the bacillus of Welch. In some cases the characteristic changes in the tissues remained localized to the affected limb; the opposite limb, or even the opposite part of the body, showed none of these. Occasionally the local changes seemed to have been arrested at Poupart's ligament, even though the blood was later found full of bacteria. In all cases, except where the *Bacillus putrificus* was found, the heart was found to be filled with bubbles of gas, and likewise other larger vessels of the body, as well as the vessels of the brain. The hemorrhage underneath the skin layer found so often in biopsies as well as at necropsy, could be explained after a histologic examination of the tissues. The toxin of the bacillus seems to exert a special selective action on the muscular layers of the blood vessel walls, so that an escape of the blood becomes almost natural.

Despite the small number of cases examined, I feel safe in venturing to say that the production of gas plays a not unimportant rôle in the causation of death in this disease. The acute attacks of pain complained of by the patient previous to death, the rapidity with which the fatal symptoms set in, can only be likened to caisson disease, in which air emboli are the chief pathologic factor.

In cases of abscess with gas, when the septicemic features of the disease have not developed, it is the toxin production, I am sure, that is responsible for the wearing down of the patient's vitality. Histologically the changes can best be summed up as hemorrhage, edema and gas production. The parenchymatous changes in the muscle fibers which are the predominant features histologically are an early and well-marked pathologic change constantly present. This undoubtedly contributes in a large measure to the pouring forth of blood cells and the general infiltration of the tissues with serum.

In limbs fixed immediately after amputation we have found this change, namely, the splitting and coagulation necrosis of the muscle fibers of the media, which seem to point to a special selective action of the virus of the disease on the muscle coats of the blood vessel walls.

Two cases that we have had occasion to study bacteriologically here have responded, one to amputation, and the other to an intravenous injection of the serum prepared by the injection of cultures of the *Bacillus perfringens*. This also points the way to further investigation.

The etiology of this infection may perhaps be explained by the positive presence of anaerobic bacilli in the outer clothing of soldiers. We have not carried out this phase of investigation very extensively, but on several occasions we have examined the clothing of soldiers who have been brought directly from the battlefield and have found these bacilli present.

That the presence of this bacillus is not alone the factor that permits of the production of gaseous gangrene infection is proved by the comparative frequency with which it is found in ordinary wounds. The contributing cause is always, I think, an associated lesion of a blood vessel that will produce sufficient gangrenous tissue for this bacillus to thrive in ease, and by the production of protamin toxins, break down the resistance of the surrounding tissues and enable it to continue its vicious cycle unmolested. We have found this associated injury in every case examined at necropsy. The absence of leukocytes in the immediate vicinity of the infecting area, their scavenger activities when no dead tissue is present to permit of the growth of the *Bacillus perfringens,* seem to me a fairly good indication of the soundness of this hypothesis. All these are facts which only an extended investigation can either completely verify or disprove. I trust it will fall to the lot of the laboratory units that will come here in the future to settle these points definitely. As I have said before, we have merely ruffled the surface. It remains for them to dive deep into the sea of nature and bring forth the pearls of truth.

INFANT MORTALITY DUE TO LABOR*

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The conservation of infant life, the prevention of infant mortality, is a subject that has been given much attention in recent years, not only in this country but also throughout the world. A strong national society has been organized to stimulate general interest in the subject, and to secure lay and professional cooperation in controlling the causes of the high mortality.

One of the important phases of this subject is an obstetric problem. Infant mortality during labor and due chiefly to labor is very high. The accidents of labor which destroy the fetus causing stillbirth, or those which injure it so that the infant dies shortly after birth are many. A study of these accidents involves a review of many obstetric problems. In such a review, we approach the most important questions of dystocia in a way that should lead to valuable suggestions for practice.

STATISTICS OF INTRAPARTUM MORTALITY

A statistical study is interesting and valuable, but unfortunately we have little data in the United States. In the mortality statistics of the United States Census Bureau, we have mortality statistics for the registration area, which embraces 65 per cent. of the population of the United States. For the rest of the country we have only the population statistics. Even in the registration area we have no record of births. Nevertheless, it may be of some value to make estimates, using the rates of birth and mortality from other countries in order to secure a more detailed picture of the subject. Even if the approximate figures be far from exact, they will lead us into no serious error. The causes of stillbirths are the same here as in Europe, and our study will show the relative importance of the different etiologic factors in intrapartum mortality. No claim is made for the scientific accuracy of the results; they are only helpful and suggestive in the study of the obstetric problem.

