GUIDELINES

For

MATERNAL CARE

PREPARED BY

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Department of Obstetrics,
University of Iowa College of Medicine
and
Statewide Perinatal Care Program

STATEWIDE PERINATAL CARE PROGRAM

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September 28, 1976

Clarence H. Denser, Jr., M.D.
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Dear Dr. Denser,

Enclosed are copies of the booklets Guidelines for Maternal Care and Guidelines for Newborn Care.

These publications are now out of print. At the current time the Perinatal Program staff is putting together revisions of the booklets, with additional topics and updating of the materials. We hope to have the revisions ready for publication early next year.

If you would be interested in receiving copies of the revisions, please contact our office in the spring of 1977.

Sincerely,

Norma Ferguson

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Clinical Nursing Specialist -
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NF/mw
Enclosures: 2

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INTRODUCTION

This booklet has been prepared to assist the physicians and nurses caring for obstetrical patients in the state of Iowa. The items discussed were determined on the basis of need (hospital survey) and are discussed from the standpoint of the ability of the individual hospitals (any size) to render the care indicated.

We feel that a reasonable adherence to the guidelines for maternal care will insure an adequate standard of care for all obstetrical patients in the state of Iowa.

These guidelines are presented with no associated regulatory authority. Since the physicians and nurses practicing in Iowa hospitals are all health care professionals, we do not feel that regulatory measures are required to insure adequate care.

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PRENATAL RECORDS

PRENATAL RECORDS

We recommend that hospital obstetrical departments receive a complete copy of the prenatal record about 2-3 weeks prior to the E. D. C.[1] This copy should be on file in the obstetrical unit so it is immediately available when the patient is admitted in labor.[2]

Many times the nurse is the only professional person to see the patient during the all important first stage of labor. Her knowledge of the patient's past medical and obstetrical history, baseline blood pressures and fetal heart rates, as well as laboratory findings during the present pregnancy will enable her to give a higher level of nursing care to the patient, and a more individualized approach to any problems. The identification of a high-risk pregnancy will alert the nurse to the patient who requires more intensive and closer monitoring during her labor.[3]

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1. Standards for Obstetric-Gynecologic Services: The American College of Obstetricians and Gynecologists, 79 W. Monroe Street, Chicago, Ill. 60603 (1969) page 28

2. Schneider, Jan: Changing Concepts in Prenatal Care Post Graduate Medicine, Vol. 53 No. 7 June 1973 91-97

3. Clausen, Flook. Ford, Green, and Popiel: Maternity Nursing Today McGraw-Hill, Inc. 1973 page 789

Current practices in prenatal care deserve reevaluation. What could be a model of preventive medicine is often a ritual of rigidly scheduled visits with little meaning. Recognition of existing or potential complications, patient education, and research into the problems of pregnancy are important elements in a model program.

JAN SCHNEIDER, M.D.
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CHANGING CONCEPTS IN PRENATAL CARE

Current Status

The first three decades of the twentieth century saw remarkable changes in the mode of obstetric care in the United States. The safety of childbearing improved greatly during this era, not only because of better health care and such medical advances as the discovery of sulfonamides and antibiotics, safer technics for blood transfusion, and more skillful methods of anesthesia, but also because of general improvement in living conditions. Maternal mortality committees were introduced in the 1920s and increasingly influenced the practice of obstetrics through their educational impact.[1] Although neonatal and infant mortality rates started to fall at the turn of the century, maternal mortality did not decline significantly until the 1930s.

In the early years of the century a campaign against the untrained midwife was effectively mounted,[2] although there was some fear of manpower shortages if midwives were banned. During this time prenatal care was first introduced to this country from Great Britain. By the 1930s it had become firmly established as a component of maternity care. It is now probably the best example of preventive medicine.

Prenatal care has been well described as "a planned program of observation, education and medical management of pregnant women directed toward making pregnancy and delivery a safe and satisfying experience."[3] Prenatal care can do much to reach these objectives, and many programs have achieved high degrees of skill and success. In spite of this, much of prenatal care offered today has become a ritual in which health education is virtually nonexistent and care is perfunctory.

Current practices in prenatal care deserve reevaluation. Like all health services, prenatal care may be viewed from three separate angles: what the professionals think the patient needs, what the patient thinks she wants, and what the system is able to deliver. Ideally, all three should be the same. To achieve this, a program of prenatal care must contain three elements: preventive intervention, health education, and research and learning in problems of pregnancy.

A Model Program

Preventive intervention—Prenatal care gives a unique opportunity to observe and care for a woman for more than half a year. Monitoring and maintaining the normalcy of gestation, identifying the need for therapeutic intervention, and reassuring and educating the patient form the crux of prenatal care.

Clinical management—The critical first visit may be considered a triage. A full history

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should be taken, including data on previous illnesses, family and social histories, previous pregnancies, menstrual pattern, and status of the present pregnancy. A complete physical examination should include pelvic examination, pelvimetry, and estimation of uterine size.

At completion of this visit the physician can fairly well assess the risk status of the individual, although results of initial laboratory tests may not yet be available. On the basis of his findings the physician can devise a program of prenatal care and identify times when particular attention may be needed. Preparations can be made for special laboratory tests, for consultation with other specialists, nutritionists, or other health workers, or for any other special services. Data can be obtained from other physicians or hospitals. He can prescribe the necessary medication, including iron supplements (usually with folic acid).

If the patient needs immunization boosters, these can be given during prenatal care, but live virus vaccines should be avoided. Obviously, rubella vaccine is absolutely contraindicated in pregnancy. Smallpox vaccination should be given only when a patient is about to travel to an area where smallpox still exists; then, ideally, the patient should also receive vaccinia immune globulin for protection.

Thus, the physician can design an individualized schedule of prenatal care for each patient and discuss it with her. The patient must understand what is being done, what is planned, and why. A program of health education during pregnancy should also be planned. As part of this, at the first visit the patient should be instructed about danger symptoms in early pregnancy and should be told to note the calendar date when she first becomes aware of fetal movement.

The timing of the second prenatal visit depends on individual factors, including the gestational date. Since this visit is usually after the twelfth week, enlargement of the uterus can be assessed. By this time the physician has the results of laboratory tests, which may necessitate modifying the original plan.

At every visit, it is critical that the physician think in terms of dates—that he compare uterine and fetal size with the norm and that he make adequate notation of their compatibility or disparity. Only in the first half of pregnancy is the physical examination useful to assess gestational age. After the twentieth week the fundal height is so much a reflection of the ultimate size of the fetus, the volume of amniotic fluid, and the depth of the presenting part within the pelvis that it is little related to fetal age. Nothing is more frustrating than belated recognition of uncertainty concerning the expected date of confinement when induced labor or cesarean section is planned. In most cases this can be prevented by estimating uterine size at 16 and 20 weeks.

The dates of quickening and of first audibility of the fetal heartbeat further help to confirm gestational age, although the accuracy of these landmarks of midpregnancy is not great. In the early weeks of pregnancy, disparity between gestational age and uterine size may raise the first suspicions of multiple pregnancy, hydatidiform mole, or intrauterine death.

In the second trimester the normal patient usually feels well; the fetus grows and there is little need for frequent visits. By the twenty-eighth week, however, the need for reevaluation arises. Certain laboratory tests should be repeated. By this stage the influence of pregnancy on iron reserve is fully demonstrable. The gestational effect on glucose tolerance approaches the maximum, and active erythroblastosis can be recognized. From this point on, fetal size can be roughly evaluated; an estimate should be recorded at each visit and compared with the average. Only by practice and honest notation will the physician learn the limits of his ability to forecast fetal size. (Unfortunately, estimates are most reliable when the fetus approaches the average and tend to be least reliable at the extremes of fetal weight, when the need for accuracy is greatest.)

As term approaches, the physician must carefully assess fetal position and presentation and, occasionally, must correct malpresentation. At this time multiple pregnancy, hydramnios, or aberrations of gross fetal development are recognizable. Early toxemia of pregnancy must be excluded at each visit.

Health education should parallel clinical care, but it is essential that the physician review with

the patient what she should do at onset of labor or if membranes rupture prematurely. The patient should have the opportunity to discuss with the physician the program planned for labor, analgesia, anesthesia and delivery. However, it is both unrealistic and unfair to make specific commitments on details of care that may need to be amended once labor starts.

Routine laboratory testing—The greatest benefit can be attained by clinical assessment both initially and throughout pregnancy. However, it is essential that certain laboratory procedures be performed routinely and any abnormalities fully investigated. The number of tests considered routine has gradually expanded as procedures have passed from research status to special investigation in occasional patients and then to use as screening devices for all.

At the first visit, blood must be checked for anemia, syphilis, blood group, Rh factor, and possible presence of antibodies. If an antibody screening test is positive, the specific antibody must be identified. Black patients must be tested at the first visit for sickle cell anemia; if the result is positive, hemoglobin electrophoresis is required to identify the specific pattern. Ideally in such cases, the husband should also be tested, with a view to offering genetic counseling to the couple.

Recently, hemagglutination inhibition tests for antirubella antibody have become customary. Knowledge of the patient's immune status is useful, but not all laboratories are equipped to give consistently reliable results on the hemagglutination inhibition test, and even in the best laboratories, antirubella titer varies greatly from day to day, with up to a four tube difference. Thus, a low level on one occasion and a higher level later in pregnancy may mean nothing. It is necessary to keep serum frozen and test two samples simultaneously to demonstrate a change in antibody level over the course of time.

A Papanicolaou smear is routine at the first visit unless the patient has had a recent negative report. Chest x-ray has been considered essential, but the low incidence of tuberculosis in most of this country makes even minimal radiation hazard unjustifiable. Skin tests would be preferable, with chest x-ray only when skin test reactions are positive.

Urinalysis for glucose and albumin is necessary at every visit. Glycosuria, although often due to a lowered renal threshold in pregnancy, indicates a need for a blood sugar determination. Albuminuria may be the first evidence of toxemia or other renal disease. The high incidence of urinary infection in pregnancy and the controversy concerning the significance of asymptomatic bacteriuria suggest the value of routine testing. This can be done on a clean-

In the care of the pregnant patient, the physician's memory or availability cannot replace an accurate record.

voided specimen by a simple screening culture, such as Testuria. When the colony count is positive, a catheter specimen for culture is indicated.

If pregnancy progresses normally and all initial laboratory findings are normal, certain procedures are routinely indicated later. At the beginning of the third trimester, blood should be checked again for anemia. An antibody screening test should be repeated on all patients. This is essential for Rh-negative patients to insure that sensitization has not occurred. It must be done early enough to permit serial amniocentesis if erythroblastosis is suspected. Repeat antibody screening in Rh-positive patients is justified because it can provide valuable information for crossmatching blood that may be required later.

The third trimester is an ideal time to detect abnormal glucose tolerance. It can be argued that a postprandial blood sugar level should be determined in all patients as a screening measure. Certainly, the procedure is indicated in any patient who has a family history of diabetes mellitus, a history of delivery of an excessively large infant or unexplained fetal death, or glycosuria, hydramnios, or any other abnormality associated with diabetes. During pregnancy, two hour postprandial blood sugar tests are more

reliable than fasting blood sugar tests. If the postprandial findings are abnormal, a full glucose tolerance test is necessary.

Special laboratory procedures—Several procedures specific to problems in pregnancy are in current use and several others are still in the developmental stage.

Perhaps the greatest recent advance has been the realization that amniotic fluid can be obtained with minimal risk to the fetus. Amniocentesis as a diagnostic test was introduced for the spectrophotometric analysis of fluid in cases of Rh sensitization.[4] The bilirubin curve plotted against gestational age gives the best prognostic indication of the status of the erythroblastotic fetus and is a better measure than serial antibody titers from maternal blood, although the latter may be valuable as a secondary evaluation. Other tests performed on amniotic fluid include biochemical, cytologic, cytogenetic and virologic assessments of fetal status.

Fetal maturity may be evaluated by the increase in the number of fetal fat cells in the amniotic fluid as pregnancy progresses, the fall in bilirubin level in normal patients, and the rise in creatinine levels as fetal kidneys mature.[5,6] The most recent, most reliable test for maturity specifically forecasts the status of fetal lung development and the risk of hyaline membrane disease. This is assessed by a rising ratio of lecithin to sphingomyelin in the fluid as the lungs mature.[7] These tests, with x-ray for calcification of the fetal distal femoral epiphysis, can be employed in order to insure that induced labor or cesarean section does not result in a fetus that is unexpectedly immature.

Cytogenetic studies of the fetal cells in the amniotic fluid are just beginning to yield their value. The best application currently is in detecting Down's syndrome early in pregnancy. The high incidence of this disorder in older gravidas suggests the value of obtaining a sample of fluid as early as possible in pregnant women beyond their mid-30s or in any woman who has previously delivered a mongoloid infant with chromosomal translocation. Fluid can rarely be obtained before the fourteenth week, and tissue culture and karyotyping require at least two weeks. Late abortion should be available to patients with a positive test.

Another laboratory procedure that has become widely accepted is measuring the estriol level in maternal urine.[8,9] Estriol is a product of the fetoplacental unit, and in normal pregnancy its level rises steadily. It is excreted in maternal urine, and unless renal disease is present, can be used to monitor fetal progress. In any pregnancy in which there is concern about fetal death, serial assessment of estriol should be initiated early in the third trimester.

Another recent addition to obstetric practice is ultrasound. With fairly simple devices, the Doppler effect can be used to detect the fetal heartbeat much earlier than it is audible through a stethoscope. Echo technics can be used for cephalometry, placental localization, diagnosis of multiple pregnancy, and identification of hydatidiform mole. However, this equipment is complex, expensive, and not yet widely available.

Special procedures also include radioisotopic localization of the placenta, arteriography, amniography, and other radiographic technics; culture of rubella virus from amniotic fluid; and special sensitive hormonal assays for trophoblastic disease.

Communication—The obstetrician is responsible for the total care of his patient during pregnancy. It is therefore important that he maintain full communication with other physicians or health workers involved in her management. This is particularly important for patients with medical complications who are seen by consultants or who are under the care of an internist. It is important for the obstetrician to understand what is happening to his patient,

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but it is even more critical that he know that it is happening!

Record keeping is necessary if data obtained by history and from previous health care facilities, consultants, and observations made during pregnancy are to be of ongoing benefit to the patient. The hospital should have this information when the patient is admitted in labor.

Occasionally it has been argued that some of the information obtained in the physician's office is of too personal a nature to become part of a hospital record. This is clearly untenable. The hospital record is a confidential document; if concern exists about its integrity, efforts must be made to insure that persons without authority do not have access to it, rather than to limit the information it contains.

Logistic difficulties need to be overcome to obtain information flow and should not be used as an excuse for inferior care. There seems to be little reason why patients should not be trusted with at least part of their record. The usual argument that this causes loss of records has rarely been tested. In at least one European country, patients carry their own standardized prenatal record, which admits them to the hospital.[10] No physician's memory is good enough or his availability so constant that it can replace an accurate record. Continuity of care is better achieved by the written word than by any amount of personal concern. Prenatal care is only as good as its record keeping.

Health education—Probably nothing is more tedious for most physicians than repetitive individual instruction of patients, yet no part of prenatal care is more amenable to improved efficiency. Because personnel in the regular channels of prenatal care have shown so little initiative in patient education, it is not surprising that other agencies have taken over by default. Patients are eager to discuss effects of pregnancy on their body and their life and to prepare for the forthcoming labor, delivery, and young parenthood.

Most organizations offering classes in preparation for childbirth do a good job. Their general philosophy includes four components: (1) instruction in the biology of pregnancy, parturition, and neonatal life and in necessary health measures in pregnancy, (2) emphasis on the normalcy of parturition and the need for active patient participation (usually with minimal analgesia and anesthesia), (3) instruction in physical exercise that may facilitate progress of labor and minimize discomfort, and (4) involvement of the father in the concept of family-centered childbearing.

At times, problems and disagreements have occurred between those involved in parent education and the medical profession. Often the physicians responsible for the bulk of obstetric care have had minimal involvement in classes for their patients, and on occasion open hostility between obstetricians and educators has resulted. A few groups do hold strangely cultish ideas with little scientific basis: here the source of disagreement is obvious. Such situations are unusual. The need for health education in pregnancy is great, and this service must again become a major component of obstetric care.

An acceptable, efficient way of imparting information is group discussion. This format shows the normal patient that others share her anxieties and discomforts. Obstetric nurses or other trained health workers can conduct group discussions. Group leaders must have adequate factual information, so that knowledge will be imparted as well as experiences shared. Although such sessions have an element of group therapy, the primary role should be educational. Leaders should be able to identify symptoms that need to be checked later by a nurse or physician.

