The Linacre Quarterly

Volume 10 | Number 1 Article 3

January 1942

Ectopic Gestation: Medical Aspects

Elmer A. Schlueter

ECTOPIC GESTATION

NOTE: These two articles, one on the medical aspects, and the other on the moral aspects of Ectopic Gestation, were prepared for the "Institutum Divi Thomae" at Cincinnati, Ohio. They are reproduced here from the *Ecclesiastical Review*. The two articles should be read together, as it will be clear from the reading that one well-reasoned and weighty moral opinion rejects at times what surgical practice recommends in dealing with ectopics. In concrete and specific cases the physician should always consult his clerical adviser. There are many angles to these problems which only the priest, under the direction of the moralist, can handle.

Medical Aspects

BY ELMER A. SCHLUETER, M.D.

CINCINNATI, OHIO

Ectopic gestation, that condition in which the fertilized ovum fails to reach and to become implanted in the uterus, but remains and continues to grow in the Fallopian tube, has long been discussed by physicians and moralists from the standpoint of treatment.

The moral aspects will not be discussed, since they are outside the particular field of physicians, but will be left to the study and the decision of moralists. The physiological and the pathologic-physiological factors, however, are to be presented here for the enlightenment, guidance, and consideration of moralists, who necessarily consult medical opinion in this matter of ectopic gestation before formulating or establishing rules of procedure for its treatment.

It must be remembered that:

1. The condition is neither common nor rare, occurring approximately as often as twinning.

2. The diagnosis is not simple but is frequently confounded with other abdominal disorders. In early cases it may be quite difficult to establish a diagnosis, as many factors, among which are mechanical, anatomical, pathological, and endocrine changes, are involved in the cause of the condition.

3. The treatment is not uniform in all details. Surgical removal of the affected tube is the only generally accepted one.

Some moralists approve surgical removal of the affected tube, basing their approval on the opinion of physicians that ectopic gestation is the direct result of disease of the affected tube, and that this disease may be the result of previous inflammation, malformations or developmental anomalies, mechanical interference or peritoneal adhesions. They say further that, although conception occurs in the tube, pregnancy is normally carried to term only in the uterus, and, therefore, pregnancy outside the uterus must necessarily be considered the result of previous pathologic changes in the tube, obstructing the passage of the fertilized ovum from the tube into the uterus. After a careful study of

all these factors, certain conclusions are drawn in order to establish a basis of treatment.

Sixty-six cases, occurring between January, 1935, and January, 1941, were carefully reviewed. These cases were admitted to two hospitals in which were delivered, during the same period, approximately 10,000 full-term babies. The frequency of occurrence is, therefore, about six per thousand full-term uterine pregnancies.

Anatomy.

At this point it is well to review the anatomy and physiology of the Fallopian tubes. The Fallopian tube, about 4½ inches long, begins within the wall of the cornu of the uterus (intramural portion or isthmus), continues throughout the edge of the broad ligament which supports it (ampullary portion), and ends as a funnel-shaped organ (infundibulum) having a fringed (fimbriated) end. This end hangs free in the abdominal cavity and is capable of enclosing the ovary in order to receive the ovum, when the follicle containing it ruptures.

Histology.

Histologically, the tube is made up of an internal mucous membrane, composed of ciliated epithelium. External to this is a layer of connective tissue, binding the mucous membrane to a layer of circular muscle and longitudinal muscle. A second layer of connective tissue binds the muscular layer to an outer peritoneal covering, which is continuous with that covering the uterus.

Physiology.

The mucous membrane is markedly convoluted in the ampulla (middle portion) and the infundibulum (distal portion). These convolutions may allow retention of the ovum until fertilization can take place. The ciliated epithelium, together with the peristaltic action of the musculature, moves the ovum along the tube toward the uterus. The male element, the spermatozoa, moves by its own power through the cervix into and through the uterine cavity and through the tube, where fertilization occurs. When fertilization occurs, movement towards the fimbria stops; the peristaltic activity and the ciliary action then propel the fertilized ovum toward and into the uterine cavity, where provision has already been made for its reception and nourishment. Before reaching the uterus, the fertilized ovum must pass through the isthmus, the narrowest portion of the tube.

