in the tissues of the body. Nevertheless, the state of nutrition alone does not account for the above results, as the middle class woman on the whole is in a better state of nutrition than the average working class woman. As a class, the former have more opportunity to lay down stores of

combustible energy, since their dietary is probably more plentiful, and their expenditure of energy in the later months probably much less than the hard working "general" patient.

The state of nutrition, therefore, although very important, must take second place to the state of TONE of the mother in general. It is probable that the amount of work done by the general patient during pregnancy is greater than that done by the private patient, whose domestic conditions may be such that a great saving in housework is attained (e.g. modern labour saving devices), or who may be able to afford some form of daily help so as to give her more leisure and confine her activities to light duties.

In comparison, the mother of the poor and destitute class usually has more than her share of arduous household duties; her surroundings are uncongenial, the work involved in ordinary domestic duties is greater as she is hampered by over-crowding, poor housing conditions, and possibly by having to go out to work herself for part of the day. Her TONUS becomes fatigued from overwork, the muscular system is already exhausted before labour begins, and the way is paved for uterine inertia.

Here, then, are roughly the three classes of women met with in practice: 1. The middle class woman in a good state of nutrition, with every opportunity to take exercise and keep herself physically fit, and certainly with more time and

facilities to rest during the day. 2. The average working class woman also well-nourished, with fewer opportunities to take exercise and to rest. 3. The poor, destitute, class of woman in a very poor state of nutrition, with no opportunities to take light exercise and rest to her system. These three factors, viz. state of nutrition, intelligent exercise of the muscular system, and a sufficiency of rest to allow of storage of energy and to prevent over-fatigue, are probably the most important factors in the prophylaxis of uterine fatigue, and the blend of all three is best seen in the average working class mother. In her, exercise and rest are properly apportioned, whilst in the middle class woman it is possible that rest predominates over exercise and predisposes to a certain degree of "flabbiness" of muscular tone.

(e) Nutrition

TABLE XXI

Total number of cases of malnutrition . . 89
i.e. 11.8% of the cases

i. Primiparae 26

Uterine Action
i. Good . . 11
ii. Average. 11
iii. Inertia. 4

ii. Multiparae 63

Uterine Action
i. Good . . 15
ii. Average. 30
iii. Inertia. 18

	Malnutrition	Uterine Action		
		Good	Average	Inertia
i. Primiparae	26	11 (42.3%)	11 (42.3%)	4 (15.3%)
ii. Multiparae	63	15 (23.8%)	30 (47.6%)	18 (28.5%)
iii. Total i & ii	89	26 (32.3%)	41 (44.2%)	22 (21.9%)

Discussion of Results

Malnutrition

Criteria

In determining whether or not a case was suffering from malnutrition a purely clinical estimate only was possible. The appearance of the patient was the best guide: some were obviously underweight and in a semi-starved condition; others were merely suffering from a secondary anaemia of nutritional origin, and presented the picture of a pale, flabby, unhealthy looking subject, although no organic disease was present; still others were elderly multiparae of lack-lustre appearance suffering from general debility and exhaustion due to child bearing.

Of the 89 cases suffering from malnutrition, 26 were primiparae and 63 multiparae. Of the primiparae 11 had uterine action better than the average, 11 were average, and only 4 had some degree of inertia, i.e. 15.3% come into the

poor uterine action class, while 84.7% were average or better than the average. Of the multiparae, 15 were better than the average, 30 were average, whilst 18 were of poor uterine action, i.e. 28.5% come into the poor uterine action class, the remaining 71.5% being average or better than the average. If primiparae and multiparae are taken together only 21.9% were poor in uterine action, the remaining 78.1% being average and better than the average, as regards uterine action.

It would appear, therefore, that a minority of the cases suffering from malnutrition developed some degree of inertia - roughly 1 in 5, the majority being quite satisfactory. Of those developing poor uterine action, multiparae are in the majority - 28.5%, as compared with 15.3% primiparae, hence multiparae suffering from malnutrition are more liable to develop inertia than primiparae. When it is understood that these figures represent the uncorrected totals, the incidence of inertia is still lower than the percentages shown, as no account has been taken of other factors simultaneously at work - e.g. contracted pelvis with malnutrition, persistent occipito-posterior position with malnutrition and so on.

The increased liability of multiparae to develop inertia when suffering from malnutrition is easily understood, when one considers that in addition to depleted stores of glycogen etc. the uterine muscle is also possibly the seat of subinvolution,

the fibrous and elastic tissue element displacing, and reducing the efficiency of the uterine muscle. In addition muscle tone is firmer in primiparae than in multiparae.

In conclusion therefore:

1. Malnutrition alone is not an important determining factor in the causation of uterine inertia; its influence is very slight, other things being equal.
2. Malnutrition shows a greater tendency to contribute towards the onset of inertia in multiparae than in primiparae.

(f) Physique as an Index to Uterine Action.

An attempt has been made to assess the patient's physique in order to ascertain what bearing any abnormality in physique has on the action of the uterus. The abnormalities may be in physique generally, as in under-development of the muscular (skeletal) system; or locally, as in cases of over-distension of the abdominal wall, laxity etc.

Four main divisions have been made in arranging the results, viz:

1. Under-development.
2. Over-distension.
3. Hypotonia.
4. Obesity.

1. "Under-development". Briefly under this heading are included those with poor muscular development, those of thin and

spare build, generally small in stature, and possibly stunted and dwarfed. Their physical strength, measured in terms of skeletal strength, is slight.

2. "Over-distension". All conditions giving rise to increased stretching of the uterine and abdominal muscles are considered in this group, e.g. hydramnios, plural pregnancy, large child, contracted pelvis with "pendulous belly", etc. Laxity of the abdominal wall, e.g. in elderly multiparae, is also included.

3. "Hypotonia". This term has been "coined" to meet those cases which clinically might be termed "flabby", "in poor physical condition". The skeletal system of muscles is well developed but owing to lack of exercise, laziness, exhaustion with a rapid series of pregnancies, or other causes, the "tone" of the muscular system is very low. Flabbiness due to obesity is excluded.

4. "Obesity". Again a purely clinical estimate; no attempt has been made to arrive at a scientific estimate, e.g. in terms of stones avoirdupois, which is obviously impossible as the stature has to be taken into consideration. Obviously the small fat woman may weigh even less than the medium sized woman of muscular build; hence the question of obesity has been settled by purely clinical observation. The so-called "inertia type" is included in this group.

Results

TABLE XXII

Physique

Abnormality in Physique	Uterine Action			
	Good (%)	Average (%)	Inertia (%) Mild	severe
Under-development etc.	9 (22.5%)	15 (37.5%)	4 (10%)	12 (30%)
Over-distension etc.	1 (5.2%)	8 (42.1%)	4 (21%)	6 (31.6%)
Hypotonia etc.	3 (11.5%)	14 (53.8%)	7 (26.9%)	2 (7.7%)
Obesity etc.	0 0	4 (33.3%)	6 (50%)	2 (16.6%)

Discussion of Results

1. Under-development of the Muscular System.

This section accounts for the greatest number of cases, viz. 40, of which 16 developed inertia. 4 of these inertias were mild, 12 were severe, i.e. 40% of the cases of under-development of physique showed some degree of inertia. It is interesting to note how these inertia cases are distributed. In 5 of the cases inertia was present in the first stage of labour, and in only one of the five was the inertia serious; these are accounted primary inertias. Of the remaining 11 cases, 10 were due to inertia arising in the second stage of labour:

Forceps deliveries 8
Failed forceps delivered as breech . . 1
Face presentation 1

The 11th case was inertia in the second stage following inertia in the first stage and was delivered by forceps.

Accordingly, of the 16 inertia cases, 6 were primary in nature, 10 were secondary. The majority of the inertias arose in the second stage of labour, i.e. in the expulsive stage. This inertia can only be due to -

1. Weakening of contractions following normal first stage.

or 2. Weakness of the auxiliary forces.

or 3. Increased resistance to descent of the foetus through the birth canal.

The first of these possibilities is, in the first place at least, unlikely. Uterine contractions which effected a normal dilatation of the cervix ought to be strong enough to give a normal second stage, other things being equal - there is no known factor at work in a normal first stage to predispose to uterine exhaustion. The second and third possibilities are probably more important, and account for the majority of the delays in the second stage.

In these women with small stature, under-development of the muscular system, etc., the bearing down forces are weak because of the poor abdominal muscles and there may be in

addition a degree of general pelvic contraction which offers increased resistance to the descent of the foetus.

9 cases exhibited uterine action above the average, 15 were average - in all 24 out of the 40 were satisfactory. 4 of the 5 inertias in the first stage were mild in degree, and labour ended normally - these were little more than simple delays due to rather weak, ineffective, contractions. Hence only 12 out of the 40 cases of "under-development" were abnormal (30%).

In conclusion, therefore, the state of development of the skeletal muscular system is no index of the action of the uterine musculature; it is only of value as a guide to the efficiency of the extraordinary muscular forces. A woman may have a very poorly developed skeletal muscular system and nevertheless have a well-developed uterine muscle unit: the two systems are entirely disassociated (vide infra, where TONUS is discussed).

2. Over-distension of the Uterus.

Over-distension of the uterus occurred in 19 cases. 10 out of the 19 developed some degree of impaired uterine action, only one comes into the "good" class, while the remaining 8 are "average". The "good" uterine action resulted in a twin pregnancy characterized by premature rupture of the

membranes two days before labour began: the long latent period between rupture of the membranes and the onset of labour contractions is indicative of lack of tone in the uterine muscle, although labour, once begun, progressed quickly.

The cause of the over-distension is as follows:

Large child	4
Plural pregnancy	9
Hydramnios	4
Small stature with normal sized child .	2
Total	19

The tension exerted by the growing ovum on the wall of the uterus is a stimulus during pregnancy to hypertrophy of the uterine muscle. Hyperplasia of the muscle cells occurs in the earlier part of pregnancy in addition to hypertrophy, but in the latter half of pregnancy enlargement of the uterus is due solely to hypertrophy. Blair-Bell[63] considered that a certain degree of tension was desirable and helped to promote hypertrophy. Certain it is that tension, or a slight degree of stretching, is necessary to maintain muscle "tonus", for if a muscle (unstriped or striped) is allowed to relax the tone is diminished and the output of work lessened. Similarly, if a muscle is over-extended, i.e. its initial length is increased, the output of work done by that muscle is very much less than when the relation of tension to length were at the optimum.

Tonus, therefore, is at an optimum value when, in the submaximal contraction which is its characteristic, there is an alteration in length without a corresponding change in tension. Unstriped muscle is therefore said to have more tone when it is shorter than when it is longer. Tonus is entirely dependent on the stimulus elicited by slight stretching of muscle fibre. Over-stretching of muscle fibre leads to (1) increase in length and (2) decrease in tension with consequent abolition, or lessening, of tonus according to the degree of stretching. As a result the over-stretched muscle unit has a smaller output of work and is more easily fatigued.

These conditions, when applied to uterine muscle which has been gradually and progressively over-stretched in the later months, explain the weak uterine contractions which are found to be characteristic of over-distension of the uterine muscle. The continual state of distension of the uterine muscle gradually overcomes the internal viscous resistance of the muscle cell with consequent loss of tension and increase in length. Tone is as a result diminished; the excitability of the muscle to contract is still present but the latent period between stimulus and response is increased, leading to a more sluggish response. The increase in length of the muscle also diminishes the output of work done. Hence the dynamic output of the uterus is lessened.