Group sessions should be scheduled at intervals for patients who are at the same stage of gestation. Thus, discussion can be directed to problems and concerns most common at that particular stage. Optional evening classes open to fathers are often popular and are essential

to encourage the family-centered concept. Educating both partners makes for a happier, often easier obstetric experience.

The greatest need for education exists at the lowest socioeconomic level, but patients from these populations attend educational sessions as irregularly as they come for medical care. Problems of motivation, transportation and babysitting must be overcome. The poorer the patient, the greater all such obstacles tend to be. Thus, some attempts at patient education are inevitably disappointing. Overall usefulness of patient understanding of fetal development, labor, delivery, newborn care, and family planning is evident—health education efforts can do much to strengthen the value of prenatal care. Enthusiasm for education, however, must not be allowed to result in meaningless schedules for this service. The costs and value of health education must be assessed in the same manner as are other components of care.

Prenatal care can be simplified by efficient scheduling of telephone discussion to replace some visits.

Research and learning—Initially, research and learning as an element of antenatal care was intended as a source of greater understanding of pregnancy and its problems. From such knowledge, modifications of obstetric management could be devised. Activity in this area is more intense today than ever before. Currently, the subject of most intensive study is fetal development; prenatal care has been termed intrauterine pediatrics and is increasingly viewed as the first phase of lifelong care.

At present, the areas in obstetric management that probably require the greatest reappraisal are the programs and means of providing prenatal care. Changes in methods of delivery need not affect content of care. However, personnel involved, distribution of tasks, and scheduling of visits should be reevaluated.

Prenatal care is time-consuming for the physician. A recent survey by the American College of Obstetricians and Gynecologists indicated that the private physician specializing in obstetrics and gynecology spent an average of 27.5 hours in his office each week and saw 90 to 100 patients.[11] Almost half of these patients came for prenatal or postpartum care.

Personnel other than the obstetrician-gynecologist can adequately perform many of the routines of prenatal care. Another study[12] showed that most physicians would delegate many office tasks to skilled obstetric nurses if they were available. Obstetric nurse-practitioners, assistants and others may be trained to perform many time-consuming prenatal care functions. Further, the office nurse can use her skills to better advantage if workers with less training perform simple tasks, such as making appointments, testing urine, and weighing the patient. The team approach ideally can be introduced into prenatal care. With supervised delegation of responsibility, all personnel can work to the maximum level for which they have been trained.

A nurse often has a more sympathetic attitude than a physician toward the minor concerns of the healthy pregnant patient. Introducing the nurse into this area of care does more than enable the physician to avoid some of the more tedious routines. The nurse's skills can complement those of the obstetrician, her schedule is more predictable, and she can see patients without the haste so typical of the overtaxed physician.

Another area of necessary innovation is the scheduling of visits. Current norms are based on gestational age and are almost identical to schedules that were devised more than half a century ago.[13] The healthy, normal patient does not need to be seen as often as custom dictates, while patients with physical or emotional problems may need to be seen more often.

If a healthy patient is fully counseled as to symptoms she should report and if the physician assesses the early milestones of uterine growth adequately, no more than three or four visits are needed between the twentieth week and delivery. A health education session can replace one or two later visits, particularly if the nurse has an opportunity to counsel the individual briefly and perhaps take her blood pressure before or after the group discussion.

If visits are less frequent, each must be better used. Prenatal care can also be simplified by efficient scheduling of telephone discussion to replace some visits, particularly those specifically designed for reporting laboratory values.

Evaluation of Care

The need for evaluating the benefits of prenatal care is obvious. However, a constant problem in establishing standards of measurement is the preselection of patients. Patients with the highest motivation for early and frequent attendance come from the social and educational population with the least inherent risk of poor pregnancy outcome. Thus, not surprisingly, in study after study the quantity of prenatal care as measured by the number of visits has correlated with fetal well-being.

The lack of significance of this association is well demonstrated when we consider low-birthweight infants born before term. The mother who delivers such a "premature" baby is deprived of that segment of care during which visits are scheduled most often, yet her baby has the highest risk of neonatal death. Clearly, cause and effect are not demonstrable in such associated situations.

Thus, hard data on benefits of prenatal care are scarce. Yet, even high-risk patients do benefit from intensive preventive care, as indicated by results of special programs such as the Maternity and Infant Care projects. Not surprisingly, such patients require more time, effort and services than most, with a much greater total cost. The multiple needs of high-risk patients require the skills of a team of health, social, educational and other personnel. Such services for high-risk patients can rarely be provided without a special program. It is paradoxical that specialist obstetrician-gynecologists generally spend most of their time looking after patients at lowest risk, while patients with the greatest inherent hazard of poor outcome of pregnancy are frequently the responsibility of trainee physicians.

Summary and Conclusion

Since its rapid development in the first decade of this century, prenatal care has gradually been reduced to a series of unimaginative routines. Reevaluation of the modes of delivery of prenatal care is necessary, with full use of the values of preventive intervention, health education, and research and learning. Increased individualization of programs of care and more efficient use of personnel, facilities and technology are required, together with full assessment of the usefulness of what is done.

When given thoughtfully and well, supported by adequate laboratory and other ancillary services, and accompanied by a flow of information and continuity of care from ambulatory setting to hospital, prenatal care surely provides the best example of preventive medicine. It benefits not only the mothers of today but the generations of tomorrow.

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MATERNAL NUTRITION IN THE PRENATAL PERIOD

Recent research developments in prenatal nutrition indicate that some of the practices that have been followed in our country in regard to diet for the expectant mother are of little value, and indeed may be potentially harmful to mother and fetus. The longtime adages of weight and salt restriction, iron and vitamin therapy, as well as the routine use of diuretics to relieve edema in a normal pregnancy have all come up for further study and investigation.

The American College of Obstetricians and Gynecologists has been active in promoting further understanding of the complex problems of nutrition during pregnancy, lactation, and the puerperium. The policy statement issued December 1, 1972 is an example of the concern.[1] The statement that wieght gain in pregnancy should not be restricted unduly, nor that weight reduction should be attempted,is a new concept to physicians and nurses who have been taught that a weight gain of 10-15 pounds during pregnancy is better for the mother, and may prevent the development of toxemia of preganacy.

Average weight gains of 22 to 27 pounds now are being accepted, with emphasis on the content of the diet and the pattern of the weight gain from week to week rather than on the total amount.[2]

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1. American Collegeof Obstetricians and Gynecologists: Policy statement on nutrition and pregnancy, Dec. 1, 1972

2. Pitkin, Roy M. et al: Maternal Nutrition, A Selective Review of Clinical Topics Obstetrics and Gynecology Vol 40. No. 6 Dec. 1972 773-785

POLICY STATEMENT ON NUTRITION AND PREGNANCY
AMERICAN COLLEGE OF OBSTETRICIANS & GYNECOLOGISTS

A woman's nutritional status before, during, and after pregnancy contributes to a significant degree to the well being of both herself and her infant. Therefore, what a woman consumes before she conceives and while she carries the fetus is of vital importance to the health of succeeding generations.

Nutritional assessment and advice are essential components of good antepartal care. The obstetrician-gynecologist, as leader of the obstetric-gynecologic health team, is responsible for including these in the care of all pregnant patients under his supervision. He may do this personally or by insuring that qualified members of his team do so. Ideally, nutritional assessment should be made before conception. Failing that, it should be accomplished at the first antepartal visit. It should be repeated at regular intervals during and following pregnancy. A basic technique of nutritional assessment is to obtain and analyze a 24 or 48 hour diet history. This should be supplemented by clinical evaluation of possible nutritional deficiencies and, where indicated, appropriate laboratory tests.

Nutritional advice to the pregnant woman depends upon knowledge of sound nutritional principles. Although the components of optimal maternal diet have not been determined precisely, several important principles may be stated:

1. Adequate intake of protein, particularly protein from animal sources, should be insured.

2. Caloric intake approximately 10 per cent above non-pregnant requirements is advisable.

3. Weight gain during pregnancy should not be restricted unduly, nor should weight reduction normally be attempted. The average weight gain in normal pregnancy is 10 to 12 kg (22 to 27 lbs).

4. Essential nutritional elements (such as sodium) should not be restricted during normal pregnancy.

5. Dietary supplements of iron and iron-containing foods are indicated during pregnancy. Other dietary supplements, such as vitamins or additional sources, may be helpful where deficiencies in nutritional status are determined.

Nutritional advice should be continued during the puerperium. Restriction of dietary intake should not be advised during the early postpartal course nor for the lactating mother.

In implementing these principles of good nutrition in obstetrics, the obstetrician-gynecologist may utilize written or pictorial materials. However, the most important factor in persuading a patient to establish sound nutritional habits before, during, and after pregnancy is continued personal encouragement by the obstetrician-gynecologist and the members of his health team, with emphasis on positive rather than negative aspects of nutrition.

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Maternal Nutrition

A Selective Review of Clinical Topics

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WE ARE PRESENTLY witnessing a renewed interest in the subject of nutrition during pregnancy. Although it has long been acknowledged that the potential influence of maternal nutrition on the course and outcome of pregnancy is profound, only recently has the full significance of this concept begun to be accepted, and with it the realization that the future health of mankind depends, to a very large degree, on nutritional foundations laid down during prenatal life. It has been stated that nutrition in pregnancy is "an idea whose time has come."

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A major landmark was the appointment of a Committee on Maternal Nutrition of the Food and Nutrition Board, National Academy of Sciences–National Research Council. After a 3-year study, this Committee issued its report[1]—a comprehensive review of available information—in 1970.

In this current flurry of interest in nutrition during pregnancy, the physician responsible for the care of pregnant women often finds himself in a difficult position. His knowledge of nutrition, in general, is deficient; formal instruction in nutritional principles is notably absent from medical school curricula and graduate programs. At sometime in his educational experience he may have been inculcated with some dogmatic concepts about the dietary management of the pregnant woman, but even these concepts are now being questioned. Thus, when faced with providing nutritional advice to

his patients, he too frequently finds himself confused and not a little bewildered.

The purpose of this report is to review the subject with respect to some of the topics in maternal nutrition which are of practical significance to the clinician—nutritional supplementation, caloric intake and sodium metabolism. This review was conducted by the *ad hoc* Committee on Nutrition of the American College of Obstetricians and Gynecologists. It is selective rather than exhaustive and is aimed at determining the proper course of management, when this can be determined.

NUTRITIONAL SUPPLEMENTATION

With few exceptions, increased quantities of those dietary components essential in human nutrition are required during pregnancy. Table 1 lists the Recommended Daily Dietary Allowances (RDA) of the Food and Nutrition Board (National Academy of Sciences–National Research Council) for certain nutritional elements, along with the additions to be made for pregnancy and lactation. In view of these increased requirements during pregnancy and the ready availability of most of these substances, it is hardly surprising that vitamin and mineral supplements are advocated as a routine measure. Indeed, their use is so deeply ingrained in obstetric practice, it is accepted by physicians and patients alike as an integral part of antepartum care.

Vitamins (Other than Folic Acid)

While animal experiments have indicated that various vitamin deficiencies have an adverse affect on the outcome of pregnancy, the specific deficiency usually must be both profound and prolonged before its effects are seen. In man, overt evidence of vitamin deficiency is very rare in developed countries, at least in this modern era. Moreover, biochemical surveys of large groups of pregnant women have generally indicated that levels of various vitamins are normal in nearly all instances.[2] There are exceptions to this general rule, as for example with vitamin B_6,[3] but the clinical significance of such

TABLE 1. RECOMMENDED DAILY DIETARY ALLOWANCES
(FOOD AND NUTRITION BOARD, NATIONAL ACADEMY OF SCIENCES-NATIONAL RESEARCH COUNCIL REVISED 1968)

Dietary requirement	*Age of females*		*Additions for*	
	16–18	*18–35*	*Pregnancy*	*Lactation*
Calories (kcal)	2300	2000	200	1000
Protein (g)	55	55	10	20
Fat soluble vitamins				
A (IU)	5000	5000	1000	3000
D (IU)	400	400	0	0
E (IU)	25	25	5	5
Water soluble vitamins				
Ascorbic acid (mg)	50.0	55.0	5.0	5.0
Folacin (mg)	0.4	0.4	0.4	0.1
Niacin (mg equivalents)	15.0	13.0	2.0	7.0
Riboflavin (mg)	1.5	1.5	0.3	0.5
Thiamin (mg)	1.0	1.0	0.1	0.5
B_6 (mg)	2.0	2.0	0.5	0.5
B_{12} (μg)	5.0	5.0	3.0	1.0
Minerals				
Calcium (g)	0.8	0.8	0.4	0.5
Phosphorous (g)	0.8	0.8	0.4	0.5
Iodine (μg)	100.0	100.0	25.0	50.0
Iron (mg)	18.0	18.0	*	
Magnesium (mg)	350.0	350.0	100.0	100.0

* Supplements of ferrous iron, 30–60 mg daily, recommended

data is unclear. Finally, controlled, double-blind clinical studies have usually failed to indicate that vitamin supplements have a salutory effect on the outcome of pregnancy.[4]

While available evidence does not indicate vitamin supplements have a beneficial effect, neither does it generally suggest harmful sequelae. A possible exception, however, is Vitamin D. Some evidence suggests (though by no means does it prove) a relationship between maternal hypervitaminosis D and the subsequent development of severe infantile hypercalcemia with craniofacial abnormalities and supravalvular aortic and pulmonic stenosis. For this reason no increase in Vitamin D intake is advised during pregnancy.[5] It should be pointed out that the recommended daily intake of Vitamin D (400 international units, pregnant or not) is the amount contained in 1 quart of Vitamin D–enriched milk.

A potential danger of routine vitamin supplementation is the false sense of security it may convey to either patient or physician regarding deficiencies of essential nutritional elements other than vitamins. Vitamins will certainly not compensate for poor food habits.

Iron and Folic Acid

The burden pregnancy imposes on the maternal hematologic system is clinically very significant. Of the many elements involved in hematopoiesis, the limiting factor in the synthesis of hemoglobin is usually the availability of iron. Iron metabolism in pregnancy has recently been summarized by Pritchard.[1] The amount of elemental iron in the term fetus is approximately 300 mg, and that required for augmented maternal erythropoiesis (necessary to prevent anemia when plasma is increased in volume during pregnancy) approximately 500 mg. In other words, the requirement for iron during pregnancy is slightly less than 1 g, concentrated for the most part in the last half of gestation. The usual diet of pregnant women contains 10 to 15 mg of iron daily, of which only 10 to 20% is absorbed. Thus, dietary iron provides only slightly more than the amount lost through the stool, urine and skin, so that other sources of iron required during pregnancy are needed. Maternal iron, stored principally in bone marrow, is available but frequently in amounts insufficient for the demand. Iron stores in healthy young American women have been found to average approximately 300 mg. Moreover, a significant proportion of women enter pregnancy with no iron stores, presumably as a result of previous pregnancies or menstrual blood loss. Thus, the relationship between pregnancy and iron deficiency states becomes obvious. The relatively small amounts of iron in the diet, coupled with low iron storage, are simply inadequate to meet the greatly increased requirements for this element for the synthesis of maternal and fetal hemoglobin. Thus, anemia is a relatively common complication of pregnancy and other iron deficiency states (ie, depletion of storage iron) virtually universal.

Iron deficiency during pregnancy is largely a preventable problem—preventable by giving iron supplements. In a number of studies in which these supplements were administered during pregnancy, the mean hemoglobin concentration of women has consistently been at least 12 g/100 ml, and iron deficiency anemia has been virtually eliminated.[1] It is therefore recommended[1] that *all* pregnant women receive ferrous iron in doses of 30 to 60 mg daily during the last two trimesters. Continued supplementation for a period of time after delivery, to replenish iron stores, also appears advisable.

There is no direct evidence that formulations other than the simple ferrous salts enhance either absorption or utilization. The same may be said of the various kinds of slow-release iron preparations. In fact, iron absorption from these preparations may be diminished if the iron content is released

too gradually. Several studies have indicated that the absorption of iron is somewhat enhanced by the concomitant administration of ascorbic acid in doses of 100 to 200 mg daily.[6,7]

Augmenting maternal hemoglobin theoretically could constitute a potential hazard by increasing the total content of iron within the body, which would have to be disposed of when blood volume returns to normal during the puerperium. However, such concerns seem groundless in view of the documented extent of blood loss during delivery. Therefore, there is no evidence that oral iron supplements during pregnancy produces a deleterious iron overload.