Let us assume the present population of the United States as about 100,000,000, that of Chicago as about one fortieth as much and that of Illinois as two and one-half times as great as that of Chicago or one sixteenth that of the United States. Then let us assume that Chicago and Illinois have about an average birth rate and mortality rate. This is true of the mortality rate of Chicago and probably reasonable for the birth rate. Of course we know very little about the mortality rate of the rest of the state, for, unfor-

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tunately, Illinois has no vital statistics law that is recognized by the Census Bureau, and therefore it is not included in the registration area.

Let us assume that the average birth rate for the United States and for Illinois is about 24 per thousand. This would give about 2,400,000 births annually in the United States, 150,000 in Illinois, and 60,000 in Chicago.

Brothers found the number of stillbirths in New York City to be about 8 per cent. of the births. This rate is probably too high, because the figures of the number of births on which it is based are too small. In various European clinics the rate is between 4 and 7 per cent. In the German empire it is between 3 and 5 per cent. There are nearly 3,000 stillbirths reported in Chicago, or, according to our estimate about 5 per cent. of the births. The number of reported stillbirths is probably everywhere somewhat below the actual number occurring. We shall probably not be far from the truth if we say that there are 120,000 stillbirths annually in the United States, 7,500 in Illinois and 3,000 in Chicago.

In considering the question of intrapartum mortality, it is necessary to determine what proportion of fetal deaths occur before the onset of labor and how many occur during labor. This fact could be determined easily if the condition of the stillbirths was reported, for, as is well known, the fetus becomes macerated when it remains in the uterus long after death. Reports from a number of clinics show that in nearly half of the stillbirths the fetuses are macerated. It is also important to observe that the proportion of macerated fetuses is much greater among the premature than among the children at or near term. These premature, nonviable and macerated fetuses represent the stillbirths due to antepartum disease of the mother or fetus, such as syphilis, nephritis, infection, etc. A study of this group is important, but not within the scope of this paper, which is concerned only with intrapartum mortality. Accepting the fact that, without great error, from two fifths to one half of the fetal deaths occur before labor, we may take from one half to three fifths of the figures given before for the number of nonmacerated or recently dead stillbirths, that is, for the fetal mortality during labor. We then have from 60,000 to 70,000 for the United States, about 4,000 for Illinois and 1,600 for Chicago.

In studying the effect of labor on infant mortality, we must consider not only the number of stillbirths, but also the number of deaths that result from accidents of labor when death occurs within a few days of birth. The census report gives over 5,000 deaths from "injuries of labor" in the registration area. This would correspond to about 8,000 from the whole United States. There are also about 29,000 deaths during the first week of life from congenital debility and prematurity in the registration area, or 45,000 in the United States. Over half of these infants are under 1 day old. It is of course impossible to say for how many of these deaths the labor is responsible, that is, how many could have been prevented by a better management of labor. The premature child is more easily injured even in a spontaneous and apparently normal labor. Strong uterine contractions with a deficiency of liquor amnii may produce hemorrhage in the brain or lungs. Likewise, lack of care immediately after birth will cause refrigeration to 94 F. or lower, which becomes the important factor in the early death of the child. It is certainly conservative to estimate that from 5,000 to 6,000 of these 45,000 deaths could be prevented by a better conduct of labor.

Moreover, there are in the registration area nearly 1,000 deaths during the first seven days from convulsions, and a like number from bronchitis, bronchopneumonia and pneumonia. This will correspond to about 1,600 deaths in the United States in each of these classes. Certainly a large number of these are due to injuries and accidents of labor, and probably nearly half are preventable. If we say that 7,000 of the 56,000 deaths in the first seven days of life not due to injuries of labor are due to labor, we are undoubtedly within the limit of conservative statements. Adding to these the 8,000 ascribed to injuries of labor, we have 15,000 children dying from labor within a week, and half of them the first day.

We then have the following figures denoting the number of deaths due to labor: For the United States, 65,000 stillbirths and 15,000 deaths subsequent to birth, or 80,000. Likewise in Illinois, 5,000, and in Chicago, 2,000. This would give a fetal mortality due to labor of 3⅓ per cent.