The variability in response to over-distension in the 19 cases in this series can be explained when it is remembered that tonus is not a constant value but varies from muscle to muscle, and from individual to individual. One of the most striking differences between skeletal and visceral muscle is the difficulty in assigning a particular value to the resting length of the latter - it adopts various lengths at different times and it is impossible to name the external influences which have brought about the change in length. These changes in length (without corresponding changes in tension) are of course merely manifestations of the phenomenon of TONUS, which varies from one individual to another.

The fact that the over-stretched uterine muscle may quickly regain its tonus merely indicates that the internal viscous resistance to elongation has not been fully overcome and the relation of tension to length is more quickly adjusted. This again will vary from individual to individual. As a rule it is noticed that in over-distension of the uterus with premature rupture of the membranes the latent period elapsing between rupture of the membranes and the onset of good labour contractions is prolonged according to the amount of over-stretching and atony present.

To summarize the foregoing:

(1) 19 cases of over-distension of the uterus have been discussed, of which 10 (52.6%) developed some degree of impaired uterine action.

(2) Good uterine action is exceptional following over-distension of the uterus.

(3) The loss of dynamic power of the uterus in cases of over-distension of the uterus is due to loss of tone.

(4) The factors bringing about this state of affairs - alteration in length and alteration in tension etc. - have been discussed in detail.

3. Hypotonia.

The word "hypotonia" has been coined to explain such conditions as flabbiness, loss of tone in general. "Tone" is not here used in the strict sense in which it is used in the preceding section: the term hypotonia can be best described by the phrase "lack (or loss) of physiological fitness".

26 cases of hypotonia are included, of whom 3 had uterine action better than the average; 14 had average performance; whilst 9 had impaired action, of whom 7 were of mild severity and only 2 (7.7%) of outstanding severity. Hence 65.3% were uninfluenced by the hypotonia present whilst of the remaining 34.7% only 7.7% had severe inertia. It will be noticed that the results only differ slightly from those referring to under-development in physique, with the difference that when some degree of inertia did arise it was of a more

mild and less severe nature.

The results do not show that loss of tone in the skeletal system in general implies loss of tone in the uterine muscle system; again the two appear to be disassociated, as in the section on under-development, etc.

This is in accordance with known physiological conditions. TONE in the skeletal muscle system is dependent on the integrity of the nervous connections of the muscles concerned: indeed tone in skeletal muscle is described as being tetanic in nature - it is held to be due to repetitive stimulation as in tetanus. It differs from tetanus, however, in that in tonic contraction all the fibres of the muscle are affected in turn, intermittently and asynchronously, whereas tetanic contraction affects a particular set of fibres with regular volleys of stimuli, so that they contract continuously and synchronously. Hence in tonic contraction fatigue of any particular fibre is avoided by giving it a rest while its neighbouring fibres are contracting; hence the fatigue produced by tetanus.

Tone of plain muscle is, however, independent of nervous connections, as shown by the fact that tone is not abolished by spinal anaesthesia, paraplegia, etc. It is, for example, easily elicited simply by stretching or distending the muscle. In some cases this may be due to stretching of the local nerve plexus in the wall of the viscus, the stimulus of stretching

causing reflex contraction of the muscle; but this cannot be regarded as the complete explanation since other smooth muscles show no response to stretching, and are nevertheless capable of developing tonus even in the absence of nerve cells. This muscular tonus in plain muscle depends on the existence of a constant tension which is small and, as has been pointed out before, does not vary with the length of the muscle. Associated with this small contractile force is an extremely high "viscosity" which enables the muscle to resist a stretch, or at least to yield to it so slowly that it is some time before any considerable lengthening can take place. Tone in plain muscle, therefore, is an intrinsic characteristic of that muscle, and is independent of outside influences, other things being equal. Tone in striped muscle is dependent on extrinsic connections for its maintenance and is hence more susceptible to outside influences, e.g. fear, emotion, extremes of temperature and so on.

To recapitulate:

(1) 26 cases of hypotonia are considered of which almost two-thirds had a normal labour, and almost one-third some degree of impaired uterine action, only 2 (7.7%) being of any severity.

(2) Hypotonia of the skeletal muscle system is no index to the tone of the uterine muscle system.

(3) The essential differences between tonus in striped and unstriped muscle have been pointed out. They are of entirely different character.

4. Obesity.

In this section is included the so-called "inertia type" described by Goodall.(64) Not only are these women fat but they are characterized by being "short-necked, flat-nosed, nasal-speeched, with thick unhealthy membranes everywhere." Many of these patients present male hirsutism and others secondary male characteristics, including a male pelvis. In addition this type is also described by Goodall as developing during labour (1) a distension of the colon sufficient to displace the gravid uterus to one side of the abdomen, and also (2) a distension of the bladder due to paresis of its musculature. Goodall, however, admits that "there are many which do not conform to this type".

Of the 12 cases of obesity collected, only 2 approximated in description to the inertia type, but in neither case was paresis of the colon or bladder demonstrable: nor were either of the cases classed as severe inertia, both being of the mild variety. The type more commonly seen in this series might be described as being fat, squat in build, with short thick neck, a tendency to hirsutism, and of poor muscular power being easily exhausted physically.

The remaining 10 cases correspond roughly to the description above, the obesity being the most marked feature present. It is remarkable that of the 12 cases not one had good uterine action (i.e., uterine action better than the

average). Four had average uterine action, whilst 8 (66.6%) had some degree of inertia of which 2 were severe, and the remaining 6 mild, in nature. Of the inertias 6 were secondary in type - fatigue setting in during the second or third stage after a normal first stage, and 2 only were primary. It would appear therefore, from the above figures, that the majority of inertias are secondary in nature and are due to early exhaustion or fatigue of the uterine muscle and abdominal muscles.

When one considers the fatty degenerative changes found in the skeletal muscles and internal organs in fat subjects the above results are easily explained. The deposition of fatty globules within the muscle substance, as in fatty degeneration, is rarely met with unless in phosphorous poisoning, chloroform poisoning, etc.; but fatty infiltration is more commonly met especially in obese subjects - here the deposition of fat occurs in connective tissue between the muscle lamellae. The result is a decrease in the efficiency of the muscle and a diminution of the reserve power: this decreased efficiency and reserve power will only be manifest when the muscle unit is working under extraordinary conditions.

The role of the pituitary in the causation of obesity is important, especially in relation to obesity in pregnancy. It is known that when the posterior lobe of the pituitary is damaged, e.g. by a tumour, the increase in obesity is one of

the main clinical manifestations, e.g. in Fröhlich's syndrome; and when the posterior lobe of the pituitary is removed in dogs there is an increase in the weight of the animal (Cushing). It is not impossible, therefore, that those cases of uterine inertia which occur in obese subjects may be due to hypo-pituitarism. The oxytocic factor (pituitrin) is present in insufficient concentration in the blood stream, with the result that the uterine muscle becomes easily exhausted from lack of the oxytocic hormone in sufficient quantity.

Goodall attributes the "inertia syndrome" in his "inertia type" of woman to endocrine imbalance, the posterior lobe of the pituitary being the main controlling factor; owing to a deficiency in supply of pituitrin the autonomic nervous control of the uterus, bladder, and bowel is disorganised.

To summarize, therefore, it is shown that:

(1) Obesity predisposes to uterine inertia, the majority of the cases (66%) developing some degree of inertia.

(2) This inertia may be due to -
   (a) fatty changes in the uterine and auxiliary muscles.
   (b) Endocrine imbalance, the posterior pituitary being probably mainly at fault.

To summarize this section on Physique, etc.:

(1) The state of development of the skeletal muscular system is no index to the action of the uterus in labour. This is concluded from a survey of the findings in under-development and "hypotonia" of physique.

(2) Over-distension of the uterus predisposes to the onset of uterine inertia. Loss of tone of uterine muscle leads to lessened dynamic output of the uterus.

(3) Obesity predisposes to uterine inertia. States of obesity and uterine inertia are both probably clinical manifestations of hypo-pituitarism (posterior lobe deficiency of hormone).

(g) Relation of Psychological Factor to Uterine Action.

In this section, the psychological analysis has shown that a rough division can be made into five groups, viz:

1. Illegitimacy
2. Hysteria and Neurosis
3. Apprehension, over anxiety etc.
4. Subnormal mentality
5. Insanity

Illegitimacy. No explanation is required concerning this group.

Hysteria and Neurosis. This group embraces all those women whose behaviour has been very poor. They do not co-operate with the mid-wife or the doctor supervising their

labour: in the first stage they may scream, or make a great deal of noise, when the pains are only of mild severity: in the second stage they may refuse to bear down, or may jump about the labour bed and make the accoucheur's work very difficult. Their behaviour is very poor generally. This type of woman often cries out for an anaesthetic or other sedative when the pains first make their appearance, or may appeal to the doctor to "bring the baby with instruments" when only early on in the second stage of labour. Such is the type of woman in this group.

Apprehension, over anxiety etc. This state of mind differs markedly from the hysterical state, in that the woman's behaviour may be all that could be desired: she shows an eagerness to co-operate with her medical attendants; she is very anxious to do all in her power to expedite the labour. But she may be over-anxious, and she worries unduly as to whether things are going well or not; she may merely look apprehensive and scared of the coming ordeal, or she may voice her fears. The more serious state is that where the fear is suppressed, for she may give no outward manifestation of being apprehensive, over anxious, or very nervous.

Subnormal mentality. In this group are included those who are almost mentally defective - the so-called "border line" cases: they are stupid women of very dull intellect.

Insanity. Cases of actual insanity (ante-partum) are included naturally in this group.

Results

TABLE XXIII

Relation of Psychological Factor to Uterine Action

	Good	Average	Simple 1st	Delay 2nd	Inertia (all degrees)	Total
Illegitimacy	7 (33.3%)	11 (52.4%)	2 (9.5%)	1 (4.8%)	0	21
Hysteria and neurosis	0	7 (43.8%)	2 (12.5%)	3 (18.7%)	4 (25%)	16
Apprehension over-anxiety etc.	6 (25%)	4 (16.6%)	3 (12.5%)	1 (4.2%)	10 (42%)	24
Subnormal mentality	0	2 (50%)	1 (25%)	1 (25%)	0	4
Insanity	2 (100%)	0	0	0	0	2
	15	24	8	6	14	67

Relation of Psychological Factor to Uterine Action

Discussion of Results

Sixty-seven cases have been collected under this heading. They will be considered under their separate subdivisions.

Illegitimacy. Twenty-one cases were illegitimate in nature. It is revealing that no cases of uterine inertia developed. There were 3 cases of simple delay, 11 cases of average duration and 7 cases with a labour better than the average (33.3%). Almost 86% of the illegitimate labours were satisfactory, only 14% being slightly prolonged. It may safely be concluded from these figures, therefore, that illegitimacy is not a cause of uterine inertia.

The only tendency to delay noticed in these cases was a slight delay during the second stage of labour, some mothers refusing to co-operate in using their auxiliary forces, possibly in the hope that delay in the birth of the child might jeopardize its chances of survival. Their behaviour in the first stage of labour was on the whole satisfactory and no inhibitory effect on the uterine contractions was detected. The contractions of labour, once initiated and established, in all cases brought about a successful delivery, the mother's psychical inhibition if present not militating against the final result.

