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From Unit "B,' Kandang Kerbau Hospital for Women.
Moderator: DR. A. C. SINHA.

(a) A Case Of Procidentia

Case Report:

GY/B/1330/55. G. K. Chinese. Aged 37 years.

Chief complaint:
Something coming down in the vagina since the 5th. confinement 2 years ago.

Obstetrical history:
Menarche at 16.
Menstrual period regular, 30/3 days.
Married at 20.
5 full term, normal deliveries at home, birth weight unknown; eldest is 8 years old, youngest 2 years old; one abortion at 5th month one year ago.
Puerperia—no proper supervision with no home help available.

Social history:
Poor financial circumstances.
Husband out of work for 2 years; paient is the sole support of the family, hawking by the roadside earning about $50. to $60. p.m..
Receiving financial assistance from the Social Welfare Dept.

Physical Examination:
General condition fair.
Looks anaemic.
B.P.—90/60. Temperature normal.
Heart and lungs—nil of note.
Abdomen soft, abdominal wall lax.
P. V. examination:
(1) Inspection—total prolapse of uterus, rugae of mucosa absent, epithelium thickened; Trophic ulceration of cervix.
(2) Bimanual—small uterus felt completely prolapsed outside vulva; deficient perineum.

Laboratory Investigation:
TR—2.04 million
Hb—39%

Management:
(1) Building up for vaginal hysterectomy at later date.
(2) Temporary ring pessary used.

Discussion:

DR. N. N. LING: Presented the case.

DR. T. K. CHONG: Read a commentary on the pathology and etiology of genital prolapse.

The pelvic organs in women are supported by two layers of tissue, the pelvic fascia (or Endopelvic fascia) and the levatores ani muscle. In certain situations, the pelvic fascia is thickened and reinforced to form the pubocervical, the cardinal and the utero-sacral ligaments which are composed mainly of fibrous and elastic connective tissue, and also a certain amount of smooth muscle fibres.

Kenneth Pacey in his thesis on the Pathology and Repair of Genital Prolapse suggested that these pelvic ligaments are only the secondary uterine and vesical supports, that they are but guy ropes which steady the organs in space, and that, without muscula support, they elongate and attenuate as readily as does any other unsupported ligamentous structure in the body.

He therefore considers the levatores ani muscles as very important supports of the pelvic viscera. This muscle is composed of 3 parts—the ischio coccygeus, ilio-coccygeus and the pubo coccygeus, of these the pubo coccygeus is by far the most important. The combined pubo coccygeus muscles form "The Three Pelvic Sling" as described by Pacey. The muscle is in a state of constant tonus whereby it carries out its supporting functions. Further, it is in a state of constant reflex activity. Any rise in the intra-abdominal pressure, from the minor rhythmical ones of respiration to the sudden marked ones of coughing and sneezing, results in its reflex contraction. The contraction imparts a lift to the pelvic organs so that no strain is imposed on the pelvic fascial ligament.

Pacey explains the genesis of prolapse as due to rupture of the anterior and middle raphes or both with consequent destruction of the slings. Rupture of the anterior raphe alone will lead to retraction of the pubo coccygeal support of the bladder, the weight of which is now entirely borne by the pubo-cervical fascia which eventually gives way and cystocele and urethrocele or both result.

Rupture of the middle raphe removes the muscle support to the uterus and vagina, the brunt of which will now be borne by the cardinal ligaments.

Rupture of both anterior and middle raphes deprives both pubo cervical and cardinal ligaments of muscular support, and vault prolapse, cystocele and urethrocele will eventually result. The wide separation of the pubococcygeus muscles must eventually lead to procidentia.

In the ethiology of Prolapse therefore, *trauma of labour* is the most important primary factor though frequently associated with other factors.

The predisposing abnormalities of childbirth which tend to cause subsequent prolapse are:—

(1) The passage of large children through the birth canal. The longer the duration the worse is the stretch.

(2) Precipitate labour.

(3) The application of forceps before full dilatation of the cervix. On traction, the forceps pull down the cervix and thus stretches both the cardinal and the utero-sacral ligaments.

Idiopathic weakness of the supporting structure of the viscera is of next importance to childbirth as a primary cause of prolapse. Perhaps both congenital and acquired factors are concerned. One example of a congenital factor is the local undevelopment of the pelvic floor in some cases of nulliparous prolapse. An example of an acquired factor is the muscular atrophy of old age, though whether the effect is essentially a sexual senescence rather than a general senescence is a debatable issue. This partly explains the ultimate prolapse at or after the menopause, the previous relaxation of the pelvic floor not having been sufficient in itself to cause it.

The third cause of prolapse is postural-strain. If postural strain is prevented during the months following delivery, most of the childbirth injuries will heal, and especially if re-educative exercises are encouraged, anaemia is corrected, the general health is improved and gradual household duties are resumed. Exercise in the postnatal period is extremely important for even with a complete perineal tear with a torn perineal body, prolapse is uncommon.

