PREMATURE RUPTURE OF MEMBRANES

by

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Introduction

The pattern of labour is not constant in all the cases. We come across patients having different types of uterine contractions. Some have inertia, some have normal contractions and some have very strong pains. Similarly, the time of rupture of the bag of membranes is also not constant. Premature rupture of membranes has been defined as rupture of the membranes before the cervix is half dilated by Rudolf. The subject "Premature Rupture of Membranes" brings into mind several questions: Is the duration of labour increased or decreased after premature rupture of membranes? Are there any increased chances of intrauterine infection? Are the foetal and maternal mortality and morbidity rates increased? Contradictory statements have been made by many obstetricians who have studied the subject and contradictory results have been reported.

William Smellie (quoted by King), in 1752, cautioned against rupturing the membranes, but stated: "The membranes appear to play no part in labour except to hold in water necessary for lubrication. Dilatation is effected by the head". Thomas Denman in 1778 stated: "The amniotic fluid enclosed in membranes procures the most gentle, efficacious dilatation of os uteri", but, he has admitted, "in many cases membranes break spontaneously before the period of complete dilatation without any material inconvenience". Baudeloque in 1789 held a gloomy prognosis for dry labour even though he did artificial rupture of membranes for induction of labour.

Norris and Eastman believe that premature rupture of the membranes prolongs labour and causes more pain. Rotation may be arrested. The cervix does not dilate properly and might become oedematous. Contraction rings are prone to develop. Uterine atony due to exhaustion and nervousness of the patient and operative interference are more common. On the other hand, Bishop neither had any increased incidence of operative interference nor increased foetal and maternal mortality and morbidity (with premature ruptures of the membranes). Calkins found definite shortening of the first stage of labour. Grier, Husbands, Kreis, Reycraft, Greenhill and others go to the extent of recommending artificial rupture of the membranes routinely in all suitable cases to induce labour electively so as to hasten the labour and to give a sense of security to the patient.

Abridged

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Methods and Material

As I was interested in finding out for myself the effect of premature rupture of the membranes, I studied 60 cases of premature rupture of the membranes with 40 control cases. I included those cases who came at full-term with history of draining or having ruptured the membranes at home or who were already in the hospital and vaginal examination was done after rupture of membranes for some other indication. I excluded those patients who could not give definite timing about the onset of draining or labour pains and also those patients who gave a vague history of draining and I could not make sure clinically, whether they were really draining. The fallacy of the passage of urine was excluded by scrutinizing the history and by careful observation. A detailed history of the patient was taken and after a general and abdominal examination, a vaginal examination was done to find out the following:

1. Dilatation of the cervical canal and condition of the cervix, whether ripe or unripe. The time of examination in relation to the onset of labour was noted.
2. Presence or absence of the bag of membranes to know whether the forewaters had ruptured or the hind waters; if present, its character whether bulging, tense or flat.
3. Condition and the level of the presenting part in the pelvis.
4. The size and the shape of the pelvis.

These patients were hospitalised irrespective of presence or absence of pains and were kept in bed. The foetal heart sounds and mother's pulse were watched half hourly. Character of pains, the character and the amount of liquor amnii and whether it was draining continuously or intermittently were noted. In some patients the membranes ruptured before the onset of pains and the interval between the onset of draining and the onset of labour was noted as the 'lag period'. When patient went into labour the progress of labour, its nature and the duration of the first and second stage (combined) was noted. It was not always possible to find out the demarcation between the first and second stage of labour without repeated vaginal examinations, which was not possible. Condition of the baby at birth, presence of caput or excessive moulding, whether asphyxiated, birth weight and its progress were noted. The mother was followed up in the puerperium for any evidence of sepsis.

For control cases, I took patients at full-term who did not give any history of leaking. This was confirmed by vaginal examination and by subsequent observation. Abnormal cases were excluded entirely from this series.