Pathologic Physiology.

Obstruction of the passage of the fertilized ovum may occur either in the convoluted portions of the tube (ampulla and infundibulum), or in the isthmus, causing retention and ectopic or tubal pregnancy. Examination of the 66 cases studied, in which the preoperative or postoperative diagnosis was ectopic gestation, showed that ectopic gestation oc-

curred in the middle (ampulary) portion 8 times, in the distal (infundibulary) portion 18 times, and in the proximal (in or near the isthmus) portion 8 times (including two cases occurring in the cornu of the uterus). It is apparent from this study that pregnancy more commonly occurs in the convoluted portion; other observers have found similar incidence. These same cases show that ectopic gestation occurs about as frequently in the right as in the left tube—31 in the right and 29 in the left tube.

The uterine hyperplasia and decidual reaction occurring during ectopic gestation are identical with that found in normal uterine gestation, as long as the embryo is alive. With the death of the embryo, however, the superficial, compact portion and, sometimes, part of the spongy layer are cast off entirely, but usually in small particles.

Etiologic Factors.

It has been stated repeatedly that pregnancy in the tube would not occur unless some pathologic change has previously taken place in the tube. These pathologic changes may be the result of previous inflammations, congenital anomalies, mechanical effects, postural changes, previous pregnancy, menstrual irregularities, abortions, miscarriages, normal menstrual back flow, and appendicitis.

The Mayo Clinic reports that chronic inflammatory changes involve both tubes about equally; the report includes in this group salpingitis following artificial abortion, gonorrhea, and the use of intra-uterine contraceptives.

In Helsingfors, Finland, from 1901 to 1930, ectopic pregnancy increased 2½ times in relation to mature women, and 4 times in relation to conceptions. Abortions increased 6 times in relation to mature women, and 11 times in relation to conceptions. Criminal abortions during this time increased to 92% of all abortions. Prematurity doubled, especially among unmarried women.

The absolute and the relative increases of gonorrhea were the most important factors in the increase of extra-uterine pregnancy; the number of infected persons increased from 5.4 to 11.6 per 1,000. Between 1920–30, adnexal inflammation, chiefly gonorrhea, was present in 76%; abortion preceded ectopic gestation in 33½%; puerperal infection after abortion and prematurity were lesser, but important, factors. Sterility of about 5 years' duration after abortion was present before the ectopic gestation occurred. Frequently appendicitis, operated and non-operated, had occurred previously.

The most troublesome inflammatory changes secondary to gonorrhea occur chiefly in the tubes, causing abscess, followed by occlusion or partial obstruction.

Although gonorrhea is one of the commonest causes of tubal inflammation, there are many others.

During menstruation, it is not unusual that some of the menstrual discharge enters the tube, as a result of back pressure within the uterus, and causes irritation to the tubal mucosa, which may result in some mild inflammatory change in the mucosa. During the menstrual cycle occurs also a normal engorgement of the tube, contributing to, or furthering, the inflammatory process set up by the irritation of the menstrual fluid. During a previous uterine pregnancy the tubal wall and mucosa show physiologic engorgement, with thickening of the mucosa and enlargement of the folds of the convolutions; some of these changes may persist after termination of the normal pregnancy. The same changes may not disappear after abortion, miscarriage, or prematurity.

Atrophic changes may occur at any time, but more commonly toward the end of the child-bearing period. These changes are characterized by the disappearance of cilia from the mucous membrane, the diminution in size of the tube and lumen, and the shrinking of the mucosal folds.