Folic acid is a coenzyme essential in purine and pyrimidine metabolism and in the synthesis of DNA. Clinical evidence of a folic acid deficiency is usually first evident in tissues that have a rapid cell turnover, notably hematopoiesis. Megaloblastic anemia of pregnancy due to folic acid deficiency, while it is not nearly as common as iron deficiency anemia, is by no means rare in the United States and folic acid supplements are probably a reasonable prophylactic measure, particularly in high-risk patients, such as those in a low socioeconomic class and those with multiple pregnancy and chronic hemolytic anemia. While there seems to be little doubt that supplements, in the recommended dose of 200 to 400 μg daily[1] would diminish the incidence of megaloblastic anemia of pregnancy, whether it would have additional beneficial effects is problematic and related to folate deficiency without anemia and its effect on the course and outcome of pregnancy. Available data with respect to this question are conflicting. Initial studies suggested an association between nonanemic folate deficiency and several different types of pregnancy wastage, particularly abruptio placentae, other causes of bleeding in late pregnancy, abortion and fetal malformations. However, subsequent studies have failed to confirm these observations. Thus, while the place of folate deficiency in megaloblastic anemia of pregnancy is relatively clear, its relationship to other obstetric complications must still be clarified.

A potential hazard of routine folic acid supplements during pregnancy is represented by the patient with unrecognized Addisonian pernicious anemia in whom there could conceivably be a hematologic response without halting the neurologic sequelae of this disease. The risk, however, seems small in view of: a) the low incidence of Addisonian pernicious anemia in the reproductive years and b) the unlikelihood that a dose of folic acid between 200 and 400 μg/day in pregnancy would mask neurologic complications.

Present government regulations require that preparations containing more than 100 μg of folic acid be dispensed on prescription only. Therefore, if a tablet or capsule containing the recommended daily amount of folic acid (200 to 400 μg) is to be used, the patient will require a physician's prescription.

Calcium and Fluoride

Increased calcium is required during pregnancy for fetal bone development. As indicated in Table 1, an increase in daily intake of as much as 50% is recommended during pregnancy and even more is needed during lactation. If calcium intake is inadequate, fetal needs will be met by demineralizing the maternal skeleton. However, dietary sources are capable of providing the amounts of calcium needed during pregnancy and lactation and represent the preferred method. For example, 1 quart of milk contains approximately 1.2 g of calcium. Although calcium (usually in the form of carbonate) is almost always included in prenatal vitamin-mineral preparations, such supplements are unnecessary in routine practice, a point of view supported by studies of dietary intake and urinary excretion.[8]

The documented efficacy of fluoride in

TABLE 2. RECOMMENDED DAILY DIETARY ALLOWANCES (RDA) FOR CALORIC INTAKE (FOOD AND NUTRITION BOARD, NATIONAL ACADEMY OF SCIENCES-NATIONAL RESEARCH COUNCIL)

Female of age	*"Reference" woman*		*RDA (kcal)*	*Pregnancy addition*
	Height (cm)	*Weight (kg)*		
12-14	154	44	2300	
14-16	157	52	2400	
16-18	160	54	2300	200
18-35	163	58	2000	
35-55	160	58	1850	

preventing dental caries has led some to advocate its administration to the pregnant woman to prevent caries in her offspring. Fluoride appears to prevent decay by being incorporated in a calcium-containing complex in dental enamel. In view of the minimal amount of calcium in the pre-eruptive tooth bud of the fetus, it seems unlikely that prenatal fluoride would have any beneficial effect. Indeed, studies in which pregnant women drank either fluoridated or nonfluoridated water failed to show any difference in the incidence of caries in children.[9]

CALORIC INTAKE

Growth is a process that requires energy. Thus, additional calories, above those needed for maintenance, are required during period of growth. Therefore, the Recommended Daily Dietary Allowance (RDA) with respect to calories is age-dependent, since more calories are needed during the younger ages when growth is taking place and physical activity is usually greater. Table 2 lists the RDA of calories for the *reference* woman at various ages, along with the amount to be added for pregnancy.

Physiologic Weight Gain in Pregnancy

The optimum total weight gain in pregnancy has been the subject of much discussion. Published data are difficult to interpret since they are based on retrospective studies of populations that were instructed to aim at certain levels of gain. Therefore, the relationship of these observations to weight gain in the *natural* state is conjectural. Reported mean figures of total weight gain during pregnancy in women who presumably were eating whenever they were hungry are in the range of 10 to 12 kg.[10,11] Another method of approaching the question is to calculate the sum of various identifiable components of maternal weight gain, as illustrated in Table 3. The values in this Table represent approximations of various studies reported

TABLE 3. AVERAGE COMPONENTS OF WEIGHT GAIN IN PREGNANCY

	Cumulative gain (kg) at end of each trimester		
	First	*Second*	*Third*
Fetus	Neglible	1.0	3.4
Placenta	Neglible	0.3	0.6
Amniotic fluid	Neglible	0.4	1.0
(Fetal subtotal)		(1.7)	(5.0)
Increased uterine size	0.3	0.8	1.0
Increased breast size	0.1	0.3	0.5
Increased blood volume	0.3	1.3	1.5
Increased extracellular fluid	0	0	1.5
(Maternal subtotal)	(0.7)	(2.4)	(4.5)
TOTAL GAIN ACCOUNTED FOR	0.7	4.1	9.5

in the literature, chiefly that by Hytten and Leitch.[12] It can be noted that the accountable gain follows a general pattern of minimal accumulation (almost all in the maternal compartment) during the first trimester, approximately 0.3 kg/wk (of which 60% is in the maternal compartment) during the second trimester, and a similar rate (of which 60% is in the fetal compartment) during the last trimester. Thus, weight gain related to products of conception takes place, for the most part, during the latter portion of pregnancy; that attributable to maternal storage is dispersed more evenly throughout the course of gestation.

The total accountable weight gain in Table 3 is somewhat less than the observed average gain, which, as noted above, ranges from 10 to 12 kg. By inference, the difference is in maternal stored fat which, in the human at least, cannot be measured precisely. Indirect methods, such as skin-fold thickness measurements[13] and underwater weighing,[14] support the hypothesis that maternal fat is stored during normal pregnancy, particularly during midpregnancy, and that the rate at which fat is stored diminishes toward zero as term is approached. Other studies,[15] however, failed to indicate maternal fat storage. If fat is stored during pregnancy, it would represent a potential energy source of considerable magnitude; for example, 2 kg of stored fat would be capable of providing 18,000 kcal. Teleologically, the reason for laying down such an energy source is unclear. It may represent a *savings account deposit* in anticipation of requirements for fetal growth during the last trimester or for milk production after birth. In any event, it is a physiologic concomitant of pregnancy and there seems to be no reason to doubt that it has some purpose.

Dietary Restriction

A large number of investigations of the effects of dietary restriction in experimental animals, principally the rat, have been carried out. In general, these studies have indicated that restricting either calories or protein profoundly affects the outcome of pregnancy. The differential effects of caloric and protein restriction are not always apparent, since both usually are limited. An interrelationship exists, even in instances in which only one is restricted, making interpretation difficult; for example, severe caloric restriction with adequate protein intake might well result in utilizing protein for basic energy requirements, in effect producing a protein deficit. Restricted diet during pregnancy generally reduces the number of offspring, the size of individual young and the survival rate. Subsequent growth of these animals is considerably inferior to that of controls, although the difference is lessened in intances in which restriction is limited to pregnancy, as opposed to both pregnancy and lactation. In addition to these morphologic sequelae of dietary restriction during pregnancy, other effects, of perhaps greater significance, have been described. Deficiencies in intellectual function (as indicated by impaired maze-solving ability) and metabolic activities (reduced feed efficiency and decreased glucose tolerance) of a permanent nature have been reported.[16,17] Recent investigations of the cellular aspects of growth have elucidated possible mechanisms by which these observations might be explained. These studies have employed determinations of DNA as an index of cell number and protein—DNA ratio as an index of individual cell size.[18] Assuming that the development of an organ or tissue involve sequential phases of hyperplasia (cell division), hyperplasia and hypertrophy, and finally hypertrophy (cell growth) alone,[19] it seems logical that restricting essential nutrients during cell division would have permanent effects, while restriction during cell growth could conceivably be reversed by later adequate nutrition.

For obvious reasons, it is not possible to conduct human experiments similar to the

animal studies summarized above. Available data, incomplete as they are, support the hypothesis that protein and/or caloric restriction during pregnancy may have profound effects on fetal growth and development. In the first place, weight gain in pregnancy (as one indication of nutrition) shows a strong positive association with birth weight. In most studies of determinants of birth weight, maternal weight gain ranks second only to duration of pregnancy. Secondly, data obtained incidently from certain *natural experiments* in maternal malnutrition are consistent with the hypothesis. In Leningrad, in 1942,[20] and Holland, in 1944,[21] both areas subjected to near-starvation in World War II, there were significant falls in fertility rate, birth weight and perinatal survival. By contrast, Great Britain in World War II,[22] gave pregnant women special treatment under food rationing policies and the stillbirth rate fell by 25%.

To summarize, dietary restriction during pregnancy in experimental animals is associated with profound effects on maternal physiologic adjustments and fetal growth and development. Some of these effects are also observed in humans, who, for various reasons, restrict their diets during pregnancy. It may even be speculated that the common obstetric practice of routinely limiting caloric intake to restrict weight gain during pregnancy could be partially responsible for the number of low birthweight babies which contribute a disproportionate share to infant mortality.

Caloric Excess

Caloric intake in excess of that required for maintaining physiologic functions will result in tissue deposition, primarily as fat, and resultant weight gain. Excessive weight gain during pregnancy has long been held, almost as an article of faith, to be associated with an increased incidence of a variety of obstetric complications, a concept which should be reexamined in view of modern scientific data.

Most interest with respect to excessive weight gain has been in relation to its suspected relationship to toxemia of pregnancy. The concept that a limited caloric intake would protect against toxemia appears to have come from the observation that the incidence of eclampsia declined in areas of Europe subjected to caloric restriction during World War I. The practice of routine caloric restriction in hopes of preventing toxemia was widely adopted by the medical profession and became ingrained in obstetric teaching and practice. To be sure, some retrospective studies indicated a relationship between excessive weight gain and toxemia (as well as other complications of pregnancy), but these reports have usually failed to distinguish between actual tissue accumulation and extracellular fluid retention. Recent and more carefully designed studies[23] failed to indicate a relationship between excessive weight gain, on the basis of fat accumulation, and toxemia of pregnancy. Similarly, the hypothesis that excessive weight gain during pregnancy predisposes to a number of other obstetric complications, such as abortion, dystocia and postpartum hemorrhage, seems to have little supportive evidence. While it is true that a larger maternal weight gain will generally be associated with a larger infant, the increase in birthweight usually is not sufficient to cause mechanical difficulties during delivery. Even if it were, limiting maternal weight gain for the sole purpose of limiting fetal size hardly seems acceptable, in light of modern obstetric concepts.

The relationship of weight gained in excessive amounts during pregnancy to subsequent obesity is of some significance. Obesity is both a common and serious condition in our society, and its long-term detrimental effects on health are undoubted. Additionally, it is likely that certain women become obese because excessive fat had been de-

posited during pregnancy and is not lost so that excess gain in subsequent pregnancies exerts an additive effect. To prevent this chain of events, it is both reasonable and defensible to limit excessive weight gain to some degree. It should be recognized, however, that the sole reason for this limitation is to prevent obesity rather than some complication of pregnancy itself. Moreover, my type of dietary restriction should not be advised without carefully considering all factors. This consideration should take into account the pattern of gain as well as the total amount accumulated. For example, the patient who gains 10 or 12 kg or more during the first half or two-thirds of pregnancy obviously has accumulated a considerable degree of excess fat. But it would be inappropriate and potentially dangerous to impose caloric restrictions severe enough to prevent further weight gain. The preferable method in such a patient would be to permit a modest gain (such as 0.3 kg/wk) during the remainder of pregnancy and make a concerted effort at weight reduction after birth.

It is axiomatic that the first requirement for optimal pregnancy outcome is to enter pregnancy in optimal condition, nutritionally as well as in other ways. This represents the ideal, which, unfortunately, is not always attainable. Difficult management problems are presented by patients who enter pregnancy significantly under- or overweight. The undernourished woman should be instructed carefully and observed closely to ensure adequate nutrition, of which the amount and pattern of weight gain are simple evidence. What of the obese woman? Many have advocated caloric restriction to hold weight constant or perhaps even lose some weight, so that after delivery the patient sustains a net loss.[24] Women themselves often view pregnancy as an opportunity to get rid of unwanted bulk. But, in the light of available evidence, the wisdom of such a course is open to question. Marked caloric restriction, even though adequate protein is provided, may result in utilizing protein for energy, making it unavailable for tissue growth and repair, fetal requirements, etc. In addition, when maternal intake is reduced to a point in which maternal fat stores are catabolized to provide obligatory energy requirements, ketosis and acetonuria result. Recent evidence[25] suggests that maternal acetonuria, from any cause, may be associated with intellectual impairment in the offspring. Pregnancy should not be viewed as a convenient or easy way of correcting undesired maternal obesity.

SODIUM METABOLISM

Few topics in obstetrics have been investigated as extensively as sodium ion and its metabolism in pregnancy. It is a matter of considerable clinical significance and involves such questions as appropriate dietary intake of sodium for the pregnant woman and the advisability of administering agents that promote sodium excretion.

Sodium Metabolism in Pregnancy

During the course of pregnancy, the maternal organism undergoes a remarkable series of physiologic changes aimed at promoting fetal growth and development while at the same time preserving maternal homeostasis. These maternal physiologic adjustments can best be viewed as an integrated system of interrelated components, all having a common purpose. It is, therefore, necessary to think in terms of the system as a whole, rather than of the individual component parts. Perhaps no other example illustrates the importance of this concept as graphically as does sodium metabolism.

Of the many changes in renal function accompanying pregnancy, the increase in glomerular filtration rate (GFR) is particularly pertinent. GFR rises early in pregnancy to levels of 50% above nonpregnant values and is maintained until near term.[26] Sodium loss related to this increase in glomerular

filtration is augmented by the natriuretic effect of progesterone. In the absence of some compensatory mechanism, changes of this magnitude would have disasterous effects on maternal electrolyte balance, particularly with respect to sodium, the principle cation involved. The compensatory mechanism in normal pregnancy is the renin-angiotensin-aldosterone system.[27] To conserve sodium when glomerular filtration is increased, renin is produced by the juxtaglomerular apparatus and, acting on its substrate, causes the release of angiotensin. In turn, angiotensin stimuates the secretion of aldosterone by the adrenal cortex and aldosterone enhances the tubular reabsorption of sodium, returning it to the circulation. Thus, the net effect of this intricately balanced system is to preserve maternal homeostasis. The lengths to which the organism goes to preserve homeostasis are remarkable; plasma aldosterone levels in normal pregnancy are increased several-fold over nonpregnant values, leading to what has been called the "secondary hyperaldosteronism of pregnancy." This somewhat simplistic explanation of sodium dynamics in pregnancy, though based on certain unproved assumptions, nevertheless seems reasonable on the basis of available evidence.

Pike and associates,[28] in recent studies of sodium metabolism in pregnant rats, have described effects on the renin-angiotensin-aldosterone system which are particularly interesting from the standpoint of potential clinical significance. Expanded fluid volume in the rat during normal late pregnancy is associated with histologic findings of increased granulation of the juxtaglomerular cells and hypertrophy and hyperplasia of the zona glomerulosa of the adrenal. Thus, as a result of increased renin and aldosterone secretion, urinary excretion of dietary sodium is decreased and sodium is conserved to provide the additional amounts required for expanded tissue and fluid compartments. When sodium intake is reduced, the increased granulation of the juxtaglomerular cells gives way to decreased granulation, and the hypertrophy and hyperplasia of the zona glomerulosa continues to the point of exhaustion. Thus, it appears that sodium restriction stresses the physiologic mechanism of sodium conservation sufficiently to cause the system to break down, so that blood volume cannot be expanded and hyponatremia develops in fluids and tissue. These experimental observations in the rat, while they cannot be applied unreservedly to the clinical situation, are certainly provocative in their implications with respect to human pregnancy.

Looking at the matter of sodium in pregnancy from a slightly different point of view, it is a simple matter to calculate the "sodium requirement" during pregnancy. Assuming the total weight gain is 11kg, of which 70% is water, the amount of sodium required to maintain isotonicity would be slightly more than 1000 mEq, approximately 25 g Na or 60 g NaCl.

Sodium and Toxemia

Toxemia of pregnancy (ie, acute pre-eclampsia-eclampsia) is a disease which has both plagued and fascinated physicians throughout recorded history. In any situation such as toxemia in which the etiology is unknown and the pathophysiology obscure, there is certain to be a plethora of theories of causation and a multitude of therapeutic regimens, all empiric. Since abnormal sodium retention and edema are essential features of the disease, sodium and toxemia are inextricably related in most minds. The fundamental question, and one to which there are, at present, no conclusive answers, is the all-too-familiar problem of association versus causation.