Incidence

The incidence of premature rupture of membranes has been variously quoted. Atkins and Embrey found it higher in cases of contracted pelvis, malpresentation or malposition, while Alberto and Gross Rudolf did not find such difference. According to Mason, it is different in multiparae and primiparae, 16% and 18%

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respectively, and in multiparae 14% below the age of 30 years and 20% above the age of 30 years. But Tonkes did not find such disparity and he gave the incidence of 14.9%. Sunde, Atkins and Norris found it more common in primiparae than in multiparae. None of them could explain why it should be higher either in primiparae or multiparae. On an average, Calkins and Sunde reported an incidence of about 14%.

Etiology

No apparent etiological factor became evident after these cases were reviewed. Trauma was not found to be a cause in any case. There were only 7 cases with significant prenatal complications, viz. 3 cases of pre-eclampsia and one each of eclampsia, mitral stenosis, hydramnios and oligohydramnios, while there was 1 case of pre-eclampsia and 3 cases of anaemia in control group. They might be the cause of onset of premature labour but not of the premature rupture of membranes.

Twenty-five per cent of patients had malpresentation and cephalo-pelvic disproportion. Out of 55 vertex presentations, 5 were posterior positions and 2 had cephalo-pelvic disproportion. There were 6 breech presentations and 2 cases of twins. The greatest incidence of premature rupture of membranes occurred between the ages of 20 and 29 years, but this is not of much significance as this is the age period during which the majority of deliveries occur. One patient gave definite history of draining during a previous delivery and one, fifth para, gave history of draining for 3-4 hours during all the deliveries. Munro Kerr has observed repetition of premature rupture in subsequent deliveries and so he attributed this phenomenon to unusual friability of the membranes but he did not find fragile membranes in all the cases. Whether the parity plays any part in premature rupture of membranes cannot be said, as no conclusions could be drawn from my small series of cases. No relationship was noted between the weight of the baby and the incidence of premature rupture of the membranes.

Much work has been done on this subject to find out the etiology. In 1950, Knox and Horner thought that infection of cervical canal might spread to the membranes lying over the internal os, making them inflamed and friable and therefore more likely to rupture prematurely. They studied the membranes after delivery. The membranes were smooth, shiny and pinkish grey in colour without any evidence of inflammation in control cases except in 2 out of 12 cases. While in the group where membranes ruptured prematurely all the membranes were dull and shaggy in appearance, rough and thick in consistency and showed evidence of acute, chronic or mixed infection. Their findings are not confirmed by any other author.

Schulre, in 1929, thought that this phenomenon occurred due to lack of tensile strength, probably due to deficient development of connective tissue layers. But Danforth, Melin and States in 1953 proved that premature rupture of the membranes was not due to the inherent weakness or strength as evidenced by

their bursting tension. Embrey in 1954 confirmed that it depends on factors other than the tensile strength. He found that the average pressure required to cause rupture of the membranes was 58.9 ± 11.5 mm. of mercury, and, according to Danforth, the membranes varied in thickness from 152 to 330 microns, but these figures did not help them to find any correlation between the thickness and the time of rupture of the membranes. So we are still in the dark as to the exact etiology though the following are some of the factors held responsible:

1. Increased intrauterine pressure, e.g. primiparity, hydramnios, multiple pregnancies and abnormal presentation. Norris, Mason and Schulre found an increased incidence of premature rupture of the membranes associated with the above conditions.
2. Badly fitting presenting part: Beck said that the presenting part acts like a ball valve and thus prevents intra-uterine pressure acting directly on the bag of membranes. When the presenting part does not fit the pelvis properly, intra-uterine pressure is directly transmitted to the bag of membranes and causes it to rupture prematurely.
3. Trauma, accidentally or purposely, is a very rare cause.
4. Undue adhesions of the membranes to the lower uterine segment which is not a constant finding.
5. Premature delivery: We do not know whether it is the cause or the effect of premature rupture of the membranes.
6. Diseases of the membranes.
7. Degenerative or inflammatory changes of the membranes.
8. According to Ballard, toxemia and syphilis appear to cause it.
9. Alberto thought metritis and its consequences can cause it.

Burnett reported fenestration of foetal membranes, hydrorrhea gravidarum, amniotic chorionic leakage and gravidatus exchorialis as the cause of premature rupture of membranes in some of the cases.