Hysteria and Neurosis. The scope and application of these terms are explained in the introductory note to this section. Sixteen such cases are considered, of whom 4 (25%) developed some degree of inertia, 5 (31%) had a simple delay, while 7 (43.8%) had an average labour. No good uterine action was found here, in marked contrast to the cases of illegitimacy. It would appear, therefore, that there is some psychical inhibition at work in cases of neurosis and hysteria.

Apprehension, over-anxiety, etc. This is by far the most important group of cases, from the point of view of psychological inhibition of uterine contractions. Twenty-four cases are reported, of whom 42% had some degree of inertia, 16% had a simple delay - a total of 58% having a delay in labour. 16.6% had an average duration, while paradoxically enough 25% had uterine action better than the average. Of the 10 cases exhibiting inertia, 7 were severe in degree - these 7 cases are considered in more detail in the section on "Uterine Inertia" (vide infra).

The discussion is more fully found in the section on psychological inhibition as a cause of uterine inertia, but it may here be stated that these results are probably due to over-action of the sympathetic nervous system with an inhibitory influence on uterine contractions. It is known that the diencephalon is the head station of the sympathetic nervous

system and the diencephalon is adjacent and in close anatomical proximity to the pituitary gland. Two factors are therefore possibly at work. (1) The emotion leads to an increased output of adrenal secretion which stimulates over-action of the sympathetic nervous system. It has been shown by various observers that adrenaline will inhibit uterine contractions (Bourne and Burn,[47] see adrenal glands section under "Endocrines", Part II). (2) The adjacent pituitary gland (posterior lobe) may be affected by sympathetic nervous stimuli reaching the diencephalon. An inhibition in the secretion of pituitrin would bring about a similar result - a delay in labour. This latter factor is still mainly theoretical and lacks experimental proof.

In conclusion it may be stated that states of over-anxiety, apprehension, etc., lead to a reflex inhibition of uterine contractions, and predispose to the onset of uterine inertia.

The 6 cases of good uterine action in this section are difficult to explain: but the majority of these cases occur in mothers who have been told to come to hospital at the ante-natal clinic: they are admitted in due course a few hours later, but the majority had commenced to have labour contractions on admission. The nervous upset at being told, or sensing, that all is not well with the pregnancy seems to predispose, in some cases, to premature labour. It may be that, as Blair-Bell found[65] small doses of adrenaline stimulate, larger

doses inhibit, uterine contractions. Recently Miller et alii have shown[49] that small doses of adrenaline stimulate uterine muscle to contract, whilst larger, clinical, doses inhibit its action.

The paradoxical results may be explained on this basis. On being told to come into hospital before term, e.g. for induction of labour, the nervous system of the mother receives a temporary shock with liberation of adrenaline - a minimal amount. After the initial shock, or upset, the mother resumes her normal rational attitude and no more adrenaline is liberated into the blood stream, but the minimal single increase of adrenaline may have already started off labour contractions, or predisposed to their onset. The apprehensive mother, on the other hand, has fear, etc., continuing during labour and is repeatedly liberating larger quantities of adrenaline into the circulation: the result is then seen as an inhibitory influence as opposed to the stimulating influence of the single minimal amount of adrenaline liberated. In the one case the action of adrenaline is very temporary and not repeated, in the other case the action of adrenaline is prolonged and repetitive, continuing to act during labour.

Subnormal Mentality. Four cases of subnormal mentality are included, of which 2 had a labour of average duration, whilst 2 had a simple delay. The number of cases is too small to

allow any conclusions to be drawn, but one would expect that the subnormal mentality, as opposed to the acutely sensitive sensorium, would tend to have a more uneventful, straightforward labour, with psychological inhibition at a minimum. The number of cases, however, precludes any conclusions to be drawn.

Insanity. Two cases of ante-partum insanity occurred during the investigation and in both cases labour was quicker than normal. Both cases were under the influence of sedative drugs during labour and chloroform à la reine was necessary in both cases to control the patient during the birth of the head. It may be that the cerebral hyper-excitability was associated with a neuro-muscular hyper-excitability with an increase of tone, but it is impossible to discuss results and draw conclusions on such a small number of cases.

To summarize the relation of the psychological factor to uterine action in labour, the salient points are:

1. Illegitimacy would not appear to be a predisposing cause of uterine inertia.
2. States of apprehension and over-anxiety definitely predispose to the onset of delay in labour. Neurotic and hysterical states cause a lesser degree of delay, but none the less definite.

(h) TABLE XXIV

Relation of Rupture of Membranes to Uterine Action

Rupture of Membranes	UTERINE ACTION					
	Good	Average	Simple Delay	Inertia Mild	Inertia Genuine	Total
I. NORMAL.						
a) Before onset of labour pains.	39(46.4%)	27(32.1%)	7(8.3%)	6(7.1%)	5 (6%)	84
b) With onset of labour pains.	12(31.6%)	18(47.3%)	5(13.1%)	3 (8%)	0	38
c) After onset of labour pains.	7(29.1%)	12 (50%)	2(8.3%)	1(4.1%)	2(8.3%)	24
II. ABNORMAL.						
a) Before onset of labour pains.	0	0	0	0	7(100%)	7
b) After onset of labour pains.	0	2 (50%)	0	1 (25%)	1 (25%)	4
	58	59	14	11	15	157

TABLE XXV

Relation of Latent Period in Premature Rupture of Membranes to Uterine Action

I. Excluding Premature Rupture in Surgical Induction, Ante-Partum Haemorrhage, Malpresentation, etc. etc.

Latent Period	Good	Average	Simple delay	Inertia Mild	Inertia Severe	Total
Under 6 hours	8	8	3	1	1	21
6 to 12 hours	7	2	0	1	1	11
12 to 24 hours	2	1	0	-	-	3
24 to 36 hours	1	0	1	-	-	2
Above 36 hours	3	1	0	-	-	4
	21	12	4	2	2	41

TABLE XXVI

II. Latent Period in Surgical Induction only (Excluding 13 medicinal + surgical induction)

Latent Period	Good	Average	Simple delay	Inertia Mild	Inertia Severe	Total
Under 6 hours	5	5	1	2	-	13
6 to 12 hours	2	1	0	-	-	3
12 to 24 hours	2	4	1	1	-	8
24 to 36 hours	0	0	0	-	-	0
Above 36 hours	3	3	0	-	-	6
	12	13	2	3	0	30

(h) Relation of Rupture of Membranes to Uterine Action. Discussion of Results.

157 cases of rupture of the membranes before, or early in, labour occurred in the series of 752 cases, i.e. 1 in 5 had either a premature or early rupture of the membranes (includes surgical induction by rupture of the membranes). The results are subdivided into I Normal, and II Abnormal labour, according to whether or not the labour was complicated. The normal cases are considered under the headings a) before, b) with, or c) after, the onset of labour pains; whilst the abnormal cases are considered only under the headings a) before or b) after the onset of labour pains, as no cases of rupture of the membranes coincident with the onset of labour pains was found.

Some explanation of the above terms is necessary.

"Before" the onset of labour pains is self explanatory - the patient was not in labour clinically when the membranes were ruptured, spontaneously or artificially. 43 of the 84 cases in the Normal column were cases of surgical induction (30 surgical, 13 medicinal followed by surgical induction), and these are included in rupture "before" onset of labour pains. Uterine contractions naturally must have been present in the spontaneous rupture cases to bring about rupture of the membranes, but the contractions had not established themselves clinically as

labour contractions - they were still pregnancy contractions.

"With" the onset of labour pains means that immediately the membranes ruptured, labour pains ensued, or vice versa. This does not mean that the patient was not already in labour - say, two fingers dilated: she may not have experienced labour pains previous to the rupture of the membranes, i.e. labour (dilatation) may have been, up to that point, painless from the subjective point of view.

"After" the onset of labour pains - the patient had been in labour some time before the membranes ruptured, but the stage of dilatation was still far from complete. This does not include cases where the membranes ruptured after the os was more than half dilated (as nearly as is possible to judge from the progress of the labour without unwarranted vaginal examination).

I. Normal.

(a) Before onset of labour pains. A latent period here exists between rupture of the membranes and the commencement of true labour. Eighty-four such cases occurred, of which 43 had surgical induction performed by rupture of the membranes. 78.5% of the cases had a subsequent satisfactory labour, of whom 46.4% had a labour better than the average. Only 13.1% developed any degree of inertia, and only 6% of these inertias were severe in degree.

(b) "With" onset of pains the same results occurred in this column - again 78.9% were quite satisfactory, 31.6% being above the average. 8% developed a mild inertia; there were no severe inertias.

(c) "After" onset of pains. Again the constant figure - 79.1% of cases were satisfactory, 29.1% being above the average. 12.4% subsequently developed some degree of inertia.

The striking fact about the above results is the remarkable constancy of the "satisfactory" results - roughly 80% were all that could be desired, while an average of 10% developed some degree of inertia of which the majority were mild in degree. It would appear, therefore, that far from being a cause of uterine inertia, or disordered uterine action, premature rupture of the membranes in a normal presentation (N.B.) is an index that all will be well in 80% of the cases, and 40% of these will have a labour speedier than the average. In no other series of control cases in this investigation was there such a high figure - 40% - signifying uterine action above the average.

It would seem reasonable to conclude, therefore, that the dilating action of the hydrostatic bag of waters has been exaggerated. The cervical reflex, from more direct pressure on the cervical ganglion by the hard foetal head, seems to be reinforced and gives rise to stronger contractions and speedier

dilatation; nor was there any increased mortality or injury to the foetal head from more direct pressure. In a recent series Drew-Smythe[66] published a series of results where the membranes had been ruptured surgically by high puncture (the method adopted in surgical inductions here), and showed that labour was shorter in duration and delivery unassisted in 91.4% of cases. If the simple delay figures are included an average figure above 90% is obtained here (almost 95%), as some of the mild inertias were normally delivered without obstetric interference.

II. Abnormal Presentation.

Here the figures are illuminating. Seven cases had premature rupture of the membranes before the onset of labour, and all seven subsequently developed a severe degree of uterine inertia. Four cases were characterized by rupture of the membranes after the onset of labour, and of these two developed inertia of which one was severe in degree, and two were of average duration. Hence of the 11 cases in this section 9 developed some degree of inertia, only one being mild in degree. The figures merely serve to emphasise that premature rupture of the membranes in the presence of a malpresentation is an indication of future trouble.

The result of a malpresentation is such that there is uneven pressure exerted on the cervix and cervical ganglia

with loss of normal rhythmic reflex contractions; the pains are irregular in rhythm, deficient in dynamic power, and shorter in duration. While uterine muscle has apparently some degree of intrinsic power of contraction (as shown by experiments on strips of uterine muscle from which all nervous tissue has been removed) it would appear that the reflex nervous connections exert a regulatory influence on uterine action, and weak and infrequent afferent stimuli result in irregular, often spasmodic and more painful, contractions. An even distribution of pressure possibly stimulates equally the ring of paracervical ganglia, with the result that the afferent stimuli reaching the spinal centres arrive simultaneously and with equal force; the resultant efferent response is equal and simultaneous. On the other hand, an uneven distribution of pressure can conceivably stimulate the ring of paracervical ganglia unequally, the afferent stimuli being unequal in force and irregular in frequency; and the efferent response is similarly affected. The result in the latter case is a varying degree of primary inertia, the stage of dilatation being greatly prolonged.