Because the evidence is overwhelming, both from laboratory and clinical sources, that the toxemic patient retains sodium abnormally,[29] it is not surprising that a restricted sodium intake should be advocated

widely for both the prevention and treatment of the disease. With respect to prevention, supportive evidence is almost totally lacking. Moreover, as outlined above, experimental data suggest that sodium restriction, at least of a marked degree, may be deleterious. Nevertheless, the routine practice of restricting dietary sodium is widespread. In regard to treatment of toxemia by sodium restriction, the evidence is conflicting. While it is usually stated that an increased sodium intake by the toxemic patient is likely to be associated with worsening of the clinical signs of her disease (increased hypertension and proteinuria, convulsions, etc), Dieckmann has described apparent improvement in some instances with sodium administration.[30] Moreover, in at least one study involving high or low sodium diets given to patients with acute toxemia, no difference in clinical course was apparent.[31] This question aside, however, there seems to be general agreement among authorities that regulation of sodium intake in a patient with clear-cut preeclampsia-eclampsia is not a major aspect of treatment.

In evaluating edema, it is very important to differentiate between generalized and dependent or gravitation types. The latter is often seen in late pregnancy as pitting edema of the feet and pretibial areas, particularly late in the day. It is related to a threefold rise in venous pressure in the lower extremities due to pressure of the uterus on the pelvic veins and inferior vena cava, often aggravated by chronic venous insufficiency. When the pressure is relieved, as by assuming the lateral recumbent position, the edema lessens. Thus, it frequently disappears overnight. Generalized edema, on the other hand, is the type usually seen with toxemia. Often the only sign is weight gain, although patients may complain rings are tight or the face appears puffy. Although this type of edema occurs with toxemia, it is also seen in patients who have no other signs of toxemia. Whether generalized edema, in such patients, may be regarded as normal is a controversial point. The bulk of evidence seems to indicate that it is simply an exaggeration of the physiologic processes outlined above, perhaps augmented by decreased oncotic pressure of plasma from the relative hypoalbuminia of pregnancy. However, the view that it represents the earliest sign of developing toxemia cannot be disproved.

The question of normal and abnormal aside, edema is a common occurrence in pregnancy. In studying normal (ie, nontoxemic) patients, Thomson[32] found that edema was reported in nearly half at some time during pregnancy and that in a third of those edema was generalized.

Diuretics in Pregnancy

The vast majority of diuretic agents employed in clinical obstetrics are derivatives of the thiazide group of drugs. These drugs act mainly by inhibiting electrolyte reabsorption in the distal convoluted tubule, thereby promoting urinary excretion of sodium and accompanying water. Potassium excretion is also facilitated and hypokalemia is a recognized complication, particularly with long-term use. Both placental and mammary transfer occur readily.

Clinical interest in diuretics during pregnancy centers about their potential utility in three situations: a) prevention of toxemia, b) treatment of toxemia and c) treatment of edema unassociated with toxemia. With respect to prophylactic use, the evidence seems quite conclusive. In spite of early suggestions that the incidence of toxemia could be lowered by routine thiazide administration, subsequent and more carefully designed double-blind studies have failed to indicate differences between treated and control groups.[33] Diuretic therapy in toxemia is somewhat less clear. Thiazides given to preeclamptic patients will usually (though not always) promote prompt excretion of sodium and water, leading sometimes to severe electrolyte depletion. This symptomatic re-

sponse notwithstanding, the question of whether they have a beneficial effect on the basic disease is unanswered and will probably remain so until such time as the basic pathophysiology of toxemia is understood. On theoretic grounds alone, however, it seems unwise to superimpose diuretic therapy on a patient with profound metabolic alterations (such as diminished plasma volume, renal plasma flow, and glomerular filtration) of toxemia. Thus, while it cannot be disproved that diuretics may be of benefit in the ambulatory management of the gravida with so-called mild (and therefore questionable) preeclampsia, authorities seem to agree that they offer little benefit and are of considerable potential harm in treating full-blown toxemia.

It is in the third potential use—for symptomatic relief of fluid retention—that diuretics are presently used most frequently. As outlined above, some degree of sodium retention is a concomitant of normal pregnancy and, although the exact mechanisms are unclear, seems to result from aldosterone secretion augmented by the salt-retaining effects of estrogen and postural factors. Therefore, the only conceivable rationale for treatment of this basically physiologic process is to provide relief of symptoms. In deciding whether to administer diuretics to such a patient, important factors to be considered include the degree of symptomatology and potential adverse effects. Complications of thiazide are uncommon, but they do occur. Among those reported in infants of women receiving diuretics are hyponatremia[34] and various hemorrhagic disorders, particularly thrombocytopenia.[35] The most frequently observed maternal side-effect is electrolyte imbalance, especially hypokalemia.[36] Thiazides are known to be mildly diabetogenic and impaired glucose tolerance or even the development of frank diabetes has been described.[37] A rare but serious complication of thiazide administration is acute hemorrhagic pancreatitis and several maternal deaths due to this condition have been reported.[38]

Patients with chronic hypertension present particular problems in management. It is customary to treat such patients, when they are not pregnant, with sodium restriction and long-term diuretic therapy. However, Pritchard has found this type of management of pregnant women with chronic hypertensive diesase to cause rather remarkable decreases in creatinine clearance, presumably as a result of impaired renal perfusion.[39] The obvious implication of these observations is that uterine perfusion may be similarly compromised.

SUMMARY OF CLINICAL IMPLICATIONS

1. Vitamin-mineral supplements should not be considered a substitute for sound nutritional counseling for the pregnant or lactating woman. a) The value of routine vitamin supplementation, at least in modern American practice, is dubious, and probably neither beneficial nor harmful (as long as excessive intakes, particularly of vitamins A and D, are avoided). With these considerations in mind, cost factors assume an important role in determining the advisability of prescribing supplements. b) Every pregnant women should receive supplemental ferrous iron, 30 to 60 mg daily, during the second and third trimesters and through lactation or, for the mother who does not breastfeed, 2 or 3 months postpartum. In addition, folic acid (200 to 400 μg daily) should probably be supplemented as prophylaxis against megaloblastic anemia of pregnancy. The clinical significance of preanemic folate deficiency is uncertain at this time. c) Other nutritional substances required during pregnancy can generally be provided most satisfactorily from dietary sources. For example, consumption of 1 quart of milk daily will provide the full recommended daily dietary allowance (RDA) during pregnancy of calcium and Vitamin D and one-half the RDA of protein.

2. An increase in caloric intake of approximately 10% over non-pregnant requirements is needed in normal pregnancy to permit the necessary adjustments in maternal physiology and provide for fetal development. a) An average total weight gain of 10 to 12 kg (22 to 27 lb) appears to be optimal. The normal pattern of gain consists of minimal gain (approximately 1 kg) in the first trimester and accumulation of 0.3 to 0.4 kg/wk during the second and third trimesters. b) There is no scientific justification for *routine* restriction of weight gain to amounts less than those above. Severe caloric restriction is potentially harmful to both mother and fetus. It impairs maternal ability to make the required physiologic adjustments of pregnancy, and it appears to have an adverse effect on fetal growth and development during prenatal life and, perhaps, later as well. c) In evaluating excessive weight gain during pregnancy, it is critical to differentiate between fluid retention and tissue accumulation. There is no convincing evidence that excessive weight gain due to tissue deposition is related to toxemia or to any other obstetric complication. It may, if not lost after delivery, be related to obesity.

3. During the course of normal pregnancy, the maternal organism goes to considerable lengths to promote sodium retention for maternal and fetal needs. Evidence of excessive fluid retention, in the form of edema, may result from an accentuation of these physiologic processes or it may represent an early sign of toxemia, but there is no reason to think that edema due to the first cause will lead to the second. a) The routine restriction of dietary sodium intake in normal pregnancy is of uncertain value. While it is probably ineffective in preventing toxemia, moderate degrees of sodium restriction are probably also relatively harmless because of the efficiency of the renin-angiotensin-aldosterone system in preserving body sodium. However, experimental data in pregnant rats suggest the need for further investigation of safety. In addition to electrolyte imbalance, a possible adverse effect of severe sodium restriction is elimination of essential nutritional components, either because they contain high sodium levels or because limiting salt impairs taste preference. b) Although diuretics are effective in promoting excretion of edema fluid due to both physiologic causes and toxemia, they are of no value in prevention of toxemia and at best are of limited value in treatment. The practice of routinely restricting sodium intake and at the same time administering diuretics on a long-term basis is potentially dangerous and has no place in the management of normal patients. c) Before diuretics are prescribed for the pregnant woman, careful consideration of the benefits to be derived must be weighed against the known maternal and fetal complications. If used, they should be given in short courses of no more than 2 to 4 days with (in the case of thiazides) attention to insure adequate potassium intake. d) The foregoing applies particularly to normal patients. Those with medical illnesses which would be treated with diuretics in the nonpregnant state (eg, chronic hypertensive disease or congestive heart failure), when pregnant, could conceivably benefit from some degree of sodium restriction, and/or appropriate diuretic therapy. Nevertheless, sodium restriction and diuretics in *any* pregnant woman is something to be undertaken only after very careful consideration.

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ROUTINE LABORATORY STUDIES DURING PREGNANCY

ROUTINE LABORATORY TESTS DURING PREGNANCY

All pregnant women should have the following tests performed early in pregnancy: serology, CBC, blood group and Rh, atypical antibody screening, urinalysis, Papanicoloaou smear, and Rubella HI screen.[1,2]

If the patient is Rh negative, the Coombs test, if negative, should be repeated early in the last trimester (26 weeks) and again at about 34 weeks.[3] If the Coombs test is positive the patient should have amniocentesis performed when appropriate.(24-26 weeks.)[3] She may have to be referred to a center where this is available if it cannot be done at the local community.

Hemoglobin and hematacrit levels should be rechecked in the last trimester to assure that the patient is having an adequate iron intake.

If the Rubella titer shows the patient is not immune, the vaccine can safely be administered during the immediate postpartum period in the hospital.[4-5] The patient can than be rechecked at the six-weeks postpartum visit to see if she has developed a positive immunity.[4-5]

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ACOG TECHNICAL BULLETIN

NUMBER 17 — JULY, 1972

MANAGEMENT OF ERYTHROBLASTOSIS

Blood group iso-immunization remains a problem to the practicing obstetrician despite major advances in this field in the past decade. Utilization of a concise algorithm aids in overcoming barriers to the delivery of optimal care to all patients. The use of these flow sheets permits the utilization and interpretation of the techniques currently available for the evaluation of the severity of sensitization and assists in determining the proper time for delivery of the affected infant.

The following graphs are largely self-explanatory. Certain points, however, require amplification.

As indicated on Flow Sheet #1, all prenatal patients require a blood type and Rh determination. Sensitization to blood group antigens may have occurred in Rh+ patients, especially in those who have had previous blood transfusions, unexplained fetal losses or previous jaundiced infants. In this Rh+ high-risk population, identified by careful study of pertinent history, and in all Rh— patients, prenatal atypical antibody screening should be performed on the patient's blood at the initial prenatal visit. If antibodies are present, the inciting antigen should be determined and, when possible, the father's zygosity determined. Iso-immunization to the Rh system of antigens (D, C,c, E,e) remains the most common form of sensitization. Significant hemolytic disease of the newborn has also been caused by antibodies to minor red blood cell antigens such as K,k, K_p^b, M, F_y^a, J_k^a, S, V_w and U. Sensitization to these antigens produces intrauterine disease similar to Rh sensitization and should be managed in an analogous manner. In all patients at risk, whether Rh+ or Rh—, antibody screening should be repeated at the 28th and 36th week of gestation. Prenatal screening for ABO incompatibilty is not currently available.

Antibody titers should be performed in those patients in whom an antibody has been identified that is known to cause erythroblastosis. The level of the antibody titer provides a basis for determining if the patient can be followed by titers or if amniocentesis is needed. Repeat antibody titers are obtained at 4 week intervals as long as the titers remain below the minimum critical level, defined here as an indirect Coombs titer of less than 1:8. Delivery is accomplished between the 36th and 38th week of gestation as long as the minimum critical level is not exceeded. Symptoms of deterioration of the fetus such as decreased fetal activity, polyhydramnios or a preeclamptic-like picture may necessitate earlier intervention.

It should be noted that the minimum critical antibody level is defined as that antibody titer below which severe involvement of infants does not occur. The experience of an individual laboratory may indicate a different minimal critical antibody level due to differences in technique or interpretation. If such specific laboratory data is available, use that titer level suggested by the laboratory performing the tests, otherwise a titer of 1:8 is recommended.

In those women in whom the antibody titer equals or exceeds the minimal critical level, i.e. 1:8 or greater, amniotic fluid analysis is utilized to evaluate the fetal condition. The technique of amniocentesis has been described in ACOG Technical Bulletin No. 8. This can be performed between the 20th and 36th week of gestation with little risk to mother or fetus. Spectrophotometric analysis of the amniotic fluid will yield a curve from which the optical density difference ($\triangle$ O.D.) at 450 mμ is calculated. The $\triangle$ O.D. is simply the deviation of the actual spectrophotometric curve from that of normal amniotic fluid at 450 mμ. It is caused by bilirubinoid pig-

ments present in the amniotic fluid. Increasing values of △ O.D. indicate more blood breakdown products in the amniotic fluid and a more severe hemolytic process in the fetus.

The interpretation of any given value of △ O.D. must take into consideration the gestational week when the fluid sample was obtained. The △ O.D. value at the appropriate gestational week is plotted on a Liley gram-like graph and from this the zone is obtained. (See example graph). Zone 1 indicates minimal fetal involvement, Zone 2 moderate to severe involvement and Zone 3 impending fetal demise. The appropriate management and time of delivery according to these Zones is outlined on Flow Sheet #2.

It must be emphasized that single amniotic fluid analyses are of little prognostic significance and that accurate evaluation of the fetal condition can only be determined by serial examinations of the amniotic fluid. It is further recommended that if the △ O.D. reaches Zone 2 levels, subsequent amniocentesis must be performed on a weekly basis, even if the △ O.D. subsequently falls into Zone 1 category. Caution should be exercised in interpreting the △ O.D. of amniotic fluid when the pregnant woman is jaundiced, a drug addict, or using hepatotoxic drugs since recent evidence has shown elevated △ O.D. unrelated to erythroblastosis in these situations. The initiation of amniocentesis after the 36th week of gestation is not recommended. Significant elevations in antibody titers detected this late in pregnancy warrant delivery of the infant rather than further diagnostic studies.

The sensitized patient continues to represent a problem optimally managed by the close cooperation of the laboratory, pediatric and obstetric personnel involved. The obstetrician should seek early consultation with his pediatric colleagues for all pregnancies in which he anticipates an infant with hemolytic disease, so that they may be properly prepared for the immediate care of the newborn. He should consider prescribing phenobarbital for the sensitized prenatal patient during the two weeks preceding delivery to activate enzymes for the conjugation of bilirubin by the fetal liver. He likewise remains responsible for obtaining adequate cord blood specimens for the appropriate laboratory studies on all infants at risk. The obstetrician should evaluate all Rh negative, unsensitized patients for Rho (D) Immune Globulin (Human) therapy as outlined in the ACOG Technical Bulletin #13.

These flow sheets are the basis for effectively managing Rh and other blood group sensitized pregnancies utilizing currently available techniques. They should be augmented by sound clinical judgement.