DR. A. C. SINHA: Then elaborated on the basic fundamental anatomical concepts of the pelvic support and called upon Professor Sheares to add any further information that he may have in the light of modern researches.

PROF. B. H. SHEARES: Did not agree with Dr. Chong in that the main genital support was the levator ani muscle. He thought it was the endopelvic fascia. Continuing he said that the pelvic supports were always considered during the operation for the repair of prolapse and that it was not always possible to repair the supports of the hernia anatomically.

DR. S. T. JAMES: Gave a detailed discussion on the treatment. Treatment would necessarily vary with the different types of prolapse. Using Malpas' classification, he suggested the following:

(1) *Primary utero-vaginal prolapse* of first and second degree — best treated by Fothergill operation.

(2) *Complete or Total prolapse.* The treatment of choice was vaginal hysterectomy with high repair of pelvic floor, or in less skilled hands a Fotherall operation might suffice. If there was any doubt about the strength of the tissue available for repair, the Le Fort or inter-position operation of Watkins occasionally found their application.

(3) *Anterior vaginal wall prolapse* (cystocele and/or urethrocele). Usually a well designed colporrhaphy operation sufficed.

(4) *Posterior vaginal wall prolapse:*

(a) enterocele (hernia of Pouch of Douglas). Closure of the peritoneal pouch though desirable, was of secondary importance to the restoration of

the prolapsed viscus to its normal position. A high colporrhapry should be done, either as a separate operation or to complete a Fothergill repair or a vaginal hysterectomy.

(b) Rectocele.

(c) Defective perineum. Treatment was posterior colporrhaphy or colpoperineorrhaphy depending on whether there was a complete tear of the perineal body, whose repair was almost a necessary complement to any operation on the anterior vaginal wall.

(5) *Nulliparous prolapse.*
Treatment was either by a tight Fothergill repair or vaginal hysterectomy and repair.

(6) *Prolapse after hysterectomy:*
The elective treatment was surgical. Non-operative treatment — pessary and re-education exercises —was permissible if the prolapse was not causing symptoms and provided the cervix had not come outside the vulva.

DR. A. C. SINHA: Considered that a pelvic floor repair was a necessity in all the operations. Further, the age of the patient had to be considered in the choice of the operation. Fixation operations were not done now-a-days.

PROF. B. H. SHEARES: Thought that the classification by Malpas was rather complicated; Malpas stressed on the occurrence of enterocole but very few such cases were seen. No cases of prolapse after hysterectomy had been encountered by him. He advocated the division of treatment of prolapse into anatomical and and unanatomical operations.

The dictionary definition of pessaries was "various appliances inserted into the vagina for various purposes."

The pessary was useful as an adjunct in various forms of treatment of prolapse e.g. in this case, where it was useful in alleviating discomfort to the patient prior to operation. The pessary could best be described as a "prop"that lifted up. The uses of the pessary in modern gynaecology were:

(1) in a woman of childbearing age where despite good postnatal care the uterus was retroverted, with a healthy cervix. Either a Smith-Hodge or ring pessary should be used. In additional perineal exercises and the avoidance of over-distention of the bladder were advised. If after three months the uterus was still retroverted, then the pessary should be discarded but the patient should continue with the perineal exercises.

(2) During the child-bearing age if symptoms due to retroversion were present.

(3) During early pregnancy if the uterus was retroverted, and marked prominence of the sacral promontory existed. This would predispose to incarceration.

(b) A Case Of Intra-Uterine Death

Case Report:

Reg. No. 2982. L.M.T. Age 41 years. Para 5. Gravida 6.
Admitted 15.2.1956 at 11.30 a.m.
L.M.P. 28.7.1955. E.D.D. 4.5.1956.
Estimated period of gestation—28 wks. 5 days.

Previous Obstetrical history:

The patient has had 5 normal deliveries and all of her children are alive.

Present history:

She has admitted on 15th February, 1956 with the following complaints:—

(1) No foetal movements for the past 3 days.

(2) Oedema of both legs with slight pain, over the right leg for 1 week.

Physical examination:

(a) **General:**
Afebrile. B.P. 110/70. Pulse 78/minute.
Heart)
Lungs) Clinically clear .
Abdomen—Soft.
Legs—slight oedema—both sides, with tenderness and redness of the right lower leg.

(b) **Obstetrical:**

Height of fundus—size of 30 weeks pregnancy.

Vertex L.O.A. Head floating. Foetal heart sounds not heard. Foetal movements not felt.

No egg-shell crackle of foetal skull bones elicited.

Height of uterus—13"

Girth — 35"

Provisional Diagnosis:

Inter-uterine foetal death with cellulitis of right leg.