The Relation of the Latent Period in Premature Rupture to Uterine Action.

The relation of the latent period following premature rupture of the membranes to uterine action has also been

studied (see Tables 25 & 26). The results are divided into two sections. In the first section spontaneous premature rupture of the membranes is considered, i.e. premature rupture in surgical induction of labour, ante-partum haemorrhage, malpresentation and other abnormalities is excluded. 41 such cases are tabulated. In the second section the latent period in artificial rupture of the membranes, i.e. in surgical induction of labour, is considered. 30 such cases are shown. The remaining 13 cases of premature rupture of the membranes, making a total of 84 with rupture of the membranes before the onset of labour, occur in cases where medicinal induction was unsuccessful and was followed by surgical rupture of the membranes - these cases are tabulated in Table 27 on the Induction of Labour section.

On comparing the two tables on the bearing of the Latent Period on subsequent uterine action, it is seen that the findings are mainly on parallel lines. As before, less than 10% had a labour more prolonged than normal, the majority - about 80% - were uncomplicated and eminently satisfactory, while the remainder - less than 10% - showed simple delay but no inertia. The majority - 21 out of 41, and 13 out of 30 - had a latent period of less than 6 hours, indicating a quick response of the uterine muscle to the stimulation evoked by removal of some liquor amnii: but it is amongst this majority

that the few cases showing some degree of inertia are found. At the other end of the scale it will be seen that those cases, where more than 36 hours elapsed following rupture of the membranes, had a high percentage of average and good uterine action - results which are difficult to explain clinically. It was found that those cases with effacement of the cervix before the onset of labour were followed by shorter latent periods than those with a fairly long conical cervix: the former were obviously in a more advanced stage of early differentiation into upper and lower uterine segments than the latter. The various conditions for which surgical induction was performed are not considered: but it is only possible to conclude from the above results that in premature rupture of the membranes the latent period is no reliable index of subsequent uterine action, labour not necessarily being shorter when the patent period is small, nor longer where the latent period is large. Lastly, the latent period does not differ materially in spontaneous as compared with artificial premature rupture of membranes.

(i) TABLE XXVII

Induction of Labour

Induction	UTERINE ACTION					
	Good	Average	Simple delay	Inertia Mild	Inertia Severe	Total
1. Medicinal	12	13	2	3	3	33
2. Surgical	11	14	2	3	-	30
3. Medicinal and Surgical	5	3	1	1	3	13
4. Incomplete Med.	7	5	1	-	-	13
	35	35	6	7	6	89

The Influence of Induction of Labour on Uterine Action.

The Inductions have been classified as (1) medicinal, (2) surgical, (3) medicinal and surgical, and (4) incomplete medicinal.

(1) Medicinal Induction, or induction of labour by the use of drugs, accounts for 33 out of a total of 89 inductions. The method of induction is as follows:

6 p.m. Castor Oil ii ounces.
7 p.m. Hot bath
8 p.m. Soap and water enema.
9 p.m. Quinine sulphate (in acid mixture) gr.x.
12 midnight do.
3 a.m. do.
6 a.m. do.
9 a.m. Pituitrin 0.5 c.c.s ½-hourly till a total of 3 c.c.s has been given, or until labour contractions begin.

Occasionally thymophysin has been substituted for pituitrin where there has been at any time during pregnancy an elevation of blood-pressure, or where there is any cardiac lesion present. Pituitrin (and thymophysin) is never given in the presence of any disproportion. Toxaemic cases are not induced by drugs - rupture of membranes is usually more satisfactory, apart from theoretical considerations.

The results are tabulated in Table 27. Including cases of simple delay 8 out of 33 had uterine action below the average, of which 3 were examples of severe inertia.

(2) Surgical Induction. Induction here is confined to high puncture of the membranes according to the method recently described by Drew-Smythe of Bristol[66] (a No.10 silver metal male catheter is quite satisfactory and has been used with considerable success). Rupture of the membranes is never performed in the presence of any disproportion. 5 out of 30 such cases had a labour below the average. No severe inertias occurred, 3 being mild only.

(3) Medicinal and Surgical. In these cases, two medicinal inductions, with an interval of 30 hours between each, have been unsuccessful. The membranes are then punctured after the second unsuccessful medicinal induction. In certain borderline cases of disporportion the head may fix after the exhibition of the drug, but labour does not begin. In these

cases the membranes may be ruptured - these cases are very few, however, and the head is considered to be well fixed, and flexed, before such procedure is resorted to.

Thirteen cases of medicinal, followed by surgical, induction are shown, of which 5 developed an inferior type of uterine action, and 3 being severe degrees of inertia.

(4) Incomplete Medicinal. This differs only from complete medicinal induction in that no pituitrin (or thymophysin is given. Only 1 out of 13 developed a simple delay during labour: no inertias occurred. This method is practised where some disproportion exists and where pituitrin is contra-indicated: e.g. in a mild toxaemia. Even in mild toxaemia the opinion has been formed that the amount of quinine given - 40 grains of quinine sulphate - has been deleterious to the foetus, which is already probably toxic and enfeebled: foetal death has been noted to follow in certain of these cases. Quinine is never given in a moderate or a severe toxaemia, but its use in mild toxaemia has had the above untoward result.

A total of 19 cases with inferior uterine action was found to occur in the 89 cases induced. This total is composed of

Simple delay	6
Mild Inertia	7
Severe Inertia	6

If group 4 is excepted - in which there was 1 case of simple delay - a total of 18 remains. Of these 18, only 5 were found to occur in surgical induction, leaving a total of 13. These 13 cases occurred where medicinal induction was practiced, in whole or in part; moreover the 6 cases of severe inertia all occurred in groups 1 and 3. Thus in groups 1 and 3 are found:

Simple delay	3
Mild Inertia	4
Severe Inertia	6

This leads one to agree with the view expressed by Bourne and Bell[67] - that induction by drugs, including the use of pituitrin, is often followed by, and is a common cause of, uterine inertia. It is remarkable that out of 13 incomplete medicinal inductions no inertia, and only 1 case of simple delay, was found, whereas in a similar series of 13 medicinal followed by surgical induction (which means that any disproportion was negligible) 3 severe and 1 mild inertia and 1 simple delay occurred. The exhibition of pituitrin has apparently caused the increased incidence of inertia, as all the other factors are the same; or else, surgical induction by rupture of the membranes following upon an unsuccessful drug induction has been followed by inertia.

Some years ago Blair-Bell pointed out[68] that pituitrin exhibition was followed by a refractory period during which the

uterine muscle would not fully respond to a second dose of pituitrin, and the period of 2 hours was given as the appropriate interval between injections of pituitrin. It may be, therefore, that in some cases the repeated injection of pituitrin predisposes to a state of fatigue of the uterine muscle by over-stimulating the muscle. The question of previous sensitization of the uterine muscle by oestrin necessarily enters into the discussion, and it is not illogical to advance the explanation that those cases which have not responded to pituitrin induction, or have developed inertia following its use, have not been sufficiently sensitized by oestrin to the action of pituitrin. That being so, the rupture of the membranes in these cases, while certainly precitipating the onset of labour, is akin to asking an insufficiently prepared, or over-stimulated and refractory uterine muscle to work under unfavourable conditions: in these cases, the result is an inertia of varying degree, according to the condition of the musculature.

In considering the purely medicinal inductions it is seen that 25 out of 33 (76%) were quite satisfactory as regards uterine action, but the remaining 24% were unsatisfactory. The feeling still remains, therefore, that induction of labour by means of drugs, including pituitrin, may predispose to the onset of uterine inertia by over-stimulating and exhausting a

uterine muscle which may, or may not, be sufficiently sensitized and prepared for the changes which bring about the onset of labour. That is to say, by using drugs of hormonic nature, without knowing the state of hormone balance in the circulation, one is dealing with an unknown (clinically) quantity, and an inertia may follow from hormonic imbalance. It is well known that induction by drugs is more likely to be successful the nearer to term they are used - the uterine muscle is then a favourably receptive and sensitized state. On account of these factors, therefore, surgical induction is to be preferred as, according to the results tabulated above, there is less liability to the onset of inertia, the hormonic balance is not disturbed or interfered with, and labour is almost certain to commence after a varying latent period.

Case No.	Age/ Parity	Aetiology	Duration of Labour	Termination	Sequelae		Primary or Secondary	Rupture of Membranes	Associated General Factors
					Child	Mother			
3	30/1	R.O.P. Oedema lip of Cx.	1. C. 4 days 2. 4 hrs 3. 20 m.	Difficult Forceps Man.Rotation	S.B. 8 lb.6 oz.	-	Primary and secondary	Premature	Economically poor.
39	32/2	Brow	1. 37 h. 30 m. 2. 3 h. 50 m. 3. - 20 m.	Perforation Difficult Forceps	S.B. 8 lb. 1 oz.	-	Primary and secondary	Early in labour	Hypotonia: obesity slight
107	25/1	Persistent R.O.P.	1. 40 h. 2. 8 h. 3. - 20 m.	Forceps Face to pubes	Alive 7 lbs	-	Primary and secondary	Premature	-
106	36/4	Contracted pelvis "Failed forceps"	1. 24 h. 2. 4 h. 3. - 20 m.	High forceps	S.B. 8 lbs	-	Primary and secondary	Premature	Malnutrition
131	40/9	Hydramnios with overdistension	1. 69 h. 20 m. 2. - 20 m. 3. - 20 m.	Normal	S.B. Prem.7/12 3 lb.2 ozs	-	Primary	Surg.Induction	Debilitated
145	39/1	Elderly primipara Oedema lip of Cx.	1. C. 6 days 2. 1 h. 40 m. 3. - 40 m.	Forceps	S.B. 7 lb.8 ozs	-	Primary	-	-
232	25/1	Hydramnios	1. 42 h. 45 m. 2. - 30 m. 3. - 20 m.	Normal	S.B. Prem.7/12 3 lb.6 ozs	-	Primary	Surg.Induction	-
248	32/1	R.O.P.	1. 60 h. 2. 4 h. 3. - 20 m.	Forceps Man.Rotation	S.B. 9 lbs	-	Primary and secondary	Premature	-
266	31/1	G.C. pelvis	1. 36 h. 15 m. 2. 3 h. 10 m. 3. - 20 m.	Low forceps	Alive 6 lb.4 oz.	Died p.sepsis	Primary	Early in labour	Poor physique