Lag Period

The period between premature rupture of the bag of waters and the onset of labour does not seem to depend on any definite factor. It was found that in the majority of patients labour sets in soon after the rupture of the membranes (Table I).

TABLE I

Onset of Labour following Rupture of Membranes

Time	No. of cases
0 - 6 hours	11
6 - 12 hours	6
12 - 24 hours	2
48 - 96 hours	1
24 - 48 hours	2
More than 4 days	3

It was less than 2 hours in 52.2% of cases and it was less than 24 hours in 87.5% of cases. In 6 patients, it was more than 24 hours, the longest interval being 10 days. In 35 patients, membranes ruptured after the onset of labour and in 25 they ruptured before the onset of labour. Out of 25 patients, 11 were primiparae and 14 were multiparae. Lag period is longer in multiparae

than in primiparae, average being 37 hours, 7 minutes in multiparae and 6 hours, 34 minutes in primiparae. The average lag period for primiparae in my series (6 hours, 34 minutes) is less than Calkin's (9.7 hours), the longest period being 3 days. Out of 14 multiparae, 9 had their lag period less than 24 hours, while out of the remaining 5, 2 had a lag period of 2 days and one each of 4 days, 7 days and 10 days. The average lag period for multiparae (37 hours, 7 minutes) is more than that of Calkins (21 hours, 14 minutes).

In the literature, cases are recorded where onset of labour was delayed for many weeks or months. Ruegg (1904) reported a lag period of as long as 120 days. Cozan (1952) reported a lag period of 116 days and Jurkowitz and Weinstein (1954) of 59 days. No correlation was found between the duration of lag period and the duration or the nature of labour. Why labour usually occurs soon after rupture of the membranes is not known. The following are some of the explanations put forward by Norris: (1) degeneration of decidua, (2) increased amount of oxygen in maternal blood, (3) excessive distension of uterus, (4) influence of various endocrinal substance, (5) pressure of presenting part, (6) trauma, and (7) foetal irritation.

Functions of Bag of Membranes and Liquor Amnii

In order to appreciate the effect of premature rupture of membranes on labour it is necessary to understand the functions of the bag of membranes and the liquor amnii in labour. The vitality of the bag of membranes in mechanics of labour was stressed as early as 16th century by Guillemeau. Beck stated that the intact membranes with their contained liquor amnii act as a fluid wedge which exerts a downward and lateral pressure on the isthmus and cervical canal and thus helps to dilate the cervix. When premature rupture of membranes occurs, the expansile fluid wedge is replaced by the head of the child which is a poor cervical dilator and causes greater trauma to the cervix and increased intensity of the first stage. But pressure of the head on the lower uterine segment frequently stimulates the uterus to contract more vigorously, so that the mechanical disadvantages of early rupture of membranes is offset by the increased strength and frequency of the expelling forces. Calkins has demonstrated that a shorter labour follows premature rupture of membranes, 10 hours in primiparae and 6 hours in multiparae. The hydrostatic theory of the bag of membranes was attacked in the 18th century and preservation of the membranes was urged only for protection of the baby, for prevention of infection and cervical laceration.

Ian Donald sums up the advantages of intact membranes as follows:

1. Maintenance of even hydrostatic pressure equally applied to the foetal surface.
2. During prolonged labour chances of infection are less.
3. No contraction ring.
4. Active retention of foetus as a result of generalised tonic contraction cannot occur.

5. Uterine rupture occurs rarely except in the presence of pathology of uterine wall.

6. Foetal asphyxia is less likely because intact membranes discourage a major degree of retraction at the placental site with its consequent reduction in its blood flow.

7. Rotation in cases of occipito-posterior is not discouraged.

8. Cord may present, but cannot prolapse.

9. Foetus is very unlikely to contract intranatal pneumonia.

10. The incidence of inertia and prolonged labour is not significantly altered by early rupture of the membranes but the effects of inertia are magnified, especially on foetal distress and maternal infection.