In the recent presidential address of J. Whitridge Williams before the American Association for the Study and Prevention of Infant Mortality, the author makes a study of 705 fetal deaths in 10,000 cases of labor. In this series were included all deaths during labor as well as those occurring during the first two weeks after birth. Selecting out the cases probably due to labor to compare with our own, I find 285, or a fetal mortality of 2.85 per cent. The causes of fetal death were as given in Table 1.

TABLE 1.—CAUSES OF FETAL DEATH

Cause	No.
Dystocia	124
Placenta praevia	22
Ablatio placentae	13
Toxemia	46
Prematurity (one eighth of the cases)	6
Unknown (not macerated)	74
Total	285

CAUSES OF INTRAPARTUM MORTALITY

We may now consider the influence of malpresentation of the fetus, other forms of dystocia and obstetric operations in determining the intrapartum mortality. Cross-presentations are almost always fatal unless corrected by operation. If the frequency is about 0.5 per cent., the number of cross-births in the United States is about 12,000 annually. An average death rate of 40 per cent. gives us a mortality of 4,800. Similar computations give us 300 deaths from cross-births in Illinois and 120 in Chicago.

Prolapse of the cord is responsible for about the same number of deaths. This accident occurs in many of the cross-births as well as in breech presentations, and is responsible for many of the deaths charged to these malpresentations.

Face and brow presentations taken together are slightly less frequent than cross-presentations, but the mortality should not average much above 12 per cent. This would give about 1,500 deaths yearly in the United States, from 90 to 100 in Illinois and 40 in Chicago.

Breech or pelvic presentations occur in about 3 per cent. of all labors. This rate would give 72,000

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breech labors yearly in the United States. The total mortality is from 20 to 25 per cent., corresponding to from 15,000 to 18,000 deaths due to this condition. Likewise we find in Illinois 4,500 cases with a mortality of about 1,000, and in Chicago 1,800 cases with a mortality of 400. In these cases, prolapse of the cord also plays a rôle.

Taken together, we have about 24,000 deaths from the three varieties of malpresentation. As the total number of births in malpresentations is about 4 per cent. of the total number of births or about 100,000 annually, we have the infant mortality about 24 per cent. This leaves about 56,000 infant deaths in skull presentations, or about 2.4 per cent. In other words, the infant mortality due to labor is ten times as great in these malpresentations as in skull presentations.

Passing now to the skull presentations, I shall consider the forceps operations. It is very difficult to estimate the frequency. In some sections, forceps are applied by physicians in from 40 to 50 per cent. of all cases. In other districts the frequency of forceps does not exceed that of well-managed clinics, that is, from 2 to 4 per cent. Probably if we consider the whole country as well as the state, we shall be conservative if we estimate the frequency to be between 6 and 7 per cent. of all labors. That would give us 160,000 forceps operations in the United States yearly, 10,000 in Illinois and 4,000 in Chicago. The mortality from forceps differs greatly according to the kind of operation. Low forceps are not very dangerous, while high forceps have a fetal mortality of from 50 to 90 per cent. Probably an estimate of 15 per cent. fetal mortality for all operations is conservative. This would give us about 24,000 deaths annually in the United States, 1,500 in Illinois and 600 in Chicago.

To these sources of infant mortality we may add eclampsia, or puerperal albuminuria and convulsions, as it is called in the International List, which is the cause of about 4,000 fetal deaths each year, rupture of the uterus, placenta praevia and ablatio placentae, everyone of which caused from 1,600 to 2,000 fetal deaths annually. Also a number of other rare accidents of labor or kinds of dystocia add to the infant mortality. Many of the deaths due to these causes have been already included under some other heading. This group adds about 12,000 deaths to those already enumerated in the preceding paragraphs.

Summing up, we find Table 2 expressing the approximate infant mortality from the causes already given.