Case No.	Age/ Parity	Aetiology	Duration of Labour	Termination	Sequelae		Primary or Secondary	Rupture of Membranes	Associated General Factors
					Child	Mother			
292	28/1	Psychical inhibition	1. 93 h. 25 m. 2. 1 h. 5 m. 3. - 20 m.	Normal	Alive 7 lb.8 ozs	-	Primary	-	-
297	32/1	Age ?	1. 49 h. 30 m. 2. 2 h. 50 m. 3. - 20 m.	Normal	Alive 7 lb.5 ozs	-	Primary	-	-
323	30/1	Over-distension Large child Slight disproportion	1. 57 h. 2. 1 h. 30 m. 3. - 20 m.	Forceps	S.B. 8 lb.13 oz.	Morbid perineal tear ++	Primary	-	Small in stature
330	19/1	General contraction of pelvis.	1. 25 h. 2. 6 h. 55 m. 3. - 30 m.	Forceps	Alive 5 lb.11 oz.	-	Secondary	-	Malnutrition
370	33/1	Retained placenta	1. 17 h. 2. 1 h. 15 m. 3. 10 h. 5 m.	Normal delivery Manual removal of placenta	Alive 6 lb.4 oz.	Died of puerperal sepsis	Secondary	-	-
394	22/1	R.O.P. flexing to a vertex - R.O.A.	1. 62 h. 10 m. 2. 3 h. 50 m. 3. - 15 m.	Normal	Alive 7 lb.13oz.	-	Primary	Premature	-
416	31/1	General contraction of pelvis.	1. 45 h. 30 m. 2. 4 h. 5 m. 3. - 35 m.	Forceps	Alive 6 lb.2 oz.	Second. repair of perinaeum	Primary	-	Psychologically poor.
430	26/1	Prematurity Retained placenta	1. Unknown 2. " 3. 6 h.	Undelivered of placenta	Alive 3 lb.9 oz.	Died	Secondary	?	Poor condition
441	33/1	Over-distension Large child	1. 58 h. 30 m. 2. 3 h. 40 m. 3. - 40 m.	Forceps	Alive 8 lb.8 oz.	-	Primary	-	-

Case No.	Age/ Parity	Aetiology	Duration of Labour	Termination	Sequelae		Primary or Secondary	Rupture of Membranes	Associated General Factors
					Child	Mother			
455	28/1	Psychological inhibition	1. 72 h. 2. 1 h. 35 m. 3. - 30 m.	Low forceps	Alive spina bifida 7 lb.5 oz.	Second. repair of perinaeum.	Primary	-	-
502	28/1	Psychological inhibition. General contraction of pelvis.	1. 44 h. 2. 3 h. 5 m. 3. - 20 m.	Forceps	Alive 6 lbs	-	Primary and secondary	Premature (Surg. Induction)	Surgical induction
516	29/1	Psychological inhibition	1. 62 h. 2. 2 h. 30 m. 3. - 20 m.	Normal	Alive 7 lb.2 oz.	-	Primary	-	-
517	27/1	Plural pregnancy over-distension	1. 45 h. 2. 2 h. 55 m. 3. - 15 m.	a) Manual rotation and forceps b) Normal	a)Alive 6 lb.5 oz. b)Alive 5 lb.10 oz.	- -	Primary	-	-
520	39/1	Psychological inhibition Fibroids	1. 46 h. 30 m. 2. 3 h. 15 m. 3. - 30 m.	Forceps	Alive 7 lb.4 oz.	-	Primary	-	-
554	29/5	Over-distension Twins	1. 45 h. 50 m. 2. a) 25 m. b) 35 m. 3. 20 m.	Normal	a) Alive 5 lb.14 oz. b) S.B. 4 lb.1 oz.	- -	Primary	-	Exhausted multipara Malnutrition
580	30/1	Fibroids	1. 22 h. 2. 6 h. 10 m. 3. - 20 m.	Forceps	Alive 7 lb.5 oz.	-	Secondary	-	Hysterical type
602	28/1	General C.of P. (? Induction)	1. 60 h. 2. ? 3. - 20 m.	Forceps	S.B. 7 lb.8 oz.	-	Primary	Premature Surg.Induction	Poor physique

Case No.	Age/ Parity	Aetiology	Duration of Labour	Termination	Sequelae		Primary or Secondary	Rupture of Membranes	Associated General Factors
					Child	Mother			
617	29/1	General C.of P. (Induction)	1. 37 h. 2. 6 h. 5 m. 3. - 20 m.	Forceps	Alive 5 lb.10 oz.	Second. repair	Primary	-	Small physique
637	26/1	Persistent R.O.P. (retained plac.)	1. 34 h. 2. 4 h. 40 m. 3. 2 h. 35 m.	Manual rotation and forceps. Manual removal	Alive 7 lbs	P.sepsis Died	Secondary	Premature	-
663	20/1	Poor condition Prematurity	1. 51 h. 10 m. 2. 1 h. 15 m. 3. - 15 m.	Normal	Alive 4 lb.4 oz.	-	Primary	-	-
672	31/1	Psychological inhibition	1. 53 h. 30 m. 2. 1 h. 50 m. 3. - 20 m.	Normal	Alive 7 lb.5 oz.	-	Primary	-	Inertia type
673	30/1	Psychological inhibition	1. 61 h. 30 m. 2. 4 h. 25 m. 3. - 20 m.	Normal	Alive 7 lb.4 oz.	Puerperal pyrexia	Primary	Premature	-
679	26/1	Unknown	1. 47 h. 30 m. 2. 1 h. 40 m. 3. - 20 m.	Normal	Alive 7 lb.7 oz.	-	Primary	Premature	-
750	26/2	Unknown (retained placenta)	1. 2 h. 20 m. 2. 1 h. 3. 3 h. 20 m.	Manual removal	Alive 7 lb.11 oz.	Died P.P.H.	Secondary	Premature	-

TABLE XXIX

UTERINE INERTIA

Aetiology	Termination		Child			Mother		
	Forceps	Normal	Alive	S.B.	Neo.	Normal	Morbid	Died
Malpresentations								
i. Occipito-posterior	4	1	3	2	-	4	1	1
ii. Other malpositions	2	-	-	2	-	2	-	-
General Contraction of Pelvis	5	-	4	1	-	2	3	1
Overdistension	3	5	4	4	-	5	1	-
Psychological Inhibition	3	4	7	-	-	5	2	-
Other causes								
Fibroids*	2	0	2	-	-	2	-	-
Prematurity	0	1	1	-	-	1	-	-
Elderly Prim.	1	0	0	1	-	1	-	-
Unknown	0	2	2	-	-	2	-	-
Retained placenta	Manual removal 3** - 1 undelivered		4	-	-	0	2	4

Fibroids* - Elderly primigravida in addition. Also Psychological Inhibition in which section the case is entered also.

** - One of which is already entered under occipito-posterior cases.

TABLE XXX

UTERINE INERTIA

Types of Inertia

1. Inertia arising in first stage of labour only ("Primary" inertia) . . . 21 (63.6%)

2. Inertia arising in second stage of labour only ("Secondary" inertia) . . 6 (18.2%)

3. Inertia persisting throughout labour i.e. "primary" followed by so-called "secondary" inertia 6 (18.2%)

Thus only 18.2% were due to a true "secondary" inertia, the vast majority suffering from primary inertia.

TABLE XXXI

MODE OF DELIVERY

Normal uncomplicated delivery (2nd and 3rd stages)	10 (31.2%)
Obstetrical interference (including 3 manual removals)	22 (69.8%)
	32*

* One case died undelivered of placenta.

Thus 69.8% required obstetrical interference to complete the delivery. The increased risk of sepsis following uterine inertia and interference is obvious.

TABLE XXXII

THE CHILDREN

(1)	Alive	25	(71.4%)
(2)	Stillborn	10 **	(29.6%)
		35*	

* Includes two sets of twins

** Includes three premature children.

TABLE XXXIII

THE MOTHER

Puerperium normal	24	33
Puerperium morbid	9	
Died	6	(18%)

Note the high maternal mortality - 18%. This is accounted for by 4 deaths, due to puerperal sepsis (2 following instrumental delivery, 2 following manual removal of placenta) and 2 immediate deaths due to post-partum haemorrhage and shock following retained placenta. The morbidity rate works out at 27.2%. The results of uterine inertia as regards the mother are disastrous, and the very high mortality rate here is largely due to inefficient uterine action in the third stage of labour.

TABLE XXXIV

PARITY

Number of primiparae	28	Total No. of primiparae in series	445
Number of multiparae	5	Total No. of multiparae in series	307
	33		752

Hence uterine inertia is almost 6 times more common in primiparae than in multiparae. The incidence of uterine inertia (severe) in the series is 4.4%.

(j) Severe Uterine Inertia.

The criteria adopted in deciding whether a case is one of severe uterine inertia or of mild uterine inertia are again mainly clinical. The duration of labour is often very difficult to assess, as a patient may be very vague as to when her pains began before admission to hospital. Very few of the cases were not in labour on admission and hence the commencement of labour can only be roughly accurate and one must be content with the patient's statement as to when true and regularized labour pains began: the error here may amount to quite a few hours in a primipara, who may easily mistake the prodromal stage of labour for real labour. Again it has been noted that labour "pains" are not synonymous with labour contractions: the stage of dilatation may be proceeding painlessly in a certain proportion of cases, and labour "pains" only make their appearance when near full dilatation. Here the error is unimportant because these painless (comparatively or actually) first stage cases do not usually have an inertia and hence they can be ruled out of this section.

All the cases classed as severe uterine inertia were seen, and the degree of inertia estimated according to the nature of the contractions, their frequency and periodicity, and the progress of true labour.

Roughly, in primiparae, these cases have a labour lasting about 48 hours or longer in primary inertia, or 4 hours or longer in secondary inertia. Retained placenta has been included as a secondary inertia.

The cases of severe uterine inertia have been collected, and will be discussed, under the following headings:

1. Malpresentations.

These accounted for 7 out of the 33 cases of inertia (severe). In 5 of these the malpresentation was an occipito-posterior position of the head, 4 of which became persistent and 1 of which rotated to a favourable anterior position of the vertex. This latter case was delivered normally; and of the four persistent occipito-posterior positions, one was delivered face to pubes, the other three by manual rotation of the child and the application of high forceps.

In the remaining two cases, one was a brow presentation which was terminated by perforation and forceps delivery, internal version being impossible owing to the large child and the liquor having drained away; the other was a "failed forceps" due to a contracted flat pelvis, delivery being by forceps. In both these cases the children were still-born. In all these malpositions, 4 out of the 7 children were still-born, 3 were alive, and there were no neonatal deaths. One mother had a morbid puerperium and died of puerperal sepsis:

in addition to a manual rotation and high forceps delivery of a live child, the placenta was unfortunately retained, and manual removal was necessary after two hours owing to early post-partum haemorrhage.

Hence, malpresentations account for 21.6% of the inertias. Interference was necessary in 85.7%, foetal mortality is 57.1%, the maternal mortality and morbidity being 14.2% (one in seven).

2. General Contraction of Pelvis.

There are 5 cases in this section which developed severe uterine inertia: indeed general contraction of the pelvis has accounted for more inertias than flat pelves, but this may be due to the fact that the rickety and the simple flat pelvis is, in this district at least, a comparatively "rara avis". The majority of cases of severe degree of flat pelvis have Caesarean section done at term; the moderately severe and borderline flat pelvis have a trial of labour, but as already stated, these trials of labour are very few in this series.

One case developed puerperal sepsis and died of puerperal septicaemia and peritonitis within 72 hours of delivery, the haemolytic streptococcus being cultured both from the blood and peritoneal cavity (Case No.266). One other case required a secondary repair of a perinaeal tear, the puerperium being thereby prolonged. One child was still-born; all deliveries were by forceps.