Acute inflammation is characterized by redness, increased local temperature, pain, and swelling. Accompanying these are interstitial, surface or mucosal, hemorrhagic, serous or purulent discharges or exudates, which may cause occlusion of the lumen or obstruction of the tube. If the inflammation persists, progressive destruction of the mucosa and invasion of the connective tissue and musculature may occur, with resulting scarring, torsions, or adhesions. When the inflammation subsides, more or less permanent damage may have occurred to bring about the same results—occlusion, or obstruction of the lumen. Low-grade continuous infection (chronic) produces retrograde changes in the tissues with similar results.

Disease of contiguous structures, particularly the ovaries, the uterus, the appendix, and the bowel, play some part by exercising an influence on the tube because of proximity or pressure: proximity, allowing infection to spread to the tube, setting up an inflammatory reaction, or including the tube and massing; pressure, causing kinks, or occlusion of the lumen of the tube.

Tubal insufflation, the procedure intended to determine the patency of the tubes, may readily set up inflammatory reactions by breaking up old scars or adhesions and, although opening the tube, promote new scars conducive to ectopic gestation.

Of the congenital anomalies, persistence of the spiral twists of the embryo is the commonest developmental change. Diverticula occasionally are found; accessory tubes or rudimentary tubes, rarely.

In asthenic states, atony of the musculature, together with sluggishness of the ciliated epithelium, may delay passage of the fertilized ovum until it is too large to pass

through the tube into the uterus.

Endocrine deficiency, particularly thyroid, pituitary, and ovarian hormonal deficiency, undoubtedly exerts some influence on the implantation and growth of the ovum in the tube as well as in the uterus.

Adhesions between contiguous structures and the tube may cause occlusion or obstruction of the tube.

There was no indication of postural effects, although the Mayo Clinic report notes that sleeping on the right side is a possible factor.

In these 66 cases, gonorrhea was never a factor; chronic inflammation was noted in 11 cases; previous salpingitis in 1; previous abortion in 2; previous miscarriage in 8; appendicitis in 6; menstrual disorders in 13; previous pregnancy in 24; cystic ovaries in 8; uterine fibroid in 2; tortuosity in 2; tubal insufflation in 1, and chronic disease in 2. A classification of the etiologic factors is presented in Table A.

TABLE A

Etiologic Factors

1. Anatomical Abnormalities		
Type	No.	
Patency of tube	13	
Occlusion of tube	7	
Adhesions of tube	15	
Tortuosity of tube	1	
2. Chronic Infections		
Type	No.	
Cystic ovaries	8	
Salpingitis-acute	1	
Previous appendicitis	6	(Coincidental 5)
Chronic disease	2	
Chronic inflammation (salpingitis)	11	(With 9 in opposite tube)
3. Mechanical Agencies		
Type	No.	
Insufflation	1	
4. Previous Pregnancy	24	
5. Menstrual Irregularities		
Type	No.	
Normal	6	
Irregular	10	
Metrorrhagia	3	
6. Interrupted Pregnancy		
Type	No.	
Miscarriage	8	
Abortion	2	
7. Previous Attacks	3	

The age limits are those of the child-bearing period; in this series 37, or 56%, occurred between 21–30; 21, or 31.8%, between 31–40; 6, or 10%, 41 and over; and 2 with no age given. Davidson reports 76% between 22 and 36 years.

The ectopic gestation in 24 cases occurred from 1 to 17½ years after the last normal uterine pregnancy; usually, however, it was from 3 to 5 years.

Pathology.

The pathologic picture of the ectopic tube is one of acute inflammatory reaction. It begins with engorgement of the entire wall, causing swelling. Very quickly the mucosal surface and the musculature are invaded by trophoblasts and the growing chorionic villi to provide nourishment for the embryo. This condition causes increased swelling, interstitial hemorrhage, and pressure, with degeneration of the supporting tissue, necrosis, and often rupture.

At times the ectopic mass is expelled from the tube (tubal abortion) into the abdominal cavity, because the mass fails to remain attached to the tubal wall, owing to endocrine insufficiency or as the result of tubal swelling and hemorrhage behind the ectopic mass.