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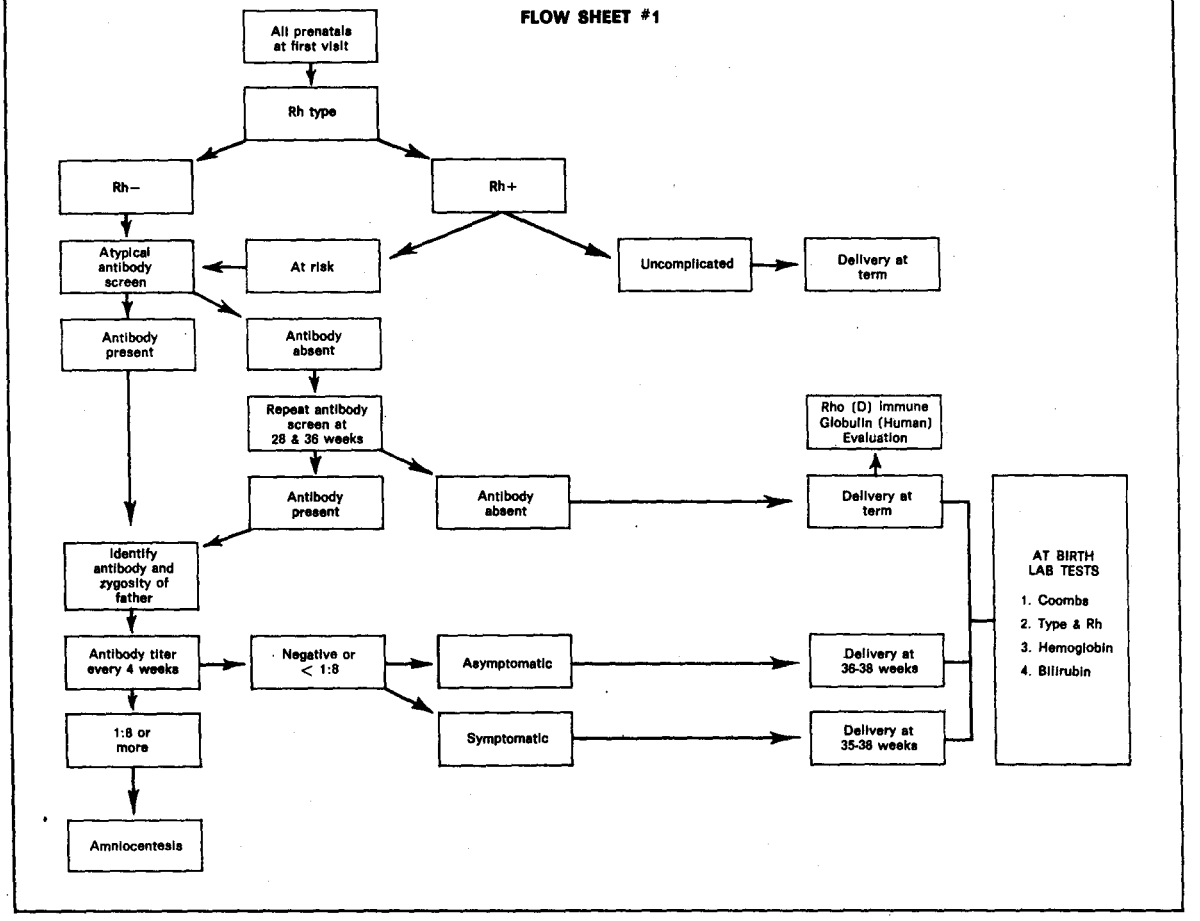
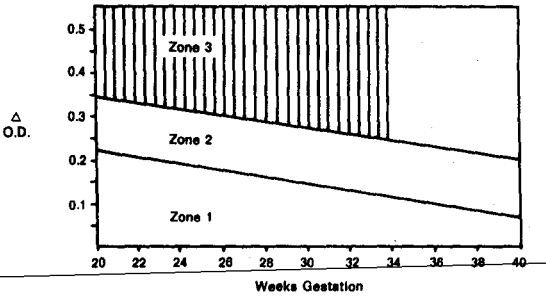
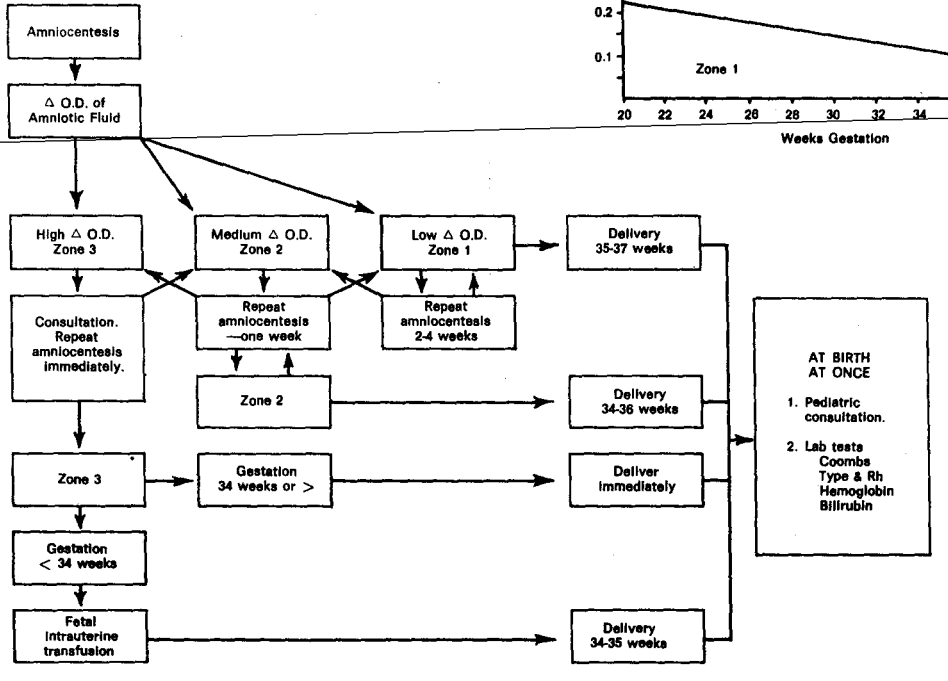
FLOW SHEET #1

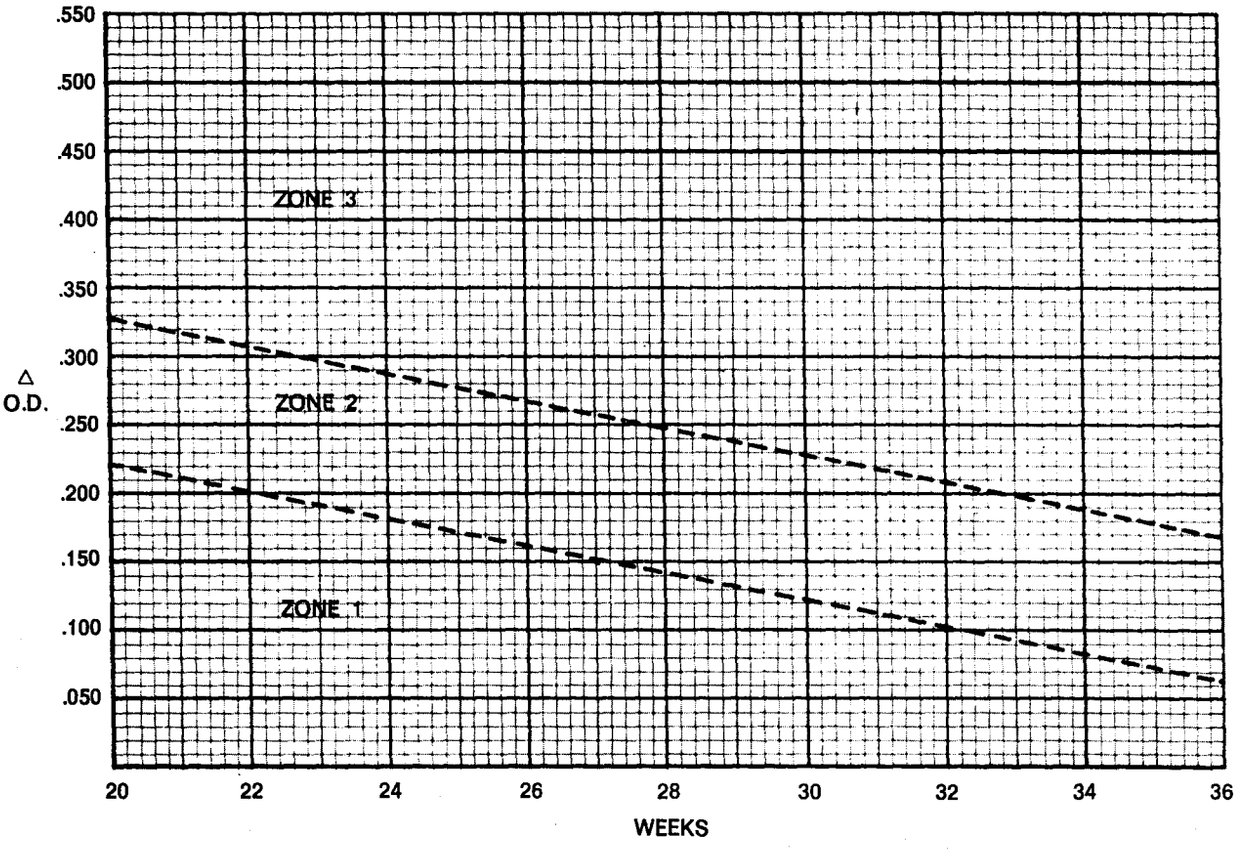


FLOW SHEET #2





Example Graph



This Technical Bulletin is prepared (with consultation from appropriate experts) by the Committee on Technical Bulletins of The American College of Obstetricians and Gynecologists. It describes methods and techniques of clinical practice that are currently acceptable and used by recognized authorities. However, it does not represent official policy or recommendations of The American College of Obstetricians and Gynecologists. Its publication should not be construed as excluding other acceptable methods of handling similar problems.

THE AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGISTS

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about any symptoms that they thought were attributable to vaccination. At that time, a postimmunization blood sample was drawn. At the eight-week appointment at the Well Baby Clinic, they were questioned about infant morbidity, and a blood sample was drawn from the infant by one of us (CSW).

A total of 236 patients (12.5%) of the total number delivered, were seronegative for rubella antibody. Although this figure is significantly lower than the 17% reported by Bowes[13] when a similar population was screened at our institution by the indirect fluorescent antibody method, the reason for this difference is not clear. Of the total, 17 (7%), refused vaccination for a variety of reasons. (This number included two patients who had been exposed to rubella during pregnancy.) Another 18 (7.6%) were vaccinated but failed to keep their postpartum appointment and were lost to followup. This left 201 patients available for study.

RESULTS

Of the 201 patients in whom paired sera were obtained, 11 were screened erroneously; they actually had antibodies to rubella at time of delivery. This was not known, however, until after the patients had been vaccinated. In none of these patients, was there a rise in titer, but arthralgias were observed in one. These patients were eliminated from the study. The remaining 190 patients ranged in age from 16 to 40. Ninety-four (50%) were nulliparous; the others ranged in parity from 1 to 7.

In 174 patients (91%), seroconversion to positive antibody levels occurred. The magnitude of their response is shown by the postimmunization titer in Table 1. Although these patients have not been challenged, studies in nonpuerperal vaccinees indicate that antibody levels are well maintained. Among the 16 patients who failed to convert to positive antibody levels, three had unequivocal vaccination-related side effects.

Of the total number of infants delivered, 88 (46.3%) were seen in followup. No seroconversion was demonstrated in this group and, in no instance, was any morbidity attributable to the mother's vaccination. This seems to confirm the fact that the attenuated virus is noncommunicable. When possible, the infants were followed to six months of age, and another antibody titer was drawn; however, the small number studied does not permit any conclusion.

TABLE 1. ANTIBODY RESPONSE TO VACCINATION WITH HPV-77 STRAIN RUBELLA VIRUS

Post-immunization titer	*No. of patients*	*Percent*
1:8 (no conversion)	16	8.4
1:16	22	11.5
1:32	42	22.1
1:64	55	28.9
1:128	33	17.3
1:256	17	8.9
1:512	4	2.1
1:1024	1	0.5

An interesting facet of vaccination was the morbidity observed. A total of 42 patients (22.1%) had symptoms, occurring either singly or in combination 10 to 20 days after vaccination, that were referable to vaccination. Although this figure approaches the morbidity reported in nonpuerperal vaccinees, it is much higher than that reported in the small group of postpartum patients studied by Boue *et al.*[7] By far, the most common symptoms were arthralgias/arthritis, which occurred in 17.8% (Table 2). In all but two patients, these symptoms were transient and, in most instances, were confined to the small joints of the hand. The two exceptions, both physicians' wives, developed polyarthralgias 10 and 12 days after vaccination, respectively. Within 72 hours, most of the joint pains had disappeared, but both patients had persistent pain, effusion and synovial thickening in the metacarpophalangeal joints of one hand. These symptoms persisted for six and eight weeks, respectively. Both have been screened, with nega-

TABLE 2. SIDE EFFECTS RELATED TO VACCINATION WITH HPV-77 STRAIN RUBELLA VIRUS

Symptom	*No. of patients*	*Percent*
Arthralgia arthritis	34	17.8
Rash	18	9.4
Postauricular swelling	4	2.1
Fever	4	2.1
Headache	2	1.0
Nausea and vomiting	2	1.0
Sore throat	2	1.0
Chills	1	0.5
Anorexia	1	0.5
Diarrhea	1	0.5
Photophobia	1	0.5

tive results, for the presence of underlying rheumatic disease.

Weibel *et al*[18] reported a lower incidence of joint symptoms among nonpuerperal vaccinees who used oral contraceptives. Of the 34 patients in our series who developed arthralgias/arthritis, 44.9% were taking oral contraceptives and 55.1% employed some other form of contraception, a statistically insignificant difference.

DISCUSSION

Adult females who are susceptible to rubella can be vaccinated safely and effectively with a live, attenuated vaccine during the immediate puerperium. This appears to be the ideal time for vaccination, since women accept contraception readily and since the risk of inadvertently vaccinating a pregnant woman whose contraception has failed is low.

The HPV-77 strain of rubella vaccine is effective during the immediate postpartum period, with seroconversion occurring in 91% of those vaccinated, indicating no suppression of the immune response. Studies in infants of vaccinees indicate that the attenuated virus is nontransmissible, and that nursing mothers need not be excluded from an immunization program.

Although morbidity associated with vaccination is higher with the HPV-77 strain than with the Cendehill strain,[10] particularly with regard to joint manifestations, side effects were transient and in no way disabling in almost all instances.

CONCLUSIONS

Mass immunization of children will almost certainly lessen the danger of contracting rubella during pregnancy and will minimize the ravages of congenital rubella. Until such time, postpartum immunization should offer protection to many susceptibles.

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THE HIGH-RISK OBSTETRICAL PATIENT

Certain existing factors and complications, either medical or obstetrical, place a percentage of obstetrical patients in the high-risk category. The high-risk pregnancy is defined as one in which the mother has a serious health problem and the fetus has a significantly increased chance of death, either before or after birth, or of later disability.[1] Ideally, these patients should be delivered in centers with optimal facilities for maternal care (optimal anesthesia, blood bank, and electronic fetal monitoring services) as well as an intensive care neonatal nursery to receive the high-risk infant. The best transport facility for the infant is the uterus, and delivering the mother where all services are available will eliminate the necessity of transporting a sick neonate by ambulance. after birth. Time and problems encountered in transport often are a critical factor in the final outcome for the infant.[2-4]

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A COMPREHENSIVE APPRAISAL OF HIGH RISK OBSTETRICS

A high-priority product of this country should be a wanted child that is well-born and healthy. Thus, emphasis upon health must begin soon after conception. Major emphasis must be placed on the provision of adequate maternity care and family planning as initial steps necessary to produce this well-born child.

High-risk obstetrical patients (of all socio-economic categories) should be identified early in pregnancy so that they may receive specialized care. An ideal, not always attainable, would be to identify such patients before they conceive. Units (preferably regional) for such high risk obstetric patients and their newborns should be developed. Admittedly, these patients require more care which is expensive. Yet, the life expectancy and value of mothers and their newborns to our community and state is greater than that of any other age group. When the high risk status of obstetrical patients is medical in origin, additional funds are required for specialist care, increased laboratory work and greater utilization of existing, and provision for newly acquired, hospital facilities.

Who are the high risk obstetrical patients? How can they be identified in sufficient time to have a salutary effect on their infant mortality and morbidity? Before listing the specific risk factors, it must be recalled that some 90% of all perinatal deaths are due to four factors: prematurity, anoxia of various causes, birth injuries and fetal abnormalities.

Less than 2% of all births in this country contribute 1/2 of the neonatla deaths and 1/3 of fetal deaths - that is, those infants whose birth weight is less than 1500 grams. It is possible to reduce the incidence of prematurity by regular prenatal visits, careful scrutiny of past obstetrical history, patient education, and improved management of the obstetrical complications that lead to premature labor. The effects of anoxia and birth trauma can be reduced with improved obstetrical management of toxemia, prolonged labor, breech and abnormal presentations, difficult forceps deliveries and by better application of newborn resuscitation techniques.

Specifically, the following patients deserve the best possible prenatal care to minimize undesirable perinatal statistics.

THE TEEN-AGE PATIENT - notorious for increased incidence of toxemia, prolonged labor, prematurity, perinatal mortality, and further compromised more recently by the higher incidence of venereal disease and illicit drug usage.

THE HIGHLY PAROUS INDIVIDUAL - contributes disproportionately to the complications of hypertension and superimposed toxemia, third trimester bleeding, abnormal presentation, prematurity and (after the age of 35 or 40 years) to Mongolism.

THE PATIENT WITH TOXEMIA OR CHRONIC RENAL DISEASE AND HYPERTENSIVE DISORDERS - there is a distinct challenge, once diagnosed, to manage by careful sedation, appropriate anesthesia with prevention of convulsions during labor and by provisions for preterm delivery because of placental dysfunction.