TABLE 2.—CAUSES OF INTRAPARTUM INFANT MORTALITY

Causes	In United States	In Illinois	In Chicago
Malpresentations	24,000	1,500	600
Forceps operations	24,000	1,500	600
Miscellaneous causes	12,000	750	300
Total	60,000	3,750	1,500

Recalling the estimate of 80,000 deaths due to the accidents of birth, we find 20,000, or 25 per cent., unaccounted for. It is interesting to compare this with Williams' findings. He gives 127 deaths due to unknown causes. Fifty-three of this number are macerated stillbirths, however, leaving seventy-four unknown, or 26 per cent. of the 285 deaths probably due to labor. This result probably corresponds pretty well with the experience of most obstetricians. For about every fourth child that dies during or shortly after the labor, the death cannot be attributed to any of the accidents of labor or forms of dystocia, already enumerated. For reasons about to be given, I shall ascribe these deaths to the kind of dystocia not yet considered, namely, abnormal uterine contractions.

Completing our table and reducing it to percentages, we have the data given in Table 3.

TABLE 3.—CAUSES OF INTRAPARTUM INFANT MORTALITY

	Per cent.
Malpresentation	30
Forceps operations	30
Miscellaneous causes, including placenta praevia, ablatio placentae, ruptured uterus, toxemia, etc.	15
Pathologic uterine contractions	25

PATHOLOGIC UTERINE CONTRACTIONS

I wish to call special attention to the last cause and try to justify ascribing so much importance to it. The danger of abnormal uterine contractions is not, as a rule, sufficiently recognized. Before labor begins, the oxygenation of the fetal blood occurs in the placenta. A continuous and abundant circulation of the maternal blood furnishes the oxygen and removes the waste from the fetal blood. When the uterine contractions begin, the maternal circulation is disturbed. So long as the contractions last only a short time and are separated by considerable intervals of relaxation, there is no appreciable disturbance to the fetus. Our main index of the fetal condition is its circulation or heart tones. The frequency of the heart tones changes but little or not at all during the early contractions of labor.

When the contractions last longer and occur more frequently, there is more disturbance in the placental circulation and more derangement in the fetal circulation. If the contractions last more than one and one-half minutes, and if the intervals between contractions are shorter than the contractions themselves, the condition is pathologic and dangerous. If the contractions become more frequent and prolonged so that there is hardly any interval, we have the condition called tetany uteri, which almost always results in fetal death.

Such excessive contractions may occur early in labor, but they generally come on later. They are often the reaction of the uterus to obstacles to delivery, and so occur in contracted pelves, bad presentations, etc. They are excited by operative interference. They are the cause of fetal death not only in the 25 per cent. of causes given in the table, but also in some of the other categories. In all malpositions, fetal death is frequently due to excessive contractions, excited by the delay to labor. It may be true that in 50 per cent. of all intrapartum fetal deaths, the immediate cause of death is excessive uterine contractions.

If it should be objected that 20,000, or 25 per cent., of all fetal deaths is too large a number to be ascribed to this cause, it must be remembered that the mortality rate is nevertheless small. From 2,400,000 labors each year we deduct 100,000 malpositions and 200,000 forceps cases and cases of toxemia, placenta praevia, etc., leaving 2,100,000, in which 20,000 deaths make less than 1 per cent. A certain number of these cases are undoubtedly contracted pelves, and the injuries produced by the pressure required to mold the head to make its translation possible are factors that combine with the excessive uterine contractions to injure the child. It is, indeed, difficult always to apportion the blame. Even when postmortem examinations are made, the findings are not decisive. A

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careful study of the clinical history is, however, often conclusive. The contractions are normal for hours, and the child in good condition. Then comes a period of perhaps only an hour or two of very severe pains, long and hard, with short intervals, after which the fetal heart tones are wanting.

It is very important, therefore, to watch the uterine contractions with great care in long and hard labors, and especially toward the end of labor, when excessive contractions are more apt to occur. The diagnosis of the contractions is easily made by watching the patient and noting the hardening of the uterus by laying the hand on the abdomen. The diagnosis of the condition of the fetus is made by counting the fetal heart tones. Whenever there is danger, the heart tones should be counted every ten to twenty minutes.

The management of excessive contractions to prevent fetal death is to control the contractions. The best means is the hypodermic injection of morphin, and anesthesia. One-fourth grain of morphin is generally sufficient in the first stage of labor. If necessary, this dose could be repeated, for at this time there is not very much danger of morphin affecting the child. In the second stage, ether may well be combined with morphin, or substituted for it. Should the obstacle to delivery that excites the excessive uterine contractions be at the obstetric outlet, that is, should the head be on the perineum and held back by a tense, unyielding vulvar ring, episiotomy should be done. If the head is not at the vulva but down in the pelvis, and the cervix is well dilated, and if, in spite of morphin and anesthesia, the danger to the child is great, forceps may be applied. As a rule, however, forceps will increase the danger of fetal asphyxiation and should not be used unless an easy and quick extraction is possible.