Investigations:

16.2.1956:

TR.—3.62 millions. TW.—5,800.

Hb—5%. DC. P.66, L.20, M.14. E.0%. no sugar.

20.2.1956: X-ray of abdomen (475/56).

Report: No significant overlapping of skull bones but there is suspicious collapse of the foetal skeleton with the mother in the erect position. Suggest a repeat in 7-10 days.

Progress Notes:

25.2.1956: General condition of patient—satisfactory Foetal heart sounds still not heard. Slight decrease of liquor amnii noticed clinically. Height of fundus now decreased to 12." Girth 35" still.

Notwithstanding the X-ray report, it was felt that this was a case of intra-uterine foetal death, and the patient was hence put on Stilboesterol tab. 5 mgm. t.d.s. for 5 days.

1.3.1956: A repeat X-ray as requested was done (594/56).

Report: There is now moulding of the skull and the foetus appears more crouched in the erect than in the supine position. Appearances likely due to foetal death.

The patient in the meantime had completed her course of Stilboesterol and was sent to Labour Ward for a pitocin drip but refused treatment, and has since gone home against medical advice.

Discussion:

DR. R. LOH: Presented the case.

DR. F. Y. KHOO: (Radiologist — General Hospital) discussed the main radiological signs of foetal death, viz:

(1) *Collapse of the foetal skull with overlapping of the cranial bones* (Spalding's sign). It was not an infallible sign because sometimes it was not seen, and also if the patient was in labour moulding would give rise to a similar picture. It was not possible to differentiate between the two. Sometime if the sign was doubtful, placing the patient in the erect posture would help make the sign more positive. Spalding's sign would show up from 10-15 days after foetal death.

(2) *Collapse of the foetal skeleton.* The dead foetus due to lack of tone yields to intra-abdominal pressure resulting in a crumbled up foetus.

(3) *Movement of the foetus.*

(4) *Presence of gas in the heart, aorta, in the blood vessels.* If present it was very significant of foetal death. Gas might appear within one to two days of foetal death; its origin was not known and it might disappear within a few days. Further, gas was more likely to appear in those babies nearer term.

(5) *Lithopoedian* — dense bones; the child was wrapped in a calcified capsule.

DR. C. S. OON: Read a commentary on the causes of the onset of labour. Reviewing the various theories she said that it was likely that more than one factor was operative and moreover these factors varied from species to species and from individual to individual. The following have been named as possible causes:—

(1) Mechanical. (2) Hormonal. (3) Nervous. (4) Nutritional. (5) Circulatory.

The theories regarding the cause of the onset of labour are legion. As early as 460-370 B.C., Hippocrates postulated that the onset of labour is determined by the nutritional needs of the foetus; that is when the placental mechanism is no longer able to satisfy the foetus, the latter seeks another environment. In the 16th, 17th and 18th centuries, French workers attributed it to muscle reaction to increasing distension of the uterus. The 19th and 20th centuries saw the development of various nervous, mechanical and hormonal theories to account for the onset of labour.

Most of the current theories have an element of truth in them, but their fault lies in the fact that each one of them is based on the assumption that there is but a single cause of labour. The theory placing importance on nerve stimulation is ruled out by the onset of normal labour in paraplegics. The effect of local mechanical conditions is made doubtful by the onset of expulsive uterine contractions at term in cases of extrauterine pregnancies. The importance of hormonal causes appears questionable following observations made on women with double uteri, when one horn may go into labour without the other (Colaco 1949).

Reynolds (1949) emphasized "that parturition begins as a result of the gradual accelerating convergence of a number of factors—structural hormonal, nervous, nutritional and circulatory — which, at a time characteristic for each species and adapted to the morphologic condition present in each, are so associated that they lead to evacuation of the uterus by its contents."

Foetal pressure on the uterine wall appears to be a significant factor and may partly explain why rupture of the membranes usually results in the onset of labour. The mechanism at work is not clear; it may be pressure on the lower segment and the paracervical nerve ganglia. The influence of increasing muscle tension is illustrated by the tendency to premature onset of labour with multiple pregnancy and hydramnios. In support of the theory that foetal nutritional requirement is a cause of onset of labour is an observation made by Malpas in 1950. He pointed out that if anencephaly is not complicated by hydramnios, it is frequently a cause of post-maturity. He suggested that this is because the onset of labour is ordinarily determined by progressive anoxaemia of the foetal brain cortex. This mechanism breaks down in anencephaly because the brain is so poorly developed.

Uterine muscle is under the influence of hormonal factors, it being a stabilising one during pregnancy. The sine qua non of parturition is the withdrawal of these factors.