THE PREGNANT DIABETIC - improved medical control of diabetes will provide a reservoir of pregnant patients. There is no better example of a patient whose problems demand expert liaison between obstetrician and internist, and ultimately the pediatrician for care of her fragile infant.

LABOR PROBLEMS AND FETAL DISTRESS - anticipation of these problems in certain obstetrical patients will provide for awareness of progress in labor, prompt recognition of dysfunctional labor aided by the ability for fetal heart rate monitoring.

THIRD TRIMESTER BLEEDING - diagnostic procedures, optimal blood banking facilities and provisions for optimal anesthesia will minimize the maleffects on the fetus as well as the mother.

ERYTHROBLASTOSIS - although immune prophylaxis will reduce the incidence of this disease, provisions still must be provided for amniocentesis with fluid analysis as well as intrauterine transfusions when indicated, and of course, exchange newborn transfusions.

CYTOGENETICS - provisions for amniocentesis and studies for sex determination, chromosomal analysis and opportunity for biochemical analysis must be available to patients with predisposition to certain fetal abnormalities.

MULTIPLE GESTATION - care for the potential complications of premature labor, third trimester bleeding, increased incidence of toxemia, and labor problems with optimal anesthesia will minimize the hazard (particularly to the second twin) of such gestations.

BREECH PRESENTATIONS - alertness to labor problems and umbilical cord prolapse, the opportunity of monitoring labor and provision of proper analgesia and anesthesia will reduce the inherent hazards to such babies.

CONCLUSIONS: After the high risk obstetrical patient is identified and provisions made for her obstetrical care, there are additional needs.

(1) Through education, the high risk obstetrical patient must be motivated to obtain the necessary prenatal care.

(2) At the local level, leaders in the practice of Obstetrics and Gynecology must be identified and provided with knowledge and techniques regarding current diagnostic and therapeutic procedures. They should be able to generate a feeling of cooperation and collaboration with pediatricians as well as internists and family physicians within that community.

(3) In addition to physicians, obstetrical nurses must be made aware of the currently available techniques and equipment for improved perinatal salvage in this sizeable group of obstetrical patients.

(4) Provisions for family planning - reversible birth control methods as well as availability of sterilization must be provided.

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TOXEMIA OF PREGNANCY

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DEFINITION

PREECLAMPSIA-ECLAMPSIA is a disease peculiar to pregnancy. Thousands of pages of print and many investigations have been devoted to toxemia of pregnancy. In spite of this, its etiology remains obscure. We must be content with a description of the pathophysiology of toxemia and continue empiric therapy until the cause of the disease is known.

The terminology of toxemia of pregnancy leads to confusion and needs revision in view of recent electronmicroscopy observations of renal biopsies. This disorder could better be classified as "acute and chronic hypertension during pregnancy," using the subclassifications of the current nomenclature only when specific diagnostic tools have been employed to pinpoint a "definitive" diagnosis. The current nomenclature is based upon the American Committee on Maternal Welfare classification and is as follows:

I. Acute Toxemia of Pregnancy (after 24th week)

- A. Preeclampsia
  1. Mild
  2. Severe
- B. Eclampsia (Convulsions or coma, usually both, when associated with hypertension, proteinuria or edema)

II. Chronic Hypertensive Vascular Disease With Pregnancy

- A. Without superimposed acute toxemia (i.e., no worsening of hypertension or the development of proteinuria)
  1. Hypertension known to antedate pregnancy
  2. Hypertension discovered in pregnancy (before 24th week)
- B. With superimposed acute toxemia

III. Unclassified Toxemia (Insufficient information to differentiate diagnoses)

This paper will deal primarily with the entity termed "preeclampsia-eclampsia," which is defined as acute hypertension after the 24th week of pregnancy. Chronic hypertensive disease with pregnancy, however, is diagnosed primarily by exclusion or is diagnosed by continued observation postpartum.

Mild Preeclampsia: The criterion for classification as mild preeclampsia is the development of one or more of the following after the 24th week of pregnancy:

— A systolic blood pressure of at least 140 mm of mercury or a rise of 30 mm or more above the usual level.

— A diastolic pressure of 90 mm or more or a rise of 15 mm above the usual level.

— Proteinuria

— Edema of the face or hands.

In general, the following chronological sequence occurs: First, edema; next, an increase in blood pressure; last, the appearance of proteinuria. Edema is almost always present and without fluid retention the diagnosis should be questioned.

The increment change in blood pressure should be recorded on at least two occasions

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six hours apart. This eliminates the problem of anxiety so often seen in the patient as an outpatient whose blood pressure is normal when she is later hospitalized. Proteinuria, as a general rule, should be persistent and present in a clean catch urine on two or more successive days. One or more of these findings must be present; thus a diagnosis of mild preeclampsia can be made only if edema of the hands and face is present. However, this should be made with some reservation unless there is also a change in blood pressure.

An important point of consideration relating to blood pressure is the decrease in peripheral resistance in the mid-trimester of pregnancy. A 90/50 blood pressure reading may be observed during the mid-trimester, which means that an increment of 30 mm systolic or 15 mm diastolic would result in a "normal" blood pressure of less than 140/90. If such a patient has edema, the possibility of preeclampsia must be considered. The absolute values in blood pressure are not necessarily as important as a change from baseline pressures, using the mid-trimester blood pressure readings for comparison.

Severe Preeclampsia: Severe preeclampsia is diagnosed if one of the following signs or symptoms is present:

1. Systolic blood pressure of 160 or a diastolic of at least 110 on two occasions, six hours apart, with the patient at bed rest.
2. Proteinuria of 5 gm or more in 24 hours which, for practical purposes, is a 3+ to 4+ urinary protein on quantitative examination.
3. Oliguria of less than 400 cc in 24 hours.
4. Cerebral or visual disturbances.
5. Pulmonary edema or cyanosis.

Pregnancy is a hypertensogenic agent and may unmask latent hypertension. This phenomenon should not be overlooked, in the primigravida, even though seen more commonly in the multigravida. The single most important criteria in trying to establish a diagnosis of chronic hypertensive vascular disease during pregnancy is the knowledge of nonpregnant blood pressure values. Additional helpful information is a strong family history of hypertension. The long-term follow-up of patients who manifest hypertension during pregnancy is an available means of retrospectively diagnosing whether or not the patient truly had preeclampsia or whether she had chronic vascular disease. This also holds true for chronic renal disease which may be difficult to diagnose during pregnancy. The clinical diagnosis of problems relating to edema, hypertension and proteinuria during pregnancy, when compared to the actual "definitive" diagnosis, are now known to involve greater diagnostic errors than had been previously thought. This does not imply that the hypertensive disorders during pregnancy should not be classified, but does mean that clinical pitfalls exist. The important consideration, of course, is whether or not active intervention of pregnancy should be undertaken if a specific diagnosis is made. Secondly, consideration must be given to the patient's future childbearing.

Etiology

There are as many theories on the etiology of toxemia as there are scholars of the disease. It can probably best be described as a disease of theories, and today the etiology is still unknown.

When Chicago Lying-in Hospital was built in 1929, Dr. Joseph Bolivar DeLee wanted to acknowledge certain past contributions to reproduction. At the top of a collonade of a portico connecting two wings of the hospital shields were inscribed with chosen names of great scientists in reproduction. (Fig. 1.) One shield remains empty and is reserved for the name of the person who discovers the etiology of toxemia of pregnancy. The recognition of the importance of toxemia still holds today.

The following etiologies have been proposed concerning toxemia of pregnancy [1]. The last two etiologic theories (16 and 17) are listed in *Williams Textbook of Obstetrics*, edited by Drs. Eastman and Hell-

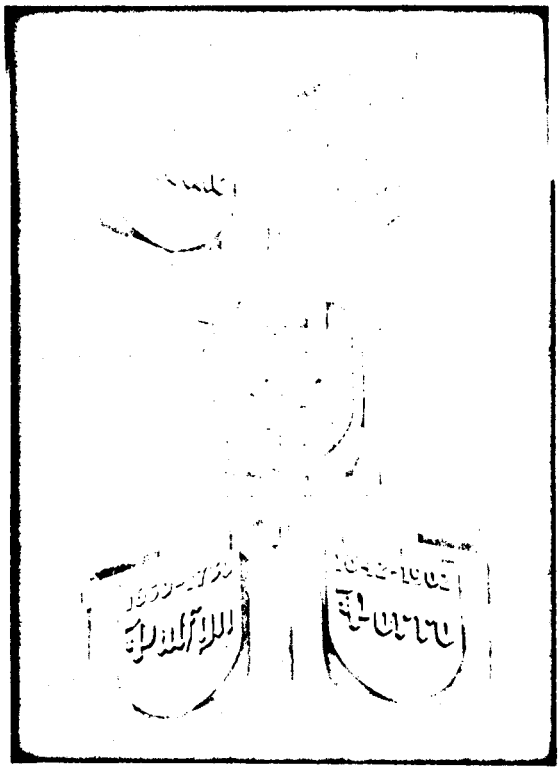

FIG. 1. Composite of shields on Chicago Lying-In Hospital with inscribed names of great scientists in reproduction. The empty shield is reserved for the name of the person who discovers the etiology of toxemia of pregnancy. (From: Zuspan. *Davis' Gynecology and Obstetrics*. Hagerstown, Md., Hoeber Medical Division, 1968.)

man [2]. The author of each etiology or the individual who was the champion of the cause is listed in parentheses and are as follows:

1. Autointoxication (Riviere)
2. Fetal elements (Veit; Hull & Rohdenberg)
3. Fetal metabolic products (Fehling & Dienst)
4. Placental theories (Young)
5. Infectious theory (Delore & Rodet)
6. Endocrines (Lang; Vassale & Sanfrongini; Williams & Wallis; Hofbauer; Kustner; Hoffmann & Anselmino)
7. Biologic reactions (Schmorl, Veit & others)
8. Mammary (Sellheim)
9. Diet (Tweedy)
10. Renal theory (Volhard)
11. Edema theory (Traube & Rosenstein)
12. Vascular theory (Haselhorst)
13. Nervous theory (Elwyn)
14. Hepatic theory (Jurgens)
15. Oxygen deficiency (Halbertsma & Rodenaker)
16. Disorders of blood coagulation (Page; Schneider; McKay)
17. Uterine ischemia (Becker; Dexter & Weiss; Page; Dieckmann and others)

Certain factors are apparent and must be explained in any etiologic theory of toxemia. These factors are consistently observed in those patients with acute hypertension after the 24th week of pregnancy:

1. Primarily a disease of the primigravida.
2. Predisposing influence: multiple pregnancy, hydatidiform mole and hydramnios.
3. Seen more often in certain regions of the world; more particularly in the southeastern region of the United States.
4. More frequent among indigent than private patients.
5. More prevalent as term approaches.
6. Disappears once uterus is empty.

EPIDEMIOLOGY

Fortunately, most pregnant patients do not develop toxemia of pregnancy. The incidence of preeclampsia throughout the United States is estimated to be 5 to 7 per cent of all pregnancies. This incidence is altered by the following: geographic location, number of primigravida patients, number of indigent patients, whether the hospital is a general hospital or referral hospital, method of reporting, etc. Because of these multiple factors we do not have good statistics on incidence for large segments of our country. The problem is further compounded by the fact that erroneous diagnoses are often made in this disease syn-

drome, and the recording of statistics is customarily an inpatient hospital procedure rather than an outpatient responsibility. If all babies in the United States were delivered in hospitals, the chance of errors in reporting would be less. Although 98 per cent of women do deliver in hospitals, the incidence of home deliveries may be as great as 10 per cent or more in some states, especially in the southeast United States. This makes our current statistics unreliable.

Doll and Hanington [3], in their international survey of eclampsia, cite the incidence in different countries as varying between 1.2 and 2.6 per 1000 deliveries. Again, the incidence of eclampsia to deliveries is not a uniform way of reporting disease, unless this is done by state or country. An erroneous figure can be obtained if a hospital is a referral hospital, such as the Eugene Talmage Memorial Hospital at the Medical College of Georgia, where the incidence of eclampsia is 1:100 deliveries.

Healthy parents and a healthy pregnancy usually produce a healthy infant. If the patient is unfortunate enough to develop toxemia of pregnancy, the fetus should become the focal point of concern. Her pregnancy then becomes statistically classified as a potential contributor to one of the four major causes of maternal mortality. Toxemia of pregnancy is frequently associated with prematurity, the greatest single cause of fetal loss in this country. Incidence of fetal and maternal mortality and morbidity is in direct proportion to the severity of the toxemia. The uncorrected fetal mortality figures for patients who have eclampsia has changed little since the 18th century and is between 25 per cent and 35 per cent [4]. What is not known is the number of babies who are born alive from toxemic mothers but have a decreased total potential.

Studies have clearly demonstrated that there is an increased number of stillbirths and neonatal deaths in pregnancies complicated by severe toxemia. [5, 6, 7, 8] Among others, Claireaux [9] has been able to demonstrate that mild and severe pre-eclampsia results in a perinatal mortality of 8 to 9 per cent, whereas eclampsia results in a three-fold increase of perinatal loss to 24 per cent. The overall perinatal mortality per 1000 total births in patients who have toxemia of pregnancy is increased at least two to three times over the expected perinatal mortality. Thus, even though the disease occurs in only a small number of patients, its presence constitutes a major problem, primarily for the fetus but also for the mother.

It has been estimated that in the United States at least 100 deaths per day occur in fetuses whose mothers develop toxemia of pregnancy [10]. The impact of this loss of life can perhaps be better understood when it is noted that it can be equated to the annual death toll in automobile accidents or from violent means, such as murder. This then should make toxemia a major concern for for all involved in reproduction, for this potential loss of life is from a disease which is essentially preventable. Certainly the severe forms of the disease are completely preventable. The time has come when a society as affluent as ours should not have a disease such as toxemia of pregnancy.

Pathogenesis

It should be repeatedly emphasized that the etiology of toxemia of pregnancy is unknown; thus the pathogenesis cannot be specifically described.

The following homeostatic relationship (Figures 2 and 3) is seen in the normal pregnant patient. This in some way becomes disrupted in toxemia of pregnancy. The placenta produces an increased amount of progesterone which contributes to a minor amount of sodium loss, since progesterone is known to be a very mild type of diuretic. This sodium loss contracts the blood volume in the vascular compartment which, in turn, activates the stretch receptor afferent arteriole and the juxtaglomerular apparatus to produce renin. Renin activates Alpha^2 globulin in the blood, which is converted to angiotensin I and, by another enzyme, to angiotensin II. The angiotensin, in turn, acts upon the zona glomerulosa of the adrenal gland to produce an increased amount of aldosterone as a compensatory

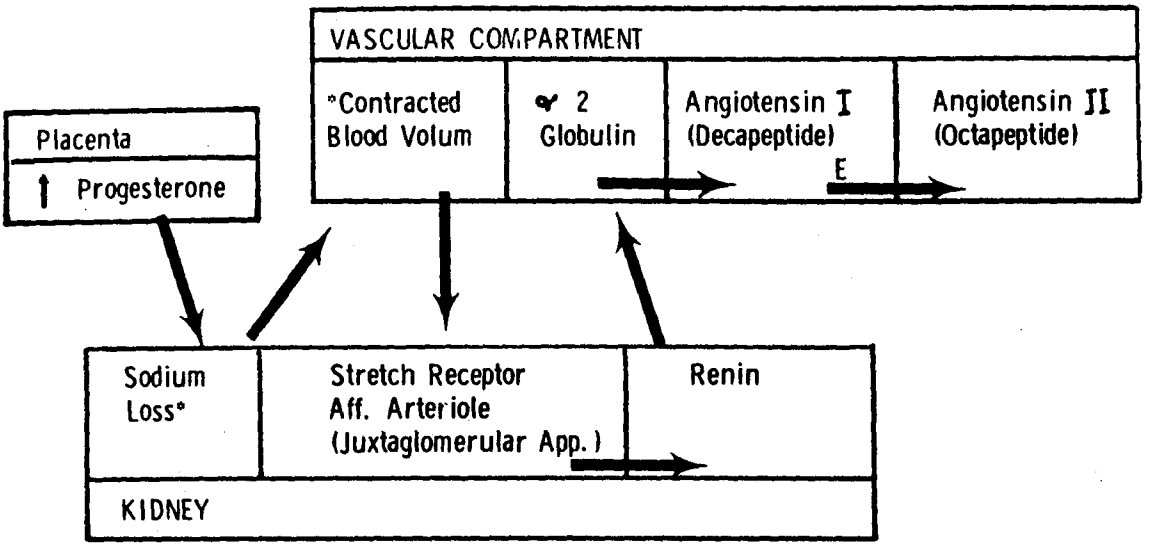

FIG. 2. Homeostatic relationship that exists in normal pregnancy. (From: Zuspan. *Davis' Gynecology and Obstetrics.* Hagerstown, Mr., Hoeber Medical Division, 1968.)

mechanism. This increases the reabsorption of sodium from the renal tubule and subsequently re-expands the water in the vascular compartment thus decreasing the stimulus on the stretch receptors which decreases renin production. This is a normal homeostatic mechanism. In toxemia of pregnancy this homeostatic mechanism is still in force but there are alterations in the degree of production from one to another, eventually resulting in disturbed pathophysiology. The disturbed pathophysiology is (a) a disease of arterioles, (b) a compromised metabolic function, (c) an increased central nervous system irritability, (d) a decrease in renal function, and (e) a catabolic disease.