Of the developmental anomalies, tortuosity was described only once. Of the chronic inflammatory changes, adhesions were found in 15 cases and cystic ovaries in 8. Physiologic changes were noted in 6 cases where rupture had occurred early, chronic salpingitis in 1, and accompanying appendicitis in 2.

A summary of the pathologic findings is given in the following Table B.

TABLE B

Pathology

Type	No. of Cases
Free hemorrhage	37
Hemorrhage in tissue	24
Rupture	28
Intact	32
Patent	13
Occluded	7
Adhesions	15
Tortuosity	1
Cystic ovaries	8
Uterine hypertrophy	6
Swelling and hypertrophy of tube	30
Necrosis	2
Degenerative changes	6
Chronic inflammation (salpingitis)	11
Invasion of wall	8
Inflammatory changes (incidental)	31
Normal physiologic changes	6

Hemorrhage may be a very serious problem, or it may be only hemorrhagic infiltration of the walls of the tube. Free hemorrhage may be extensive, often amounting to 3,000 cc. of blood free in the abdominal cavity. Free,

serious hemorrhage may occur with or without rupture of the tube, but more commonly with rupture. If rupture of the tube occurs, hemorrhage may seriously endanger the life of the patient and most rarely does the embryo survive. In these cases the tube ruptures and the embryo becomes attached to a neighboring structure, *i.e.*, the bowel, the peritoneum, the uterus, the ovary. There have been reports of the living foetus being removed surgically at term, but in most cases of the above attachments death ensues and the foetus forms a calcified mass.

As a general rule, the opposite tube undergoes normal physiologic changes characterized by engorgement and swelling, often simulating to the untrained observer an acute inflammatory process. However, when chronic inflammation is present, it is found with equal frequency in both tubes.

A summary of the condition of the opposite tube follows.

TABLE C

Pathologic Changes in the Opposite Tube

Type	No.
Normal	3
Previously removed	3
Ruptured	1
Patent	1
Occluded	1
Adhesions	7
Tortuosity	1
Chronic inflammation	8
Acute inflammatory changes	5
Physiologic changes	2

Diagnosis.

The diagnosis of chronic ectopic pregnancy is based on prolonged bleeding, little discomfort, but more or less constant pain in either quadrant and often on the opposite side, rectal irritation, nausea, and vomiting. Davidow, of the Michigan State Medical Society, reports that 74% were diagnosed tube-ovarian or cul-de-sac abscesses; in the same report, 150 of 218 cases showed evidence of tubal inflammation, from acute to hydrosalpinx.

The sedimentation rate offers an aid, in this, that it is slower than in acute inflammatory processes but more rapid than the average normal, except with secondary infection. The white count shows a total rise, but there is no change in the shift; the red count shows a secondary anemia.

The diagnosis of acute ectopic pregnancy is not always easy. The history of a sudden development of pain in the lower abdomen, of a delayed or missed period after a period of sterility, accompanied by vaginal bleeding and signs of shock, together with a tender cervix, are salient points in the diagnosis. In this series, 11 had no menstrual delay; 15 missed 1 cycle; 8, two cycles; 10, three cycles; 1, four cycles; 2, six cycles; 80% showed signs of shock; all had low abdominal pain and tenderness, and only 50% reported pelvic mass and tenderness.

Further aids in the diagnosis may be obtained from the white blood count and sedimentation

rate. Pregnancy tests are also of value. A negative Ascheim-Zondek test simply excludes a living tubal pregnancy, but does not rule out a dead tubal pregnancy. A positive test indicates a living foetus which may be uterine or extra-uterine. The uterus is often enlarged, adding to the difficulty of diagnosis and simulating early threatened abortion.

Vaginal bleeding occurs with destruction of the uterine decidua, which does not begin till death of the foetus; therefore, the non-bleeding cases are the more dangerous, in this, that the foetus in the tube is growing and may cause rupture of the tube at any time, with serious internal hemorrhage.