Thus, it will be seen that, though the role of leaking membranes in the causation of placental insufficiency, increasing the bad effects of abnormal labour, on foetal morbidity and mortality rate and maternal mortality rate is accepted by many, there is no unanimity of opinion regarding the functions of the bag of membranes in the dynamics of the 1st and 2nd stages of labour.

Effects of Premature Rupture of Membranes on Labour

The average duration of labour (1st + 2nd stage) following premature rupture of membranes was 9.706 hours while it was 8.607 hours in control cases. The difference of 1.099 hours is too small to be of any significance. The closer study of Table II, which compares the duration of labour among control cases and among the patients with premature rupture of the membranes, will reveal that there is a noticeable though small increase in the incidence of prolonged labour following premature rupture of the membranes.

TABLE II

Duration of labour	Control	Premature rupture of members
0 - 6 hours ..	21	12
6 - 12 hours ..	14	17
12 - 24 hours ..	4	7
24 - 48 hours ..	1	3

Table III compares the duration of labour in primiparae and multiparae.

The duration of labour is increased significantly by 4 hours, 55.73 minutes among the multiparae though in primiparae there is not much difference (22.8 minutes only).

Most of the deliveries occurred

TABLE III

Cases	Duration of labour (1st + 2nd)	
	Primiparae	Multiparae
Premature rupture of membranes ..	12 hrs 5.7 min	9 hrs. 31.73 min.
Control ..	11 hrs. 43.88 min.	4 hrs. 36.00 min.
Difference ..	0 hr. 22.08 min.	4 hrs. 55.73 min.

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within 24 hours of the rupture of membranes (Table IV).

TABLE IV

Time interval between the delivery and rupture of membranes	Number delivered
6 hours or less ..	7
6 to 12 hours ..	16
12 to 24 hours ..	9
24 to 48 hours ..	4
48 to 56 hours ..	2
7 days or more ..	2

Whether there is any correlation between the type of rupture of the bag and the duration of labour can be seen from Table V.

the average duration being 8 hours, 58.9 minutes. Calkins, Embrey, Schulre, Ballard, Mason and Tonkes have reported a definite shortening of the 1st stage of labour, the 2nd stage remaining unaffected. Keith was so definite about this that he has gone to the extent of advocating routine artificial rupture of membranes for elective induction of labour. Embrey found duration of labour varying with duration of the lag period: with 24 hours, incidence of prolonged labour is increased by half, and with 72 hours likelihood of prolongation is doubled. Though my series is too small to draw any conclusions, it can be seen from Table VI that the duration of labour is

TABLE V

Type of rupture	No. of normal deliveries	No. of abnormal deliveries	Duration of 1st + 2nd stage
Forewater ..	25	14	11 hours 24.64 min.
Hindwater ..	15	6	9 hours 56.00 min.
Difference ..			1 hour 28.64 min.

TABLE VI

Lag period	1st + 2nd stage	3rd stage
	hrs. - min.	hrs. - min.
0 - 6 hours ..	10 - 12	0 - 8
6 - 12 hours ..	10 - 40	0 - 8.25
12 - 20 hours ..	10 - 50	0 - 10
20 - 48 hours ..	6 - 38	0 - 8.5
7 - 10 days ..	4 - 32.5	0 - 7.5

There is a difference of 1 hour, 28.64 minutes, cases of the forewater rupture having longer duration than the hindwater rupture. Although it is a small difference, patients are longer in labour if forewater ruptures prematurely.

In cases where membranes ruptured before the onset of labour, there was no prolongation of labour, practically constant following a lag period of less than 20 hours and it is shorter with longer lag period.

The character of pains did not vary much among the group of patients with premature rupture of membranes and control patients. But Calkins has reported that pains are of better quality with premature rupture of membranes than they

would have been had membranes remained intact. Some of the authorities also think that longer the lag period, shorter is the first stage and vice versa.

The duration of the third stage of labour in the present series was not affected, average duration being 8.87 minutes in cases of premature rupture of membranes and 9.75 minutes in the control series. There were no increased complications or increased blood loss though Calkins mentions a definite increased blood loss and increased complications of third stage of labour. Only one patient had mild post-partum haemorrhage. Bishop thinks that labour is shorter even in abnormal presentation with premature rupture of membranes as compared to those abnormal cases where it ruptures late in labour. He found that in spite of a long lag period the labour was short.