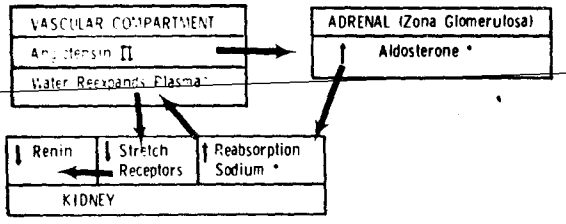

FIG. 3. Homeostatic relationship that exists in normal pregnancy. (From: Zuspan. *Davis' Gynecology and Obstetrics.* Hagerstown, Md., Hoeber Medical Division, 1968.)

The scheme in Figure 4 attempts to depict the development of situations which occur in normal pregnancy (A), are exacerbated in early preeclampsia (B), and ultimately become severe preeclampsia and eclampsia (C). It is noted that the increased level of steroid hormones in the pregnant patient, along with sodium intake, contributes to an increased retention of sodium which is seen in the pregnant patient [11, 12]. It has been observed that there is an increased aldosterone secretion rate in the pregnant patient [13, 14, 15]. A positive sodium balance is seen throughout pregnancy and is associated with an increased retention of fluid in the pregnant patient [16]. This loop is seen in the normal pregnant patient. The patient with toxemia of pregnancy may develop relative uterine ischemia, although this phenomenom has not yet been documented. A decrease in intracellular potassium [17] infers an increase in intracellular sodium in severe toxemia. Renin activation occurs; the increased intracellular sodium changes the vascular reactivity and sensitizes the blood vessels to pressor agents. This increase in vascular reactivity is observed if angiotensin, norepinephrine or epinephrine are given to the toxemic patient [18, 19, 20]. Ultimately, the blood pressure increases in preeclampsia and intravascular fibrin de-

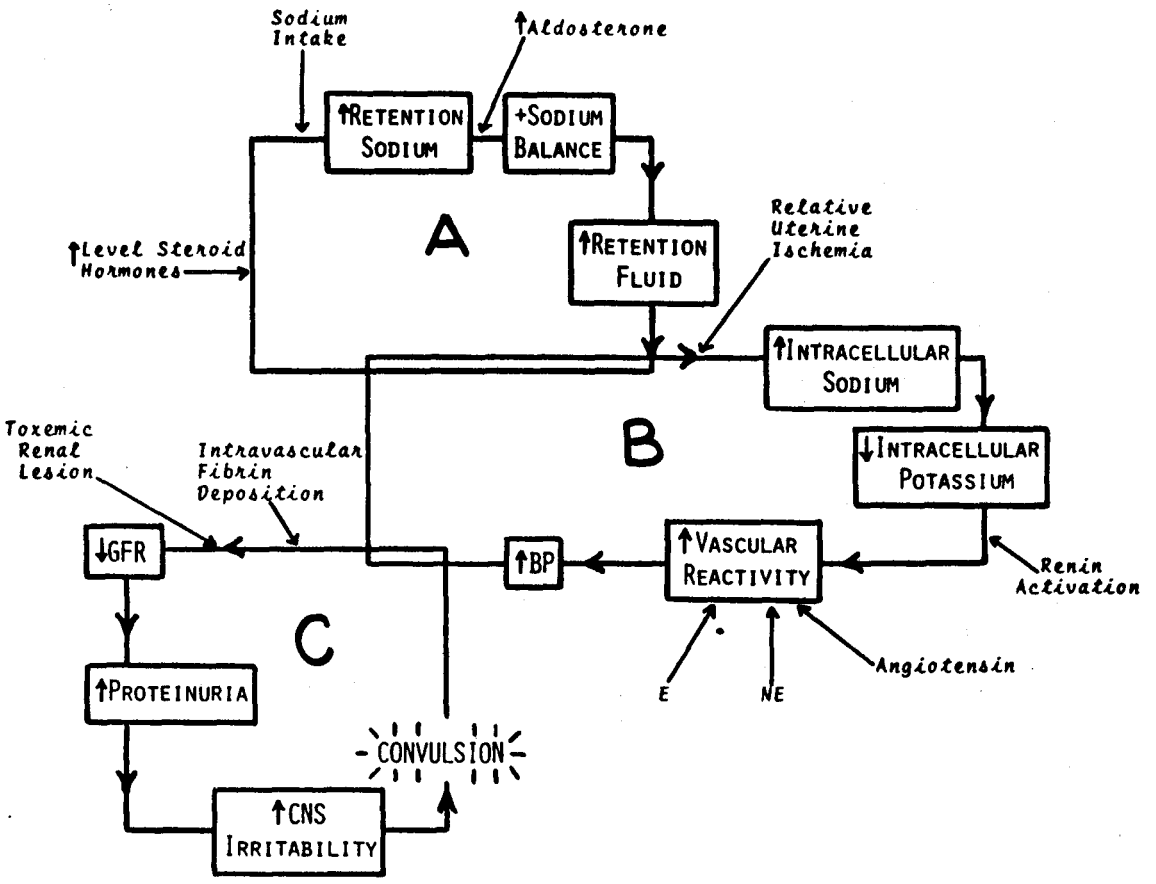

FIG. 4. Pathogenesis of preeclampsia. Loop A, Normal pregnancy; Loop B, Preeclampsia; Loop C. Severe preeclampsia-eclampsia. (From: Zuspan. *Davis' Gynecology and Obstetrics.* Hagerstown, Md., Hoeber Medical Division, 1968.)

position may occur. The toxemic renal lesion of glomerular endotheliosis could account for the decrease in glomerular filtration rate and further exacerbation of proteinuria. Increased central nervous system activity ensues as the toxemia worsens and eventually leads to a convulsion. What triggers the normal relationship as seen in Loop A to the abnormal relationships in Loops B and C is unknown.

The pathophysiology of preeclampsia is as follows:

(1) *Disease of arterioles:* Increased vascular reactivity to pressor substances is a common denominator in pregnancy and is associated with vasospasm. Norepinephrine has been shown to cause an increased amount of cardiovascular responsiveness in the pre-toxemic patient, as shown by Rabb et al [19], and subsequently by the unpublished work of Talledo, Chesley and Zuspan [18]. Zuspan et al [21] demonstrated that epinephrine elicits a hyperactive response from the cardiovascular system of the preeclamptic patient. This vasospastic condition of the arterioles decreases blood flow to vital organs, especially the uterus, and compromises placental function which places the fetus in a precarious position for survival. In all probability this altered uterine blood flow and encroachment on placental nutrition accounts for the large number of stillbirths that occur in the severe forms of preeclampsia.

(2) *Compromised metabolic function:* Sodium is retained in toxemia of pregnancy.

There is an increase in the sodium space and an increase in the total exchangeable sodium [11]. The more severe toxemic patient retains ingested sodium and clinically her condition worsens [12]. It is postulated that there is an increase in intracellular sodium which displaces potassium. Balance ward studies on eclamptic patients have shown an intracellular potassium deficit which infers this sodium replacement. This could explain both the increased cardiovascular reactivity and the increase in central nervous system irritability [17].

(3) *Increased central nervous system irritability:* The more ill the preeclamptic, the greater the central nervous system irritability. This is most easily demonstrated by testing for hyperactive reflexes. Signs of central nervous system irritability, such as apprehension and anxiety, precede a convulsion.

(4) *Compromised renal function:* The toxemic lesion, as recently described by the electronmicroscopists, can explain the decreased glomerular filtration rate that is present in the severe toxemic patient. As a general rule, the more ill preeclamptic patient also exhibits degrees of oliguria; occasionally the 24-hour output may be less than 400 cc.

(5) *Alterations of vascular compartment:* Hemoconcentration occurs as the preeclamptic condition worsens. The blood volume of the more severe preeclamptic patient may approach the nonpregnant state and a deficit of 1000 cc or more is not unusual [21]. The hematocrit has been used for many years as a guideline to clinical improvement and, characteristically, is high when the ill preeclamptic-eclamptic patient is initially seen. Subsequent therapy and mobilization of fluid from the extracellular compartment, as diuresis occurs, decreases the hematocrit.

(6) *A catabolic disease:* Previous balance ward studies on eclamptic patients substantiate the catabolic effect of the severe forms of preeclampsia [17]. A positive nitrogen balance is usually seen in the normal pregnant patient; however, in the eclamptic patient a negative antepartum balance is associated with a positive postpartum nitrogen balance. Brewer, among others, has long been a staunch advocate that decreased protein intake is primary in the disease [22].

Clinical Manifestations

Preeclampsia can be either mild or severe. The convulsive form is known as eclampsia. Mild preeclampsia is the most common type seen. As the disease progresses in the last trimester of pregnancy it is often difficult to diagnose the dividing line between abnormal fluid retention and mild preeclampsia. The most important point to be emphasized, however, is that once a diagnosis of preeclampsia is made the patient must be hospitalized. Outpatient therapy leads only to procrastination. The progression of signs and symptoms from mild preeclampsia to severe preeclampsia and then to eclampsia may not necessarily be in this particular order, as a patient who exhibits mild preeclampsia may be convulsing within a relatively short period of time. As a general rule a patient does not go from abnormal fluid retention to eclampsia, for a period of time is needed for such progression to take place. Therefore it is important to identify those individuals who constitute this high-risk group and to institute a program of more careful antenatal care. Prevention is the password to the elimination of this disease.

The clinical signs and symptoms are as follows:

A. *Mild preeclampsia:* The transition from abnormal fluid retention to mild preeclampsia is usually insidious. Edema other than dependent edema is a significant finding and is usually associated with a weight gain of more than two pounds during the preceding week. The blood pressure may be greater than 140/90; however increments in blood pressure of greater than 30 systolic and 15 diastolic are more significant. A diagnosis of mild preeclampsia can be made if any one of these findings is present. Characteristically, the development of edema occurs first; next, the change in the blood pressure is seen; last will be the ap-

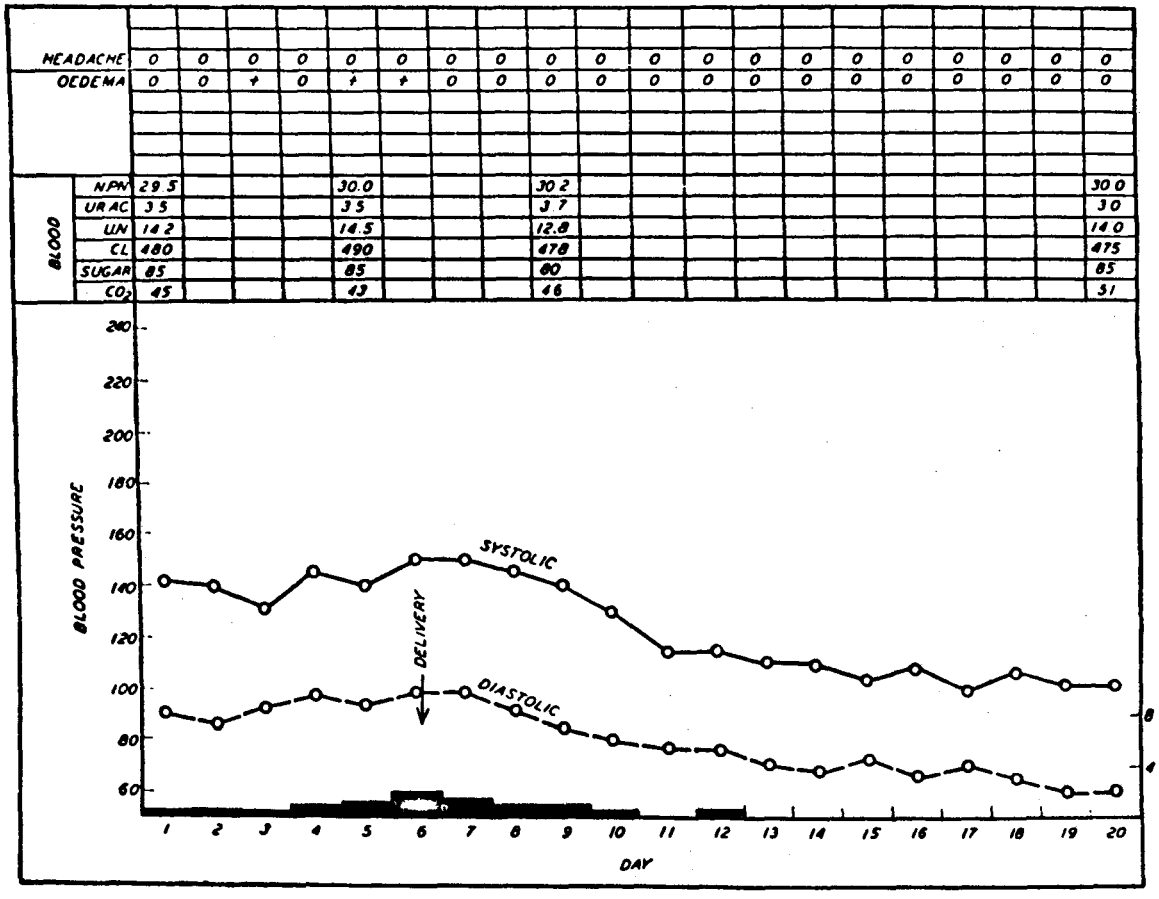

FIG. 5. Graph of characteristic findings in a patient with mild preeclampsia. (From: Zuspan. *Davis' Gynecology and Obstetrics.* Hagerstown, Md., Hoeber Medical Division, 1968.)

pearance of proteinuria. The amount of proteinuria is less than 2 gm per 24-hours in mild preeclampsia. Figure 5 illustrates graphically a patient with mild preeclampsia and shows the gradual rise in blood pressure, the appearance of small amounts of albumin in the urine and the development of edema. The patient was essentially asymptomatic and did not have headaches. The blood chemistries were within normal limits. This is the characteristic pattern of a patient who has mild preeclampsia. Once delivery is achieved, it can be noted that the blood pressure returns to prepregnant levels and proteinuria gradually disappears and is usually absent by the time the patient leaves the hospital. A mild postpartum diuresis is usually seen within the first 48 hours after delivery, in excess of that anticipated in the normal pregnant patient.

Once preeclampsia develops, and in spite of all forms of therapy and good response to that therapy, the disease continues to involve the maternal organism until the products of conception have been delivered. The alterations of pathophysiology are dependent upon the severity of the preeclampsia.

B. *Severe preeclampsia:* Edema is more generalized, clinically more apparent and usually associated with a weight gain of more than 6 lbs the preceding week. Blood pressure is in excess of 160/110 and there is marked proteinuria of 3-4+ or greater than 5 gm per 24 hours. There are varying degrees of severe preeclampsia and often the patient with severe preeclampsia is more ill than the patient who has convulsed and has eclampsia. The patient with severe pre-

eclampsia should be identified as an obstetric hazard, as an emergency situation exists for both mother and fetus.

Figure 6 illustrates graphically the patient with severe preeclampsia. It is not unusual for a patient to complain of headache, sharp epigastric pain and difficult vision, as these are premonitory signs of an impending convulsion. The progression of the patient from severe preeclampsia to eclampsia may take place during the period of observation. The patient develops increased central nervous system irritability, clinically characterized by increased reflexes and twitching of the fingers and toes. Opthalmologic examination occasionally shows retinal hemorrhages as the disease progresses. Later a generalized convulsion is observed.