Microscopic study of the ovary, when available, reveals the presence of trophoblasts (foetal cells) in varying stages; *i.e.*, actively growing, not living, degenerated, or necrotic. Section of the ovary further shows varying traces of corpus luteum of pregnancy, depending on the state of the trophoblasts. That there is a definite relationship between corpus luteum of pregnancy and trophoblasts seems apparent, in this, that with dead trophoblasts there was an absence of corpus luteum, but with a living foetus and living trophoblasts there was good corpus luteum of pregnancy. It would appear, therefore, that injury to the pregnancy causes death of the foetus; death of the foetus is followed by degeneration of the corpus luteum; after the degeneration of the corpus luteum, the trophoblasts may continue to grow for some time. This indicates a hormonal influence of the foetus on the corpus luteum. It is known that in uterine pregnancy the corpus luteum hormone supports continued development of the foetus, but its absence in early pregnancy causes miscarriage. It seems also that the corpus luteum is dependent on the foetus. Borner found that, when the implantation of the ovum is disturbed, degeneration of the corpus luteum begins.

Sampson noted that only the cases of ectopic pregnancy which had intact uterine decidua did not have vaginal bleeding; the action of the tubal pregnancy, through the corpus luteum, on the uterine decidua is indirect, uterine bleeding being the first sign of degeneration of the corpus luteum caused by the death of the embryo. When the uterus was removed in cases of intact ectopic pregnancy, the uterine decidua was uninjured and the corpus luteum was in good condition. When the foetus was dead, the corpus luteum was absent or degenerated and the uterine decidua thrown off. Uterine bleeding does not occur until some time after the death of the foetus. Internal hemorrhage, however, may and often does precede the uterine bleeding. When this internal hemorrhage is not recognized promptly, shock may be profound.

In the series under consideration, it is of interest that 37 had vaginal (uterine) bleeding and 6 were reported to have uterine hypertrophy. The bleeding, for

the most part, was from a few hours' to several weeks' duration. Some of the most acute cases were not bleeding from the vagina. Vaginal (uterine) bleeding occurred in 26 of the ruptured ectopics, and in 11 of the intact ectopics. In 44 cases the pathologic report noted the presence of chorionic villi, but did not define the state; while in 6 cases the pathologic report noted degenerated chorionic villi.

Differential diagnosis must rule out other intra-abdominal lesions; that this is not a simple matter will be seen from an examination of the diagnoses made in the cases studied (Table D). Although all were based on intra-abdominal pathology, it shows that 18 different diagnoses were made and that the chief condition found was ruptured ectopic or tubal pregnancy; one case showed rupture of the tube opposite the site of the ectopic.

TABLE D

Preoperative Diagnosis		*Postoperative Diagnosis*
Type	No.	
Ruptured ectopic	23	10 Ruptured; 13 Intact
Acute appendicitis	4	3 Ruptured; 1 chronic salpingitis with appendicitis
Cystic or ruptured ectopic	3	2 Ruptured; 1 Intact
Ectopic pregnancy	16	3 Ruptured; 10 Intact, 2 not operated, 1 uterine pregnancy
Ectopic or salpingitis	3	2 Ruptured; 1 Intact
Ectopic or appendicitis	1	1 Ruptured with acute appendicitis
Tubal abortion	3	1 Ruptured; 2 Intact
Abdominal mass	2	2 Ruptured
Ovarian cyst	1	1 Ruptured
Ectopic or tubal abortion	2	1 Ruptured; 1 Intact
Abdominal pregnancy	1	
Exploratory laparotomy	1	1 Ruptured
Chronic endometris or ectopic	1	1 Intact
Bilateral ectopic	1	1 Ectopic with rupture of opposite tube
Ovarian pregnancy	1	1 Ruptured ectopic
Ectopic and endocerviatis	1	1 Intact ectopic
Oophoritis and cholecystitis	1	1 Ruptured ectopic
Hypertrophic endometritis	1	1 Intact ectopic

Treatment.