The role of *oestrogen* in the onset is unsettled. It was long ago demonstrated that oestrogens can produce abortions in animals and that the level of oestrogen in the blood and urine is highest about the time of labour in both human and animals. So, the onset of labour was explained by a rising oestrogen and a falling progesterone influence by Aschheim and Zondek (1927). Cohen and other (1935) and confirmed by Smith & Al (1938) showed that not only is there a gradual rise in the level of oestrogen in the urine as pregnancy progresses, but, whereas during pregnancy 90% is present in a biologically inactive form, at the onset of labour it becomes mostly free and active. But Clayton & Marrion (1950) denied that there is any evidence that oestrogen in circulation becomes freed from glycuronic acid at the onset of labour. Jeffecoate (1940) claimed that when the progesterone level falls as in missed abortion or intrauterine foetal death, oestrogen will promote uterine contraction and expulsion of the products of conception. However, in recent years after further observations, he has questioned his own statement as it was not proved by a controlled investigation. Smith & Smith (1949) showed that even massive doses of oestrogen (e.g. 121 mgm. Stilboesterol daily) in late pregnancy will not induce premature labour.

Ancel and Bouin (1912) put forward the idea that progesterone allays spontaneous uterine contractions and lowers the sensitivity of the uterus to oxytocics. In animals, the removal of the corpus luteum during pregnancy leads to expulsive uterine contractions and abortion, whereas the administration of progesterone delays the onset of labour. Such observations gave rise to the idea that parturition begins following progesterone withdrawal. Several workers supported this theory that progesterone has an inhibitory influence on the myometrium. Then Schultz (1931) and Moir (1933) demonstrated that progesterone does not inhibit contraction of the human uterus or prevent its response to oxytocin. In fact, it causes contractions of lesser frequency but higher amplitude. It has been found the excretion of pregnandiol reaches a maximum at term; this, indirectly supports the view that withdrawal of progesterone is not a cause of the onset of labour.

The view that oxytocin plays a part in the onset and maintenance of expulsive uterine contractions is gaining popularity. It has been shown that uterine action is disturbed to a marked degree in the case of diabetes insipidus in both women and animals (Harris 1949). Late onset of

labour seen commonly in obese women and in the dystrophia dystocia syndrome might be explained by a hypothalmic — pituitary disturbance.

Oxytocin normally released from the pituitary, is believed to be liberated from the placenta as well, in response to degeneration or ischaemia.

Today, intravenous pitocin drip to induce labour is widely used. It is recommended that a sensitivity test should first be done, and those women showing any sensitivity should not have any more of this hormone. The usual strength of the solution used is 5 units of pitocin to 1 pint of 5% dextrose, starting, at 8 minims per minute and increasing by 4 minims every half hour to a maximum dose of 30 minims per minute. Meantime, the patient should be carefully watched by a medical officer. The frequency, duration and amplitude of the contractions should be recorded on a tocograph if possible, otherwise careful clinical note should suffice. The intravenous method can be better controlled and stopped at once when necessary.

Finally, the above is not a complete list of the possible causes of the onset of labour, and it is not possible that any one single factor can be the cause. It is more possible that parturition results from an interplay between the mechanical relationships of the foetus and the uterus, the hormones and other secretions of the placenta, and the response of the maternal endocrine and nervous system to the foetus and its placenta.

DR. A. C. SINHA: Said that in cases of intra-uterine foetal death, nature would throw out an unwanted ovum or dead baby. Surgical methods of including labour were indicated when medical methods had failed. There had been several cases in which labour was successfully induced by surgical means wtihout harm to the mother.

PROF. B. H. SHEARES: Did not recall any case of intra-uterine foetal death where spontaneous labour was postponed for more than four weeks. He therefore advocated non-interference, so as not to convert an aseptic process into a septic one. Pitocin was used not to initiate labour but to ripen the cervix — in doses of 30 units over 48 hours. After 5 to 6 days the dose was repeated. The process might have to be repeated three to four times before the cervix was ripened.

DR. J. LEWIS: Said that a German preparation "UTUS" paste had been used as a means of inducing labour.

PROF. B. H. SHEARES: Though there was a great danger of rupturing the membranes while inserting the paste, and it should therefore be confined to inducing abortion up to the six months of pregnancy.

DR. T. H. LEAN: Mentioned the possibility of a dead foetus in utero giving rise to a lowered fibrinogen content of the blood and thus to a bleeding tendency.

DR. A. C. SINHA: Commented that vaginal bleeding when the contents of the uterus were surgically removed in cases of missed abortion was a real danger. He had one case of cervical missed abortion was a real danger. He had one case of cervical missed abortion which when evacuated resulted in uncontrollable haemorrhage. A hysterectomy had to be done, but the patient died.

DR. C. S. OON: Recalled a recent case of accidental haemorrhage with a bleeding tendency. Hysterectomy was done and the patient recovered after being transfused with two pint of fresh blood.