One more case might be added to this section, but it has been included in the section on psychological inhibition, but a general contraction of the pelvis was present and delivery was by forceps also, the child and mother being alive and normal. Hence all six cases were terminated by forceps deliveries.

Three of the six cases had induction performed.

1. Case No.502. Mrs.K.M. At term. Albuminuria a trace. B.P.190/60. No oedema. Surgical rupture of membranes to induce labour. Pains began soon after but did not improve and Mist. Sedativ. 1 oz. given (chloral and bromide mixture).* Pains ineffective and infrequent most of next day but improved towards evening, when 0.2 c.c.s thymophysin was given. Pains became strong and regular after the injection of thymophysin but again they deteriorated. Mist. sedativ. 1 oz. again given, but patient had a very poor night. The following afternoon mist.quinine 2 drachms (gr.v) was given for 2 doses, with an immediate improvement in the pains. Meconium was then passed, and a forceps delivery resulted in a live child being born. Third stage normal. Surgical induction was preferred in this case to drug induction by pituitrin owing to albuminuria and high B.P. The head was well fixed and almost through the pelvic brim.

---

* Mist. sedativ. contains in 1 oz: Chloral hydrate gr.30.
Pot. Bromid. gr.30
Extr. Hyoscyam. gr.¼
Extr. Cannab Indicis. gr.1/16.

2. Case No.602. Mrs. E. McG. 7 days from term. Drug induction on 16.4.37 unsuccessful; repeated 18.4.37, unsuccessful. Thymophysin 12 units given on 22.4.37 - unsuccessful. Membranes ruptured 23.4.37. Labour contractions did not begin till 26.4.37 - a very long interval - when Morph. Sulph. gr.1/6 was given. The contractions became inertia in type the following day and pituitrin 0.25 c.c.s was given for 4 doses at ½-hourly intervals (27.4.37), with no improvement, and mist.sedativa 1 oz. was given, repeated in 4 hours ½ oz. On the following morning there was no bearing down, pains were ineffectual and on examination the cervix was fully dilated. A forceps delivery resulted in a still-born child of 7 lbs.

This case was characterised by having repeated inability to pass urine, catheterisation being necessary frequently. In addition, bowel action even with enemata was very sluggish, and in the puerperium meteorism developed.

The head was through the brim at beginning of labour.

3. Case No.617. Mrs. A.M. 10 days from term. Drug induction 4.4.37 and again 6.4.37 - both unsuccessful. Thymophysin 12 units on 7.4.37, but labour did not begin till 8.4.37. Thereafter inertia type of labour resulted, the mother becoming gradually exhausted, poor bearing down etc. The head was well fixed at beginning of labour. Delivered by forceps on 10.4.37 after sedative treatment; child 5 lbs 10 ozs, alive.

Discussion

It has been maintained by certain authorities that uterine inertia is more likely to supervene after induction of labour, Bourne and Bell.[69] The above results would tend to bear this out, 50% of the inertias in general contraction of the pelvis following on medicinal (two) and surgical induction (one). One feature common to all these cases of general contraction is that the heads were well fixed before inducing; the women were of small proportions generally with poor muscular development. The end result in all cases was the same - the mother became exhausted, tired out with two or more sleepless nights, and had not the strength to bear down when the cervix was eventually fully taken up; indeed, looking at the patients one could, and did, easily mistake the stage of labour. One was surprised in most of these cases to find that dilatation was complete, the mother simply lying in bed moaning when a pain supervened and making no attempt to bear down. This was probably due to the fact that the pains were by this time comparatively feeble, and the mother's condition was that of early exhaustion.

The feeling persists that the stimulation of the uterus by oxytocic drugs immediately prior to labour in generally contracted pelvis leads to early exhaustion of the uterine forces, with

secondary maternal exhaustion subsequently. The course of labour is already more prolonged normally in these cases, and the difficulty increases with the descent of the head through the pelvis during labour, without bombarding the uterine muscle with stimuli prior to labour. Although thymophysin is claimed to conserve the forces and to minimise the onset of inertia, no effect followed its use in two of these cases; but it is only fair to state that it was used in each case after the exhibition of pituitrin. Roques and MacLeod[70] do not support the claims put forward by the adherents of Temesvary in regard to thymophysin in a recent investigation.

Overdistension of Uterus.

There are 6 cases in this category: 2 due to hydramnios, 2 to plural pregnancy, and 2 to large children (relative to the mother). Both cases of hydramnios were premature labours at 32 weeks, the degree of distension being moderate, but not gross: both these children were stillborn, deliveries being normal. Of the plural pregnancy cases, one required interference in the form of manual rotation of an occipito-posterior position and forceps delivery, the second child being born normally. The other case of twin pregnancy terminated normally, but one of the children was stillborn. It is to be noted that there are 8 deliveries in this group although there are only 6 cases under consideration, the other two deliveries

being accounted for by the plural pregnancies. The remaining two cases have large children as the cause of the over-distension, the weights being 8 lbs 13 ozs and 8 lbs 8 ozs. Both deliveries terminated by forceps, the larger child being stillborn. Over all there were 4 stillbirths, 3 forceps deliveries and 1 morbid puerperium due to secondary repair of an episiotomy which broke down in the puerperium.

In one of the cases there was present a slight degree of disproportion - with the 8 lbs 13 oz. child, so that the returns are exaggerated accordingly, this case accounting for one forceps delivery, one stillbirth, and the only morbid puerperium. The large head was well fixed at the beginning of labour but its passage through the pelvic brim was very slow, the weak pains being partly, though not wholly, responsible.

In the section on "Physique" are collected all the cases of overdistension - 19 in all. Of these only one had a good uterine action (5.2%), 8 had an average labour (42.1%), 4 had a mild inertia (21%) while 6 (as described above) developed a severe inertia.

From a consideration of the above figures some degree of inertia developed in 10 out of 19 cases of overdistension (52.6%), hence there is a much greater liability for inertia to develop in a uterus which is overdistended prior to the onset of labour. The cause of this inertia is probably to be

found in the fact that an overstretched muscle tends to have its "tonus" diminished, and lowered muscular tone predisposes to uterine inertia (see TONUS in the Discussion in section on "Physique" Part IV (f)).

Psychological Inhibition.

With the exception of the malpresentation and malposition group, this group accounts for the greatest number of inertias. 7 cases of genuine inertia had some abnormality in their psychological factor which resulted in, or contributed towards, the onset of inertia.

1. Case No.292.

Mrs. M.H. Characterized by being over-eager to have good pains and worrying unduly because she did not do so. Sedative treatment given - morphia and mist. sedativa. Pains increased and regularized only after thymophysin 0.2 c.c.s, two doses given. Thereafter quick progress and normal delivery of live child.

2. Case No.455.

Mrs. R.R. External cephalic version performed three times during ante-natal period. Mother ultimately highly strung and apprehensive lest anything should happen to child. During labour co-operated well with medical and nursing staff, but primary inertia supervened. Sedative treatment given. Delivery by forceps, spina bifida, alive. Subconscious suppressed fear present throughout, although co-operation and behaviour were otherwise ideal.

3. Case No.502.

Mrs. K.M. Case of subconscious inhibition - mother over-eager to please, and upset at slow progress of labour. Sedative treatment given followed by thymophysin 0.2 c.c.s - temporary effect only, and quinine sulph. gr.v for 2 doses. Forceps delivery owing to foetal distress and no progress being made. Alive.

4. Case No.516.

Mrs. B.T. Two patients in labour in adjacent labour beds, separated only by a screen. Both patients making much noise about pains, and behaving rather poorly: in fact they seemed to be vying with each other as to who should be first delivered. Illustrative of the adverse effect two people of similar poor temperament may have on each other. Good progress after artificial rupture of membranes when 3 fingers dilated and sedative treatment. Normal delivery. Live child.

5. Case No.520.

Mrs. M.R. Elderly primipara, very anxious and apprehensive of ordeal before her, refusing to be reassured that everything was progressing normally. In addition she had a fear of being delivered by the night staff to whom she had taken a dislike on admission. Actually she was delivered by low forceps during the night. Small uterine fibroids also present. Child alive.

In this case the fear was manifest and outspoken.

6, & 7. Cases No.672 and 673.

Again two patients of poor behaviour in labour in adjacent beds; both making more noise than necessary, and vying with each other as to progress.

Both cases treated by sedative drugs; both were delivered normally of live children, the second case progressing much more satisfactorily after the first case had been delivered and removed from the labour ward.

From a consideration of the above data it is seen that there is some complex psychological factor at work. In some it is fear, which may be manifest or suppressed - either type seems to have the same deteriorating effect on the character of the labour contractions. In others it is over-eagerness to behave well in an endeavour to do everything possible in the way of obedience to facilitate and expedite the birth of her child. In the remaining cases is seen the effect which results from having two women of the same nervous temperament in adjacent beds within hearing of each other: they almost seem to compete (probably unconsciously) with each other. The one hears the other making much noise during a pain, and not to be outdone she attempts to emulate her neighbour. The good progress which often results from separating such cases is direct evidence of the adverse effect these cases have on each other, and there is something to be said for separate labour rooms for people of such highly strung temperament.

It is noticeable that all seven children were born alive, four of them normally (it is possible that one of the three forceps deliveries was unnecessary, being performed by another doctor), and from this it would appear that the child is never

in any great danger, owing to the feebleness of the uterine contractions. The treatment in all cases was by sedative drugs in the first instance, followed in one case by rupture of the membranes, and in two others by thymophysin and quinine, with good results.

In the section on the "Relation of the Psychological Factor to Uterine Action" Part IV (g), it will be seen that 24 out of the 47 cases were due to fear, apprehension, over-anxiety etc., while of the 14 inertias in that section 10 were due to these factors - viz. fear, emotional states, apprehension, over-anxiety, etc.

Another notable factor in these seven cases of psychological inhibition is the age and parity. All the cases are primiparae, and their ages range from 28 to 39 years of age, the average age being 30.5 years. It is interesting to note that this is the age-group in which the greatest number of inertias are found (see Graph III).

Other Causes of Severe Inertia.

(a) Uterine fibroids, complicating pregnancy account for two cases of uterine inertia, both deliveries being instrumental, and both children being born alive. The puerperia were normal.

The relation of uterine fibroids to uterine action in labour has already been discussed (Part IV, Maternal Disease Section). Six cases of uterine fibroids complicating pregnancy

were collected, and two of these developed uterine inertia which was primary in one case (Case No.520) and secondary, following upon a simple delay in the first stage, in the other (Case No.580). It has been noted before that the inertia cases occurred amongst two out of three elderly primigravidae whilst the multiparae had a normal labour. No cases of retained or adherent placenta occurred in any of the 6 cases where fibroids were diagnosed.

(b) Prematurity.

When one considers the elaborate preparations which Nature makes to bring about a successful issue during parturition, it is not surprising to find that the accident of premature labour brings many complications in its train. Within a few weeks of term the uterine muscle is probably structurally fully developed, but the important changes associated with the early differentiation of the uterus into upper and lower segments are not fully developed. In addition, the biochemical changes brought about by endocrine activity are incomplete, and probably the sensitization of the uterine muscle by oestrin to the action of pituitrin is the most important of these changes. Whatever factor or combination of factors bring about the onset of labour (and the endocrine factor is probably very important in this respect), the fact remains that the uterus is relatively unprepared for the advent of parturition so that the process is liable to undergo some alteration in nature.