There is only one treatment of ectopic pregnancy — surgical removal of the affected tube. Surgical opinion is unanimous. Surgeons insist on the removal of the tube as soon as the diagnosis is made or the ectopic tube found. This surgical opinion is founded on the assumption of these factors: The tube is definitely pathologic by reason of acute inflammation incidental to the ectopic gestation; it is likely that the tube was pathologic prior to the ectopic gestation; the mortality rate is

low, especially before rupture, and not high at the time of rupture or after rupture, the removal of the tube and ovary does not greatly lessen the likelihood of subsequent pregnancy.

It is probable that the foetus is dead by the time vaginal bleeding occurs; but that absence of bleeding does not necessarily imply intact ectopic pregnancy. The removal of both tubes and ovaries is not justified in the child-bearing period; the probability of a recurrence is slight. It is probable that it may be seriously doubted whether or not there is sufficient evidence to assume that the tube has been the seat of previous disease.

It is the opinion of most pathologists that the acute inflammatory process occasioned by the presence of an ectopic gestation so distorts the previous structure or previous structural changes that it is impossible to observe any old pathology. The Mayo Clinic report gives only 13% chronic tubal inflammation, as against 16% in this series.

That the tube is the seat of acute pathology is unquestioned; ample evidence of swelling, cellular reaction, hemorrhage, hemorrhagic infiltration, and necrosis is present in nearly all cases, except possibly those of tubal abortion. Davidow reports 130 out of 218 cases showing evidence of tubal inflammation; in this series all showed evidence of acute inflammation.

Prognosis.

The mortality rate is low, from 3% to 5%. In this series only 1 death occurred in 66 cases—less than 2%.

The likelihood of subsequent pregnancy after removal of the tube is not reduced, certainly not by 50%. Of 84 patients of 100 ectopic reported by the Mayo Clinic, 31 became pregnant, 28 (33%) intra-uterine and 3 ectopic (36%). These 28 patients had 47 intra-uterine pregnancies; 32 full terms, 3 still-births (syphilis in 2), 4 premature, and 10 miscarriages. According to the same report 21 pregnancies occurred in 14 patients having both ovaries after removal of the tube, and 11 pregnancies in 7 patients having one ovary after removal of the tube and ovary.

The removal of both tubes, or of both tubes and ovaries, at the time of operation for ectopic gestation in one tube is not justifiable. The severe hemorrhage and shock, or the acuteness of the disorder, precludes, or should preclude, more than immediately necessary surgical procedures. The opposite tube, or tube and ovary, commonly shows physiologic reaction owing to the ectopic; this condition invariably disappears after the removal of the ectopic, and the tube returns to normal.

The probabilities of recurrence are generally conceded to be remote, not more than 5%. In this series only three had had previous ectopic pregnancies, which is about 4.5%; about 43% have normal uterine pregnancies.

Although there have been recent reports of term delivery of unruptured ectopics with a living normal child (Richards), this is most unusual.

If there is no operation and the ruptured or ectopic tube remains undisturbed, the foetal membranes may become attached to the peritoneum of the abdominal wall or the peritoneal covering of the abdominal organs, thereby giving rise to an intra-abdominal pregnancy. One of two things then may take place: either the foetus continues to develop to term, when it must be removed by surgical interference, or, after some time, the foetus dies and becomes a foetal cyst or calcified mass. These latter conditions commonly cause considerable discomfort, but quite often are asymptomatic. Gounet reports a patient carrying to full term a uterine pregnancy ten years after discovery of and without disturbing an extra-uterine calcified foetus. Schuhl reports 89 uterine pregnancies, 13 abortions, 45 premature, in the presence of retained extra-uterine foetal cysts in 56 women. The mortality was 23% in 39 cases. Funck-Brentam, Demelin, and Klein report that pregnancy in cases of retained foetal cysts may evolve normally.