Incidence of operative interference is increased in the present series, 4 caesarean sections and 5 forceps, giving an incidence of 6.6% and 8.33% respectively. Embrey, Alberto and Norris also think that the incidence of operative interference increases in these cases. But Bishop and Ballard do not agree and they think that cases requiring operations showed other abnormalities besides premature rupture of membranes, while Embrey believes it is increased considerably when lag period is 48 hours or more.

Effect of Premature Rupture of Membranes on Mother

There was one maternal death that could probably be attributable to premature rupture of the membranes. This patient was draining for 4 days before labour set in. No other abnormality was detected. She had intrauterine infection and liquor was foul smelling. She had very high temperature on the 4th day and died after a normal delivery, labour lasting 3 hours and 10 minutes only.

Seven patients had fever for 2 days, giving an incidence of 11.66% morbidity. Two of these had normal deliveries, 3 lower segment caesarean sections and 2 forceps deliveries. Only one patient had draining for 5 days and one for 10 days before the onset of labour.

In control series there was no death, and only two patients had fever for 2 days, giving a figure of 3.33% morbidity.

It is found that chances of intrauterine infection increase if labour is induced medically or surgically compared to those cases where no artificial interference is done. The cases where first stage was prolonged were more prone to infection compared to the patients who had long latent period and short first stage of labour according to Morton and Wolf. Infection can develop even if no vaginal examination has been done as the bacteria from vulva might multiply in the capillary layer of fluid in vagina and readily invade open amniotic sac. Maternal morbidity is increased by 20% according to Calkins. He considered that if infection does not occur within 72 hours after rupture of membranes, it is less likely to occur afterwards.

Woltz advocates penicillin routinely in all cases of premature rupture of the membranes and he

observed that staphylococci were grown from the heart's blood in cases of premature rupture of membranes in patients who died of septicemia and who did not have any antibiotics. But Roth (1954) states that infection is not more common without the use of antibiotics, and administration of antibiotics during the lag period does not reduce the incidence of infection, while they have a beneficial effect during labour and puerperium.

Effect of Premature Rupture of Membranes on Babies

Out of 62 babies in the present series, 7 were stillborn, but only in two cases the intrauterine death was probably attributable to prolonged draining, with 21 hours and 4 days as lag period, which has given a high incidence of 11.29% foetal mortality. Of the other five, one death was due to a tight knot in the cord. One baby had exomphalos, two deaths were due to cord prolapse and one baby was lost because of the after-coming head being arrested. In the present series, 12 babies were asphyxiated while there was no death and no case of foetal asphyxia in control series. One baby had fever for 3 days after lower segment caesarean section, done 18 hours after draining, while in the control series there was no case showing infection.

Votta, Munro Kerr and Tonkes have reported increased infant mortality in cases where membranes ruptured prematurely. Mcphail and Hall (1941) showed that there was an increased incidence of mild and serious apnoea, and in neonatal deaths. According to them, the incidence of severe apnoea in premature rupture of membranes is 16% (5.9% in normal cases). But Ballard and Bishop disagree with them. Mayer has noted feeble heart sounds in delayed rupture of membranes, attributing it to foetal compression due to persistent bag of membranes. Embrey goes to the extent of saying that foetal loss is actually diminished when labour sets in within one hour after rupture of membranes but he agrees that it is doubled when lag period is over 48 hours and trebled when it is 96 hours. Wolf thinks that the effect on the foetus is unfavourable when patients are treated actively after premature rupture of membranes. Foetal morbidity is high because of intrauterine pneumonia, septicemia due to placental infection, cord inflammation and infection of amniotic sac.

The causes of intrauterine death in cases of prolonged draining are:

(i) Placental insufficiency due to retraction of uterus directly on placental site which was the cause of stillbirth in one case.