In the one case included in the Inertia Section labour was modified by the occurrence of a primary inertia. Delivery however was normal. The primary inertia was no doubt due to the relatively unprepared state of the uterus. Another case of prematurity is found in this section but it has been included amongst the group headed "retained placenta": in this case the placenta was adherent and the mother died undelivered of the placenta (vide infra). Here again inefficient uterine action was at least partly responsible for the catastrophe, although delivery was perfectly normal and uneventful.

The incidence of prematurity as a cause of uterine inertia in this series is very small. Prematurity as a cause of uterine inertia is probably relatively uncommon.

(c) Elderly primigravidae.

The elderly primigravida is usually associated with a labour which is pathological or potentially so, and at the mention of the term "elderly primigravida" one imagines all kinds of obstetrical difficulties. Actually, in this series of 752 cases, 19 elderly primigravidae are included and only 2 of these developed a severe degree of inertia, i.e. only 1 in about 10. One of these elderly primigravidae also suffered from uterine fibroids (the case being recorded in Table 29 and marked *). The other, Case No.145, was an example of an

absolute primary inertia in a primigravida of 39, the labour lasting about 6 days from start to finish - indeed in this case, which was observed throughout, it was very difficult to say when labour truly began, as the contractions were established rhythmically for a few hours at a time only, being followed by an interval completely devoid of contractions. The figures have already been considered in the section relating to Age and Parity, and are shown in Tables 2 and 3. The incidence of severe inertia in elderly primigravidae works out at 10.4% of cases, whilst 5 out of the 19 - 26.3% - had labour better than the average. Hence in this series, at least, elderly primiparity does not rank as a common cause of severe uterine inertia.

There are two factors which tend to make labour in elderly primigravidae more protracted. The first is an increase of the fibrous elements of the cervix resulting in an increased resistance to dilatation. The second is the nervous condition often found to exist in primigravidae who are about to undergo the ordeal of labour relatively late in life - psychological inhibition of uterine contractions. These primigravidae are often, but not invariably, highly strung and of nervous temperament. Of these two factors the latter is probably the more important. The mental attitude of the majority of the elderly primigravidae was surprisingly good in this series and

this may account in no small measure for the low incidence of severe inertia amongst them. The other factor - increased fibrosis of the cervix - is more constant and is less likely to vary as much as the psychological factor. Bourne and Bell[71] maintain that in the majority of cases of delayed dilatation of the cervix with good contractions, the delay is due to spasm of the cervical muscle rather than fibrosis of the cervix. This would suggest that the nervous element (leading to inco-ordination of the musculature), is probably of more importance in these cases as a cause of delayed dilatation than the fibrosis found in the cervix.

(d) Unknown Cause.

In two cases there has been difficulty in assigning a cause for the uterine inertia. Case No.297 was that of a primigravida of 32 years who developed a primary inertia, no known cause being discoverable. The age of the patient may account for this as the age falls within the age group 31-35 in which the incidence of uterine inertia was noticed to be greatest (see Table 3). Case No.679 was that of a young primigravida of 26 who had a primary inertia following premature rupture of the membranes in a normal presentation. It is possible that this case may have been due to cervical spasm, although this is difficult to prove unless the cervix is actually felt to contract actively during a uterine contraction.

Deficient innervation of the uterine muscle is a cause of inertia found in many textbooks of Midwifery, but there is no clinical or anatomical justification for this theory.

Retained Placenta.

Four cases of retained placenta due to inefficient uterine action have been included. Uterine inertia of severe degree supervening in the third stage of labour ended in the deaths of all four mothers.

1. Case No.370. See Table 28.

The labour was normal and uneventful until after the birth of the child. The placenta remained attached to the fundus, and owing to the fact that conditions were favourable, expectant treatment was adopted. Ten hours later a free loss of blood and deterioration in the mother's condition resulted in a manual removal of placenta being effected. The mother died of puerperal sepsis.

Separation of the placenta was performed without difficulty. The catastrophe was attributed primarily to impaired retraction.

2. Case No.430. See Table 28.

Premature labour supervened at 35 weeks. Delivery was normal. Separation of the placenta did not occur. There was only slight loss of blood from the uterus, the total amount not exceeding 1 pint. The condition of the patient gradually deteriorated despite intravenous infusion of gum acacia and blood transfusion. The outstanding feature of this case was not loss of blood, but shock which was progressive. The

patient did not rally sufficiently for manual removal to be attempted, and she died 7 hours after birth of the child.

Prematurity, allied to poor general condition of the patient, was thought to be the cause of adherent placenta.

3. Case No.637. See Table 28.

Following manual rotation and forceps delivery of a persistent occipito-posterior position of the head, the placenta was retained. Owing to the amount of blood lost after delivery manual removal was performed. The mother died of puerperal sepsis 14 days later.

Secondary inertia was held responsible for the above emergency, following upon an exhausting and prolonged labour.

4. Case No.750. See Table 28.

In this case of severe uterine inertia, no cause could be assigned to the inertia which developed. Following a quite normal, short, speedy first and second stage, a severe secondary inertia supervened immediately after delivery, the placenta being retained.

Case No.750. Mrs. D.G. Primigravida, aged 26 years. Previous stillbirth (prim.breech). Extremely anxious to have a live child. Labour induced - at term - no disproportion. Two medicinal inductions were unsuccessful and surgical induction by rupture of membranes performed. Following a short latent period a normal delivery was made. Immediately afterwards, a free loss of blood occurred - slight post-partum haemorrhage. The uterus was "gathered up" and compressed from the abdomen. The uterus remained flabby and the patient's condition gradually deteriorated. Intra-

venous gum acacia 2 pints was given, followed by blood transfusion 1 pint. The patient's condition rallied and manual removal was performed quickly under ether anaesthesia, as there was still a steady trickle of blood. The patient collapsed suddenly and died about 2 hours after the manual removal, no further blood loss having occurred.

This case is quoted at some length for two reasons:

(1) The mother was young, healthy, and one would have said in excellent condition for parturition. The uterine contractions were very efficient in the first and second stages, but rapidly deteriorated immediately the second stage was completed.

(2) Two unsuccessful medicinal inductions were followed by surgical induction.

The picture presented by this patient was that of increasingly severe shock, aggravated by loss of blood. The uterine inertia was increased by the condition of shock and the clinical condition became one of complete uterine exhaustion - the only case of true uterine exhaustion seen in this series: the uterus was "gathered up" from the abdomen for the space of 30 minutes without a single uterine contraction being palpable. As stated before in the discussion of Induction of Labour, it is possible that this inertia in the third stage of labour - a so-called "secondary inertia" - may have been due to the use of oxytocic drugs in the unsuccessful medicinal induction of labour.

Inefficient uterine contraction naturally leads to inefficient retraction, and in three of the four cases this

was present; the fourth case was considered to be primarily due to prematurity, although premature labour may pave the way to incomplete and inefficient muscle contraction and retraction. The causes of these untoward results have been ascribed to secondary inertia which supervened following (1) dystocia, (2) premature labour, (3) induction of labour, and (4) normal labour with previously good contractions of the uterus, with in addition induction of labour as an important contributory factor.

Secondary inertia which supervenes during the third stage of labour is therefore to be regarded as one of the most dreaded and catastrophic events in obstetrics. Where inertia is of severe degree the mother is in imminent danger of losing her life, either through the immediate danger of haemorrhage, or the more remote danger of puerperal sepsis. Retained placenta is a condition which gives rise to a severe degree of obstetrical shock which is often further aggravated by post-partum haemorrhage; but even where the haemorrhage is slight the shock may be very severe, and the state of inertia already present is still further aggravated, until a condition of complete uterine exhaustion may be produced. Complete uterine exhaustion is happily a rare event in obstetrics, but where it does occur it is attended by a high maternal mortality. In no stage of labour is uterine muscle efficiency so desirable

as in the third stage of labour. The vast majority of the maternal deaths in this series are due to uterine inertia in the third stage of labour.

P A R T V

(1) Conclusions

(2) Acknowledgements

(3) Bibliography.

CONCLUSIONS

1. 752 cases of labour have been considered. In these the action of the uterus in normal and abnormal labour has been studied and the influence of certain factors on uterine activity noted.

2. Age

   i. Uterine action is best in primiparae below the age of 20 years; in multiparae, below the age of 25 years, and also in the age-group 31 to 35 years.

   ii. Uterine action is poorest in primiparae between the ages of 31 and 35 years; in multiparae, above 35 years of age.

3. Parity

   i. Uterine inertia is more common in primiparae than in multiparae. In genuine uterine inertia the proportion is almost 6 to 1.

   ii. Uterine action better than normal, when it does occur, is more often found in primiparae, 1 in 4 primiparae having a labour better than the average, as compared with 1 in 6 multiparae.

iii. Multiparae tend, as a class, to have a more uniform uneventful labour. Primiparae are more liable to suffer from the extremes of uterine action, good and bad, and to have a less uniform labour.

4. Maternal Disease

i. Not attributable to pregnancy.

a) Cardiac Disease.

Cardiac disease is not a common cause of impaired uterine action; in this investigation uterine inertia has been the exception in cardiac disease. The routine treatment of cardiac disease by rest, diet, rich in glucose, and the administration of salts of calcium has helped to ensure good uterine muscle activity, in addition to good cardiac muscle performance.

b) Nervous Disease.

Provided the reflex nervous paths are intact, labour, once initiated, progresses uneventfully to a successful conclusion. The higher nerve centres do not control parturition, but merely modify and regulate its course. Labour is largely a matter of the local myogenic contraction, and the local reflex activity of the uterus itself.

c) Uterine Fibroids.

Elderly primigravidae with fibroid uteri are more prone

to develop uterine inertia. Multiparae are relatively unaffected. The effect of fibroid and other tumours of the uterine wall is to interfere with the local myogenic contraction.

d) Anaemia.

Anaemic states per se are not a common cause of impaired uterine activity. Ischaemia of uterine muscle may be a factor in the causation of uterine "pains".

e) Constitutional Disease.

The number of cases of constitutional disease is insufficient to allow definite conclusions to be drawn. Diabetes mellitus and extreme obesity are probably causes predisposing to impaired uterine output.

ii. Attributable to Pregnancy.

a) Albuminuria of Pregnancy.

(1) Uterine action is better than the average in more than half the cases (53.8%). 89 cases of albuminuria are considered.

(2) Uterine inertia is the exception, and when it does occur can often be ascribed to some other complicating factor. When inertia does occur, the degree of inertia bears no relation to the severity of the albuminuria.

(3) In albuminuria of pregnancy, in addition to being

shorter in duration, labour is on the whole less painful. Five "painless" labours are considered. Factors which predispose to shorter and less painful labours are salt-free or salt-poor diet, increased oedema or vascularity of the tissues, particularly of the pelvic floor and birth canal.

b) Ante-partum Haemorrhage.

i. Seventeen cases of ante-partum haemorrhage are considered. Seven were due to accidental haemorrhage, ten to placenta praevia.

ii. Uterine action is more efficient in placenta praevia than in accidental haemorrhage due to -

(1) Methods of treatment are designed to exert greater pressure on the cervical and paracervical ganglion.

(2) Increased vascularity of lower segment, cervix, and pelvic floor in placenta praevia.