However, these are the exception, not the rule; certainly, it is not recommended that ectopic pregnancy be not treated, hoping for the development of a foetal cyst.

Summary.

In conclusion, it is certain, from careful review of the literature and a study of a number of cases, that all tubal pregnancies show acute incidental inflammation of the tube; few show demonstrable chronic inflammatory changes. Occlusion or patency of the tube obviously is not a critical etiologic factor, but rather a result of ectopic gestation.

One of the certain signs of a dead foetus is vaginal bleeding. A negative Ascheim-Zondeck shows death of foetal cells; a positive, living cells. Lack of staining (spotting) may be a danger sign, rupture may occur at any time. The foetus dies first, degeneration of corpus luteum occurs next, causing the casting off of uterine decidua, with uterine bleeding. Surgery offers the only satisfactory treatment.

REFERENCES

1. A. M. Richards, *British Medical Journal,* 3565:807, May 4, 1929.
2. D. M. Davidow, *Michigan State Medical Society,* 34:376, June, 1935.
3. Gounet, *Bull. de la Soc. de Gyn. et d'Obstet.,* 28:438, June, 1939.
4. *Mayo Clinic Proceedings,* Volume XII, Nos. 41–648.
5. J. Haffner, *Nordisk Medicin-Gothenburg,* 7:1488, Sept., 1940.
6. Emil Novak, et al., *Am. Jour. Obstet. & Gyn.,* Vol. IX, No. 3, 295, March, 1925.
7. Rubin, I. C., *Am. Jour. Obstet. & Gyn.,* 28:698, November, 1934.

8. Masson, J. C. & Simon, H., *Surg. Clinic, North Amer.*, 7:1601, December, 1927.

9. Hodgson, J. C., *The Lancet*, 1:565, March, 1932.

10. Schiowa, B. P., Garfunkel & Shapiro, J., *Monatschr. fur Gerburtsh. u. Gynak.*, 92:292, 1932.

11. Jones, A. P., *Amer. Jour. Surg.*, 20: 633, June, 1933.

12. Harkness, R. C., *Brit. Med. Jour.*, 2:245, 1920.

13. Hingston, C. A. F. and Mudaliar, A. L., *Indian Med. Gaz.*, 58:528, 1923.

14. P. B. Blond, *Am. Jour. Obst. & Gyn.*, 40:271, August, 1940.

15. Apalajahti, A., *Acta. Soc. Med. Fenn —Duodecim*, B, 16:1–190, 1931.

16. Tenny, B., *New Eng. Jour. of Med.*, 214:773, April 16, 1936.

ECTOPIC GESTATION—II

Moral Aspects

By RIGHT REVEREND MONSIGNOR JAMES W. O'BRIEN, S.T.D.

Norwood, Ohio

There are two decrees of Holy Office referring directly to the moral aspects of the case of ectopic gestation. The first, issued May 4, 1898, states that it is licit to perform the operation called laparotomy for the purpose of removing the ectopic foetus, provided that serious effort is made to save the lives of both the mother and the child. The text of this decree and the text of the others referred to in this article are appended. The obvious sense of this decree is that the operation is illicit unless the foetus is viable, because otherwise there could be no serious effort to save its life. Any doubt as to its meaning, however, is taken away by the subsequent decree of March 5, 1902, which declares that the ejection of the immature foetus is always illicit. This decree further lays down certain conditions for the licitness of hastening the birth of even the viable foetus. This decree calls attention to the previous one and states that its own regulations are in conformity with it.

Besides these two decrees directly concerned with the ectopic foetus, there are two others that have a bearing upon it, even if only indirect, namely, those of May 28, 1884, and of August 14, 1889, the latter excluding any operation directly harmful to the foetus or to the mother. At first there was some question about the meaning of the decree of 1884, because of the wording "tuto doceri non potest," but the difficulty is removed by the explicit and unescapable assertion of the succeeding one.

These decrees of Holy Office, therefore, even if they are under-