(ii) Uterine infection: Even though the mother might escape infection the child might succumb a few days later. Holland and others thought that it became infected by swallowing contaminated amniotic fluid, but Slemons showed that in a certain proportion of cases the bacteria make their way through the amnion, covering the foetal surface of the placenta and, after invading the large vessels which lie underneath it, gain access to foetal circulation and give rise to general septicemia. He designed it as placental bacteremia and held that it plays a part in late foetal mortality.

One-third of the foetal mortality in premature rupture of the membranes is due to uterine infection.

(iii) Cord prolapse is more common but this point is disputable. Bishop says it is not a frequent accompaniment, contrary to widespread opinion, because when the presenting part is able to descend far enough to exert pressure upon the forewaters to produce rupture of the membranes, it will not be so poorly adapted to inlet as to produce cord prolapse in these cases and there were no cases of cord prolapse in his series. In my series, there are two cases of cord prolapse out of 60, i.e. an incidence of 3.3%.

Management of Premature Rupture of Membranes

The patient should be hospitalised with rest in bed. Antibiotics are given only to some cases where it is necessary. Frequent vaginal examinations should be avoided and a sterile vulvar pad given. Foetal heart sounds should be observed every half an hour, and mother should be watched for rise of pulse and temperature, dehydration, exhaustion, etc. Reassurance to the patient, procuring good sleep and maintaining nourishment are important during the lag period. Labour is not allowed to be unduly prolonged.

When the membranes rupture before the onset of labour, there is again divergence of opinion as to whether active measures like medical induction or surgical measures like metreuynter or Voorhee's bag should be used. But now-a-days surgical methods are out of fashion. We have tried castor oil followed by warm enema in two cases without result. Of the active measures, pitocin drip, 5 units of pitocin to a pint of 5% glucose solution, given very slowly and carefully, seems to be a safe and successful measure as advised by Ryan.

Summary

1. In order to assess the effects of premature rupture of membranes on labour, 60 cases, where membranes ruptured prematurely at full-term, were studied along with 40 control cases and the results have been carefully studied and evaluated.

2. Twenty cases were abnormal —4 breech, 2 twins, one hydraminos, 4 lower segment caesarean section, 5 low mid-cavity forceps, one maternal death, one normal delivery with complete perineal tear and 2 cord prolapse.

3. No apparent etiological factor became evident.

4. In 87.5% of cases the lag period was less than 24 hours and in 52.2% of cases it was less than 2 hours. The average duration of lag period for primipara was 6 hours 34 minutes, and for multipara 37 hours 7 minutes. No relation was found between the duration of lag period and the duration and the nature of the labour.

5. The duration of labour was prolonged in cases where membranes ruptured prematurely. The mean duration being 9.706 hours as compared to 8.607 hours in control cases.

6. No prolongation of labour occurred where membranes ruptured before the onset of labours, mean duration being 8 hours 58.9 minutes.

7. Incidence of artificial interference is increased — 6.6% caesarean section and 8.33% low mid-cavity forceps rate.

8. No effect was noticed on duration of 3rd stage and there was no increased incidence of post-partum haemorrhage.

9. Incidence of asphyxia was high —12 babies had it out of 53.

10. There was one maternal death and 7 stillbirths, out of which two were probably attributable to premature rupture of the membranes.

11. There was an increased incidence of puerperal sepsis, 11.66% morbidity in cases where membranes ruptured prematurely compared to 3.33% morbidity in control cases.