(3) The pathological changes in accidental haemorrhage reduce the efficiency of the musculature of the uterus.

(4) The type of patient in placenta praevia is not debilitated over a long period by co-incident toxaemia, as in accidental haemorrhage.

5. Social and Economic Factor (including Nutritional Factor).

i. The best uterine action is found in the average working class woman. In her, nutrition, muscular

tone, and general behaviour, mentally and physically, are in proper proportion.

ii. The tendency to develop uterine inertia is greatest amongst the poorest class of society. This is attributed to states of under-nutrition and exhaustion of the muscular system, voluntary and involuntary.

iii. The middle class mother is intermediate in uterine performance between the average working class and the very poor, impoverished mother.

iv. Measures which aim at improving "tonus" and states of nutrition combat the tendency to develop uterine inertia in a certain proportion of cases. States of malnutrition alone do not give any increased incidence of impaired uterine action, its influence being slight, other factors being equal; states of malnutrition in multiparae show a greater tendency to the development of impaired performance than in primiparae.

6. Physique.

i. The state of development of the skeletal muscular system is no index of the action of the uterine muscle in labour. Tonus in voluntary and involuntary muscle is of essentially different character.

ii. Overdistension of the uterine muscle predisposes to the onset of uterine inertia; this is shown to be due to loss of tonus.

iii. States of obesity predispose to the onset of impaired uterine muscle action, probably due to hormonal influence, the pituitary gland being mainly at fault. The "inertia type" of woman is found in this class.

7. Psychological Factor.

i. States of over-anxiety, apprehension, and suppressed fear lead to a reflex inhibition of uterine contractions and predispose to the onset of uterine inertia. This is attributed to the liberation of adrenaline into the circulation with inhibition of uterine contractions.

ii. Illegitimacy is not a factor predisposing to uterine inertia.

iii. Neurotic and hysterical states, as compared with states of apprehension, over-anxiety, and suppressed fear, cause a lesser, but none the less definite, degree of delay in labour.

8. Relation of Rupture of the Membranes to Uterine Inertia.

157 cases of premature rupture of the membranes have been considered.

i. Premature rupture of the membranes, whether spontaneous or artificial, leads in normal presentations to a labour which is shorter in duration in 40% of cases.

ii. (a) The incidence of uterine inertia is not any greater in normal presentations with premature rupture of the membranes. Delivery is normal in almost 95% of normal presentations.

(b) It is much higher in abnormal presentations and positions. This is ascribed to the fact that the pressure of the presenting part on the cervix is irregular and uneven; the cervical reflex is imperfectly stimulated, and uterine contractility is inadequate.

iii. The duration of the latent period in premature rupture of the membranes, whether spontaneous or artificial, is no reliable index of the duration of labour subsequently.

The duration of the latent period does not differ materially in spontaneous as compared with artificial rupture of the membranes (surgical induction).

9. *Induction of Labour*.

Eighty-nine cases of induction of labour are considered.

i. Induction of labour by means of drugs, which include pituitrin, predisposes to the onset of impaired uterine action.

ii. If medicinal induction as above is unsuccessful and is followed by surgical induction by rupture of the membranes, the possibility of uterine inertia developing is increased.

10. i. Uterine inertia is due to faults in the uterine forces in 64.7% of cases, the remaining 36.3% being due to faults in the passenger and passages (malpresentations, malpositions, and contraction of the pelvis).

ii. Uterine inertia, especially supervening in the third stage of labour, adds greatly to the risk to the mother. The maternal mortality in this series is 6 in a total of 752 cases, or 8 per 1,000. This high figure is attributed to uterine inertia in the third stage of labour, which is considered to be the most dreaded complication of labour.

ACKNOWLEDGEMENTS

I would like to tender sincere thanks to E.C.Dutton, M.B.,F.R.C.S., Medical Superintendent of Stepping Hill Hospital, Stockport, for the facilities granted in collecting and investigating this series of cases, and also to the Sisters and other members of the nursing staff, Obstetrical Department, Stepping Hill Hospital, for their kind assistance in compiling the records of labour etc.

BIBLIOGRAPHY

1. Hofbauer, J.: J. Amer. Med. Ass., 1929, 92, 540-544.
2. Frankl, O.: J. Obstet. Gynaec. B.Emp., 40, 297.
3. Whitehouse, Beckwith, H., and Featherstone, H.: J.Obstet. Gynaec. B.Emp., 30, 565.
4. Gemmell, A.A.: J. Obstet. Gynaec. B.Emp., 1926, 33, 259.
5. Dahl, W.: Zeitschr. f. Geburtsh. u. Gynäkol, 1916, 78, 539. Quoted by A.A.Davis in J.Obstet.Gynaec. B.Emp., 40, 491.
6. Davis, A.A.: J. Obstet. Gynaec. B.Emp., 40, 493.
7. Johns Hopkins Hosp. Bull. Baltimore, 1925, 36, 280-298.
8. Winton, F.R., and Bayliss, L.E.: Human Physiology, 1930, p.62.
9. Heller and Holtz: (1932) J. Physiol., 74, 134.
10. Blair-Bell, W.: Proc.Roy.Soc.Med. (Obstet. & Gyn.) 1915, 8, 71.
11. Blair-Bell, W., Datnow, M.M., and Jeffcoate, T.N.A.: J. Obstet. Gynaec. B.Emp., 40, 564.
12. Fontes, F.: Compt. rend. soc. de Biologie, 1929, 102, 212, Cited by Blair-Bell in J.Obstet. Gynaec. B.Emp., 40, 565.
13. Winton, F.R., and Bayliss, L.E.: Hyman Physiology, 1930, p.69.
14. Dudley, H.W., and Chassar Moir; Brit.Med.J., 1935, I, 520.
15. Ganner, P.J.: Brit.Med.J., 1935, 2, 205.
16. Mitchell, D.A., and Bradbroke, H.N.: Brit.Med.J., 1935, 2, 205.

17. Dale, H.: J.Physiol., 1929, 68, 97.

18. Walker and Henderson : Canad. Med. Ass. J., 1934, 30, 158.

19. Lancet, Editorial, 1936, 4, 903.

20. Drew-Smythe, H.J., and Thompson, D.Joan: J.Obstet.Gynaec. B.Emp., June 1937, p.478.

21. Allen, E., and Doisy, E.A.: Physiol. Review, 1927, 7, 600.

22. a) Robson, J.M.: J.Physiol., 1935, 84, 130.
b) Parkes, : J.Physiol., 69, 463.

23. Robson, J.M.: J.Physiol., 1933, 79, 139.

24. Robinson, A.L., Datnow, M.M., and Jeffcoate, T.N.A.: Brit.Med.J., 1935, I, 749.

25. Falls, F.H., Lackner, J.E., and Krohn, L.: J.Amer.Med.Ass., 106, 271.

26. Jeffcoate, T.N.A.: Brit.Med.J., 1937, I, 720.

27. Cohen, S.L., Marrian, G.F., and Watson, M.: Lancet, I, 228, 674.

28. Dahlberg, C.: J.Obstet. Gynaec. B.Emp., 1935, 42, 953.

29. Knaus, H.: Arch. Exp. Path. Pharmak., 1927, 124, p.152.

30. Robson, J.M., and Paterson, S.J.: Brit.Med.J., 1937, I, 312.

31. Morgan, T.N.: J.Obstet. Gynaec. B.Emp., 1935, 42, 80.

32. Allen, E.: Amer. J. Physiol., 1930, 92, 612.

33. Robson, J.M.: Brit.Med.J., I, 1936, p.1035.

34. Hain, A.M.: Quart. J. Exp. Physiol., 1932, 22, 249.

35. Collip, J.B.: J. Amer. Med. Ass., 1935, 104, 556.

36. Blair-Bell, W.: Brit.Med.J., 1909, 2, 1609.

37. Cow, D.: J.Physiol., 1918, 52, 301.

38. Robson, J.M.: J.Obstet. Gynaec. B.Emp., 40, 500.

39. Clark, A.J., and Knaus, H.: J. Pharmacol., 1926, 26, 347.

40. Bourne, A.W., and Burn, J.H.: Lancet, 1928, 2, 694.

41. David, J.C., and Vaneed, C.: Indian Med. Gaz., 1929, 64, 73.

42. Cushing, H.: Lancet, 1930, 2, 119, 175.

43. Queen Charlotte's Obstetrics, 1933, p.579.

44. Queen Charlotte's Obstetrics, 1933, p.491.

45. Ham: Amer. Med. J. and Record, May 1933.

46. Diasio: Amer. Med. J. and Record, Oct. 1930.

47. Bourne, A.W., and Burn, J.H.: J.Obstet. Gynaec. B.Emp., 34, 247.

48. Blair-Bell, W., Datnow, M.M., and Jeffcoate, T.N.A.: J.Obstet. Gynaec. B.Emp., 40, 563.

49. Miller, E.G., Cockrill, J.R., and Kurzrok, R.: Amer. J. Obstet. Gynec., 33, 154.

50. Queen Charlotte's Obstetrics, 1933, p.116.

51. Kreis, J.: J.Obstet. Gynaec. B.Emp., 41, 958.

52. Chassar Moir: Trans. Edinb. Obstet. Soc., 1934.

53. Ivy, A.C., Hartman, G.G., and Koff, A.: Amer. J. Obstet. Gynec., 22, 338.

54. Theobald, G.W.: Brit. Med. J., 1936, 2, 1307.

55. Queen Charlotte's Obstetrics, 1933, p.257.

56. Prys-Jones, T.B.: Brit. med. J., 1936, 2, 627.

57. Fairbairn, J.S.: Textbook of Gynec. with Obstet., p.204.

58. Cameron, S.J., and Thompson, H.: Lancet, 1932, 2, 731.

59. Nahmmacher: 1930, Zbl. GynHk., 54, 1820.

60. Fagin, J., and Reynolds, S.R.M.: Amer. J. Physiol., 1936, 117, 86.

61. Reeb, and Israel: Gynéc et Obstét., 1933, 27, 193, from Abstract by H.C.Mack, Internat. Abstract Surg., 57, 237.

62. Karpati, J.: Zbl. Gynäk., Leipzig, 1935, 59, 2593-2640. From Abstract, J. Amer. Med. Ass., 1936, 106, 83.

63. Blair-Bell, W.: J.Obstet. Gynaec. B.Emp., 1933, 40, 544.

64. Goodall, J.R.: "The Inertia Syndrome", J.Obstet. Gynaec. B.Emp., 41, 257.

65. Blair-Bell, W.: J.Obstet. Gynaec. B.Emp., 34, 249.

66. Drew-Smythe, H.J., and Thompson, D. Joan: J.Obstet. Gynaec. B.Emp., June 1937, p.744.

67. Bourne, A., and Bell, A.C.: J.Obstet. Gynaec. B.Emp., Vol.40, p.423.

68. Blair-Bell, W., Datnow, M.M., and Jeffcoate, T.N.A.: J.Obstet. Gynaec., 40, 559.

69. Bourne, A.W., and Bell, A.C.: J.Obstet. Gynaec. B.Emp., 40, 426.

70. Roques, F., and MacLeod, D.H.: J.Obstet. Gynaec. B.Emp., 39, 340.

71. Bourne, A.W., and Bell, A.C.: J.Obstet. Gynaec. B.Emp., 40, 439.