MORTALITY FROM THE USE OF FORCEPS

The prevention of intrapartum death in case of bad presentation involves the correct management of these forms of dystocia, and cannot be entered on in detail in this paper. In regard to the deaths due to forceps operations, it must be said that forceps are employed much too frequently—often on account of the entreaties of the patient and her friends, often to hasten the labor for the convenience of the physician. Whenever forceps are used for these reasons, and the child is lost, its death is rightly charged against the operation, which is not far removed from the category of malpractice.

There is a great difference in the danger of high forceps and low forceps. The former has been used far too much in contracted pelves. We have learned that other operations are safer both to mother and to child. Cesarean section or hebosteotomy is in proper cases to be chosen, while the induction of premature labor must be considered in cases seen early enough in pregnancy. Above all, these cases should be studied long and with care. Exact diagnosis, not only of presentation and position, but also of station, should be made. One who rushes into a high forceps operation thinking he has to do with a head in the pelvis, and then is astounded to find that he must exert all his strength to extract a child that is dead or dying, should learn that obstetrics has advanced in recent years, and that the high forceps is an operation that is very nearly discredited.

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THE LATER HISTORY OF THE TYPHOID CARRIER H. O.

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In two previous articles, the history[1] of a typhoid carrier, H. O., and his treatment by typhoid vaccine[2] have been reported. The more recent developments in his case show that he was not cured by the vaccination or by the extirpation of the gallbladder, and that after an interval of many months, during which typhoid bacilli could not be found in the feces, he infected other persons.

Through the courtesy of Surgeon R. M. Woodward, medical officer in command of the United States Marine Hospital in San Francisco, I have had the privilege of drawing on the records of the hospital for the greater part of the material here presented.

REVIEW OF THE EARLIER REPORTS

The sailor, H. O., was sick with typhoid fever in the United States Marine Hospital in San Francisco from Nov. 15, 1907, to Jan. 15, 1908. During a period of four years, from March 6, 1908, to March 19, 1912, he worked on several steamships, but most of the time on the *Acme*. During this period he infected twenty-seven officers and sailors with typhoid fever. Four of them died. An investigation by the San Francisco Department of Health into the cases on the *Acme* brought H. O. under suspicion as a carrier and the source of the infection. He was studied in the City and County Hospital for a period of two weeks in December, 1911, but typhoid bacilli were not found by the city bacteriologist in a series of examinations of his feces, undoubtedly owing to intermittence in the discharge of the bacilli in detectable numbers.

The State Board of Health later gathered circumstantial evidence which pointed to H. O. as a carrier responsible for the cases, and a specimen of feces obtained March 3, 1912, was found to contain many typhoid bacilli.[1] H. O. did not handle or serve food on shipboard, and it is probable that the infection was transmitted to his comrades in various ways, but chiefly through the water in a cask from which the men dipped drinking water.

March 19, 1912, Surgeon James M. Gassaway offered to admit H. O. to the United States Marine Hospital. Since that time, except for a short period of parole, H. O. has remained voluntarily at the hospital, although technically under quarantine by the California State Board of Health. Every effort has been made by the staff of the hospital to free him from the carrier state.

The article by Currie and McKeon[2] takes up the history at this point. Between March 28 and Oct. 14, 1912, specimens of feces were examined weekly and biweekly at the laboratory of the United States Public Health Service in San Francisco. The typhoid bacillus was isolated twelve times, but not later than June 19. Examination of the urine consistently give negative results.

In an attempt to cure H. O., an autogenous typhoid vaccine was administered between April 27 and June

1. Sawyer, Wilbur A.: A Typhoid Carrier on Shipboard, THE JOURNAL A. M. A., May 4, 1912, p. 1336.
2. Currie, Donald H., and McKeon, F. H.: History of a Typhoid Carrier, THE JOURNAL A. M. A., Jan. 18, 1913, p. 183.

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