References

1. Atkins H. T.: Am. J. Obst. & Gyn.; 58, 565, 1948.
2. Bainbridge M. N., Nixon W. C. W., and Smythe C. N.: Brit. J. Obst. & Gyn.; 65, 189, 1958.
3. Ballard Margaret B.: Am. J. Obst. & Gyn.; 32, 905, 1936.
4. Beck Alfred: Obstetrical Practice; 5th Ed., William Wilkins & Co., Baltimore, 1951.
5. Bishop Edward H.: Am. J. of Obst. & Gyn.; 48, 45, 1944.
6. Biskind J. I., and Biskind L. H.: Am. J. Obst. & Gyn.; 73, 750, 1957.
7. Bock A.: Am. J. Obst. & Gyn.; 32, 905, 1936.
8. Brown: Post-graduate Obstetrics & Gynaecology; 2nd Ed., Butterworth & Co., London, 1955.
9. Burnett C. W. F.: Brit. J. Obst. & Gyn.; 64, 79, 1957.
10. Calkins L. A.: Am. J. Obst. & Gyn.; 64, 871, 1952.
11. Coatz Alberto S.: Am. J. Obst. & Gyn.; 32, 905, 1936.
12. Cootz A. S.: Am. J. Obst. & Gyn.; 46, 611, 1943.
13. Cozan Henry A.: Am. J. Obst. & Gyn.; 63, 920, 1952.
14. Danforth D. N., Melin T. W., and States M. N.: Am. J. Obst. & Gyn.; 65, 480, 1956.
15. Donald Ian: Practical Obstetric Problems; 2nd Ed., Lloyds & Luke, London, p. 372, 1959.
16. Eichner E.: J. Obst. & Gyn. Brit. Emp.; 61, 536, 1954.
17. Embrey Mortyn P.: J. Obst. & Gyn. Brit. Emp.; 60, 37, 1953 and 61, 793, 1954.
18. Endress P.: Am. J. Obst. & Gyn.; 35, 735, 1938.
19. Finlaison F. H.: J. Obst. & Gyn. Brit. Emp.; 57, 423, 1950.
20. Gross B.: J. Obst. & Gyn. Brit. Emp.; 65, 489, 1958.
21. Gross Rudolf: Am. J. Obst. & Gyn.; 43, 915, 1942.
22. Hanley Banard J.: Am. J. Obst. & Gyn.; 64, 221, 1952.
23. Kautsky J.: J. Obst. & Gyn. Brit. Emp.; 62, 659, 1955.
24. King Arthur G.: Am. J. Obst. & Gyn.; 32, 301, 1936.
25. Knox I. C. and Horner J. K.: J. Obst. & Gyn. Brit. Emp.; 57, 833, 1950.
26. Lyandres I. M.: J. Obst. & Gyn. Brit. Emp.; 56, 303, 1949.
27. Maizeles Gerald: J. Obst. & Gyn. Brit. Emp.; 47, 237, 1940.
28. Mason Lyman W.: Am. J. Obst. & Gyn.; 26, 394, 1933.
29. Mayer A.: Am. J. Obst. & Gyn.; 17, 327, 1927.
30. Morton J. H., Peabody C. S., Newdrop J. and Adair Fred L.: Am. J. Obst. & Gyn.; 43, 422, 1942.

31. Munro Kerr: Operative Obstetrics; 6th Ed., Bailliere, Tindal & Cox, London, 1956.
32. Norris Charles C.: J. Obst. & Gyn. Brit. Emp.; 32, 905, 1936.
33. Pattison Stanley D.: Am. J. Obst. & Gyn.; 69, 233, 1955.
34. Power H. A.: Am. J. Obst. & Gyn.; 57, 608, 1949.
35. Ryan T. J.: Brit. J. Obst. & Gyn.; 65, 71, 1958.
36. Schulre Margaret: Am. J. Obst. & Gyn; 17, 20, 1929.
37. Schumann: Am. J. Obst. & Gyn.; 62, 633, 1956.
38. Short C. R.: Am. J. Obst. & Gyn.; 43, 915, 1942.
39. Sunde A.: Am. J. Obst. & Gyn.; 35, 734, 1938.
40. Tonkes E.: Am. J. Obst. & Gyn.; 55, 906, 1948.
41. Votta R. A.: J. Obst. & Gyn. Brit. Emp.; 62, 313, 1955.
42. Wickmann S. E.: Am. J. Obst. & Gyn.; 35, 757, 1938.
43. William's Obstetrics: Eastman, 5th & 11th Ed., Appleton, New York, 1924 and 1956.
44. Winter F. W.: Am. J. Obst. & Gyn.; 35, 905, 1936.
45. Wolf W.: J. Obst. & Gyn.; 56, 142, 1948.
46. Woltz J. H. E. and Starkak T. S : J. Obst. & Gyn. Brit. Emp.; 56, 143, 1946.