The blood chemistries are within normal limits except for the uric acid which is elevated in preeclampsia and is often associated with a decrease in the glomerular filtration rate. The BUN may increase if oliguria progresses. Urinary estriol decreases as the severity of the preeclampsia increases and is inversely related to the amount of urinary protein. Following delivery the severe preeclamptic patient improves dramatically with a decrease in urinary protein and a gradual decrease in blood pressure which should reach normal limits by the 6th week postpartum. The patient may have a profound diuresis, and the typical facies of the severe preeclamptic patient is dramatically changed in the first three days postpartum. Often a decrease in blood pressure is associated with this diuresis and loss of sodium in the urine. The major problems that develop in the severe preeclamptic pa-

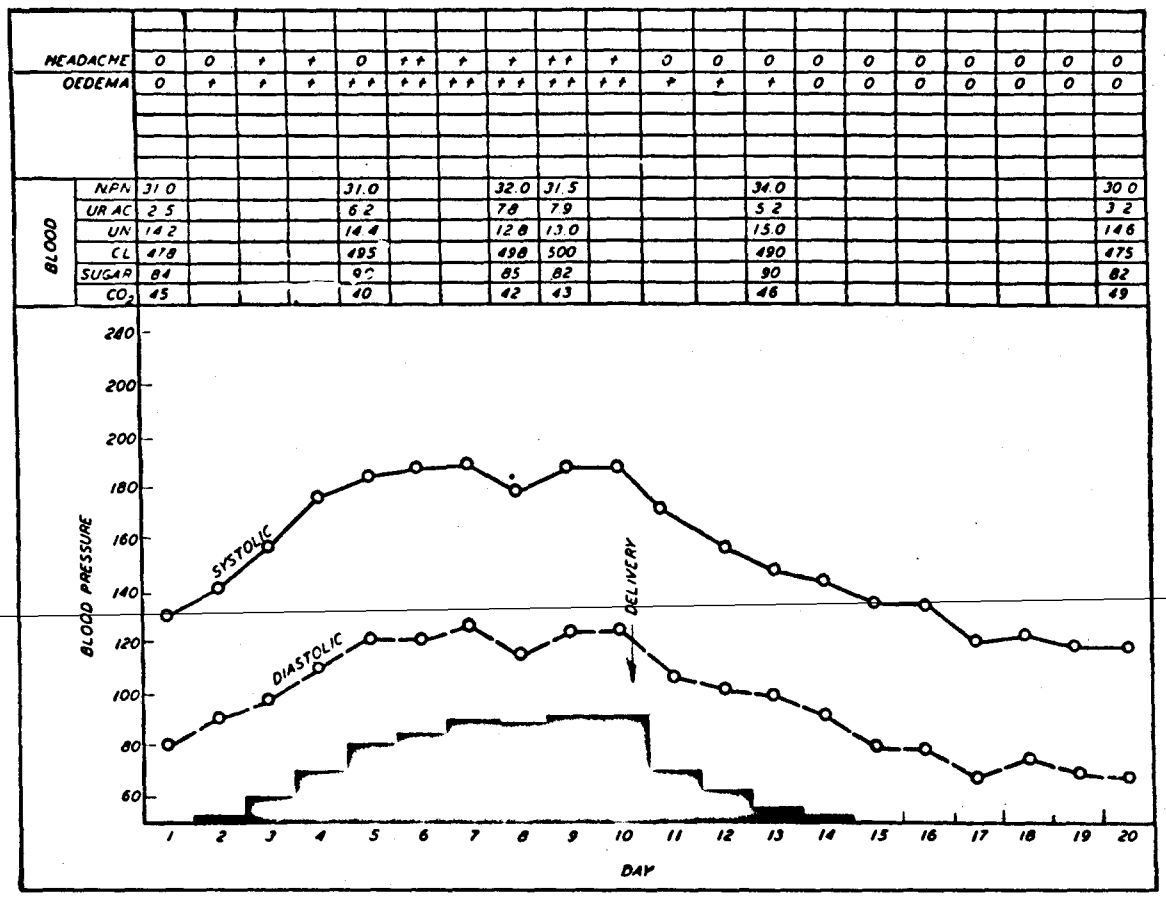

FIG. 6. Graph of characteristic findings in a patient with severe preeclampsia. (From: Zuspan. *Davis' Gynecology and Obstetrics.* Hagerstown, Md., Hoeber Medical Division, 1968.)

tient are: (a) cerebral vascular hemorrhage, (b) abruption of the placenta, with or without hypofibrinogenemia, and (c) fetal demise.

C. *Eclampsia:* Hippocrates used the Greek word from which "eclampsia" was derived to indicate the sudden onset of a fever. The Greek word means "sudden onset, or a flash," and indicates the sudden appearance of the disease. This, however, is a complete misnomer. Eclampsia does not suddenly develop in a normal pregnant patient, for there has to be a transition from abnormal fluid retention to mild preeclampsia and hence to severe preeclampsia or eclampsia. Often the transition from mild preeclampsia to eclampsia may occur in a very short period of time and hence be thought of as sudden onset. The onset of eclampsia may be insidious in that the patient has minimal or no symptoms until she is suddenly rather seriously ill. The term "eclampsia" merely means that a patient who has the triad of edema, hypertension and proteinuria in the last trimester of pregnancy has a convulsion. Again, there are degrees of severity of eclampsia, but the patient who has eclampsia has the most severe form of the disease since this is associated with increased maternal and fetal mortality and morbidity. The clinical description of the patient with eclampsia is the same as that of the patient with severe preeclampsia, except that she has also had a convulsion and may or may not be in coma. Frequently the premonitory signs of convulsion are blurring of vision and epigastric pain, with or without associated headache. Figure 7 graphically illustrates a patient who has had eclampsia and indicates con-

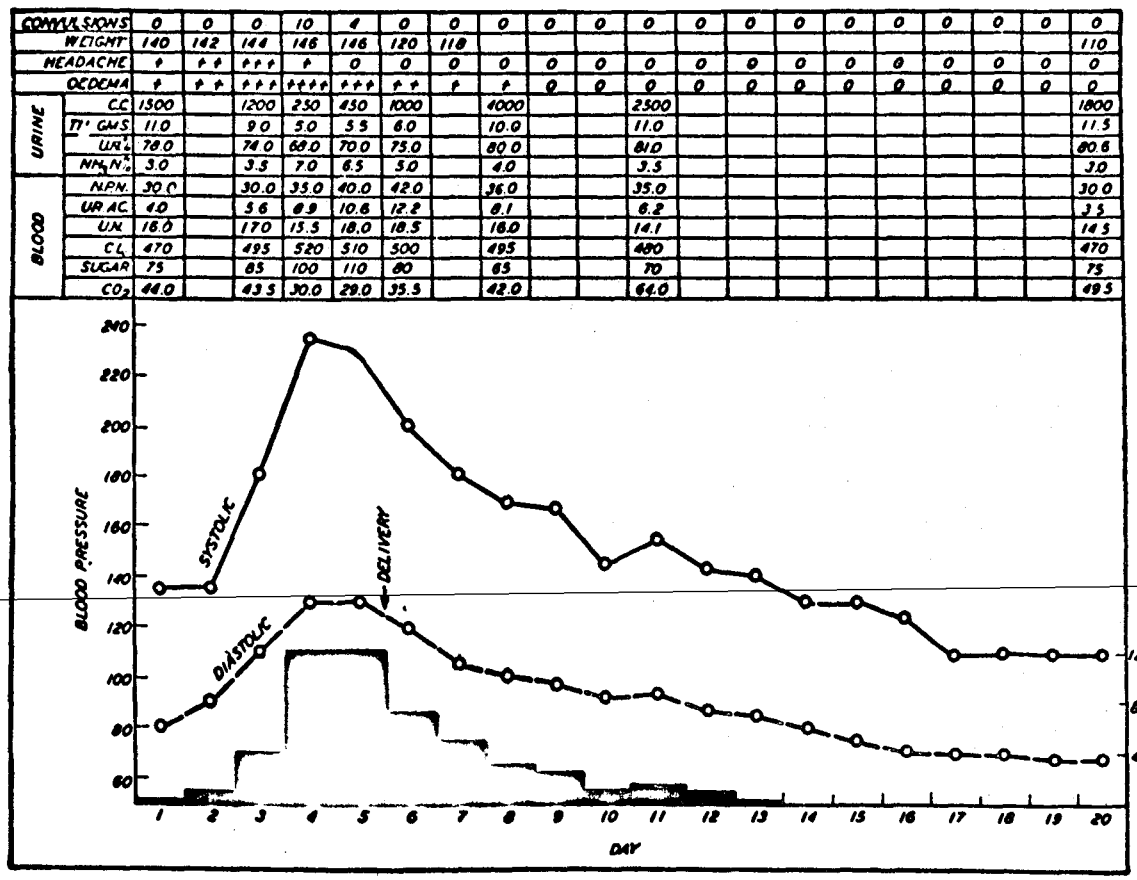

FIG. 7. Graph of characteristic findings in a patient with eclampsia. (From: Zuspan. *Davis' Gynecology and Obstetrics.* Hagerstown, Md., Hoeber Medical Division, 1968.)

vulsions on the two days preceding delivery, associated with a weight gain and increased edema. It should also be noted that the urinary output gradually decreases to 250 cc on the day preceding delivery and is 450 cc on the day of delivery. Subsequently, a diuresis of 4000 cc was noted on the third postpartum day and was associated with a decrease in weight. The NPN and the uric acid both increased as the disease worsened. The blood pressure changed in two days from 140/90 to 230/120 and was associated with an increased proteinuria. Once delivery was achieved, a dramatic improvement was seen in the blood pressure, and it reached prepregnant levels at the end of the third week postpartum. A gradual diminution in proteinuria was seen, and there was no protein in the urine after a week postpartum. There was a marked decrease in weight in the postpartum period. The course could be considered rather typical of most eclamptic patients.

Complications

The most severe maternal and fetal complications obviously result in death. Table I illustrates maternal and perinatal mortality in eclampsia. Maternal mortality varies from zero to 13 per cent, while perinatal mortality ranges from 10 to 37 per cent. During the past four decades maternal mortality has gradually decreased to less than 10 per cent. Perinatal mortality, however, continues to remain in the vicinity of 25 to 35 per cent. The challenge for the obstetrician-gynecologist is to deliver a living fetus in toxemia of pregnancy. Perinatal mortality is partially dependent upon the length of gestation when severe preeclampsia occurs. The two lowest figures in this table deserve some comment: the studies of Pritchard and Stone [25] and those of Zuspan and Ward [8]. The common denominator in these studies was the liberal use of parenteral magnesium sulfate. Rather than the magnesium sulfate itself, it was probably the absence of multiple additional medications that accounted for these lowest of perinatal mortality figures.

The following severe maternal complications associated with toxemia are usually not seen in mild preeclampsia, but are associated with the severe forms of preeclampsia and eclampsia:

A. *Abruptio placenta:* Abruptio placenta is seen more frequently in patients who have acute hypertension during pregnancy and is

TABLE I.

Perinatal and maternal mortality from eclampsia.

Years	Institution	Investigator	No. of patients	Maternal morality (per cent)	Perinatal morality (per cent)	
1920-40	Medical College of Georgia	Torpin and Coppedge	350	12.8	31.0	
1930-60	University of Cincinnati	Bryant and Fleming	253	1.6	27.7	
1931-40	Margaret Hague Hospital	Chesley and Somers	169	7.1	33.5	
1931-50	Chicago Lying-in Hospital	Dieckmann	120	0	21.4	
1942-65	University of Alabama	Jones and Williams	192	5.4	27.3	
1949-58	Hospital Central Valencia, Venezuela	Gavaller and Aguero	107	9.3	30.7	
1950-59	Mat. Conc. Pal. Caracus, Venezuela		405	10.0	28.8	
1950-59	Queen Charlotte's Mater. Hosp., (London)	Claireaux	46	0	24.0	
1950-60	Madras Medical College (India)	Menon	260	2.7	32.1	
1951-58	University of Tennessee	Adams and Cameron	220	6.8	23.5	
1955-64	University of Mississippi	Newton	92	2.2	32.0	
1956-64	Medical College of Georgia	Zuspan and Ward	46	4.0	37.0	
	If fetal heart tone present:					
	Old treatment		27	0	27.0	
	New treatment		19	0	10.0	
1938-63	University of Virginia	Thornton	168	4.7	21.6	[23]
1955-66	University of Texas Southwestern	Pritchard & Stone	69	0	21.0	[24]
	Fetus >1000 Gm		0	0	11.0	
1963-66	National University of Mexico – Hosp. Centro Mex.	Lopez-Llera	107	10.3	28.7	[25]

more common in patients who have preeclampsia. A four-year review of cases from the Medical College of Georgia hospital [27] revealed 39 cases of hypofibrinogenemia, of which 54 per cent were associated with abruptio placenta and 23 per cent with preeclampsia-eclampsia. Therefore, the incidence of abruptio placenta is in direct proportion to the incidence of hypertension during pregnancy, and more particularly to the incidence of preeclampsia-eclampsia. Dieckmann [28] in his studies at Chicago Lying-in Hospital identified 30 per cent of 186 cases of abruptio placenta to be associated with toxemia of pregnancy (hypertension, edema, and albuminuria).

B. *Hypofibrinogenemia:* The animal work of McKay et al. [29] has demonstrated a generalized fibrin deposition in experimental toxemia. This has not been critically evaluated in the human since hypofibrinogenemia is not clinically diagnosed until the fibrinogen level is 100 mg% or less and it does not occur frequently enough for careful clinical study. I would suspect that we might observe a decrease in fibrinogen if serial determinations of fibrinogen levels were made in patients who had severe preeclampsia. Twenty-three per cent of the 39 cases of hypofibrinogenemia that occurred at the Medical College of Georgia hospital during the four-year period were associated with preeclampsia-eclampsia. Thus, the patient who is identified as having severe toxemia of pregnancy should be followed more critically with fibrinogen levels.

C. *Hemolysis:* The occasional severe preeclamptic patient may exhibit clinical signs of hemolysis as manifested by jaundice. It is not known whether this is hepatocellular damage or red blood cell destruction from other causes. Periportal necrosis of the liver, a common autopsy finding in eclampsia, could explain such a phenomenom. Hepatocellular changes probably occur in the liver of those patients who have toxemia but eventually improve. I have seen this on at least half a dozen occasions in severe preeclamptic patients who eventually recovered. These patients had abnormal liver function tests, especially the enzyme determinations.

D. *Cerebral hemorrhage:* This is the most common cause of death in toxemia of pregnancy and is the one central theme that worries all clinicians when they manage patients who have acute hypertension during pregnancy.

E. *Opthalmologic abnormalities:* Temporary loss of vision, lasting for as long as seven days, has been observed. Hemorrhage will occasionally occur in the eye grounds of a patient. This acute phenomenon should warn the clinician of an impending vascular accident.

F. *Pulmonary edema:* In a study group of 69 eclamptic patients [23] this was seen only once. As a general rule it can be easily managed by phlebotomy and the conventional therapy for acute cardiac failure.

G. *Necrosis of the liver:* The hepatocellular damage of periportal necrosis is related to the generalized arteriolar vasospasm. This was originally thought to be pathognonomic of eclampsia, but has subsequently been seen in other diseases. Subclinical hepatocellular damage can best be determined by liver profile studies. The most sensitive are serial enzyme determinations. We feel it is hazardous to do liver biopsies on the eclamptic patient.

H. *Kidney:* The toxemic lesion in the kidney has been referred to as glomerular endotheliosis and was described in detail by Spargo, McCartney and Winemiller [30]. Photographs from their study show a normal patient (Fig. 8.) and a patient with this toxemic lesion (Fig. 9.). These authors concluded that the lesion consisted of a swelling of the endothelial cytoplasm without involvement of other structures and was entirely reversible. Because of this they proposed the name glomerular endotheliosis, which is the terminology now used to describe the toxemic lesion in the kidney. Other renal problems that may develop are associated with problems relating to abruptio placenta and may result in renal shutdown that necessitates renal dialysis.

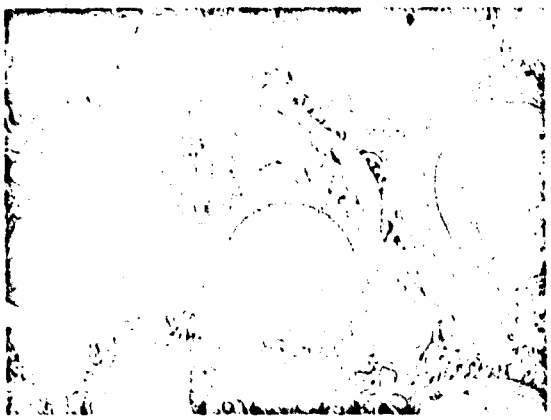

FIG. 8. Electron micrograph. Normal pregnant patient. The capillary lumens are wide and the glomerular components are normal. (From: Spargo, McCartney and Winemiller. Arch. Path. 68: 593, 1959.)

FIG. 9. Electron micrograph. Toxemic lesion. Glomerular changes believed to be pathognomonic of pre-eclampsia-eclampsia. There is narrowing of the glomerular capillary lumens, an increase in the cytoplasm of the endothelial cells, and subendothelial deposits of osmophillic material. There are no consistent changes in the basement membrane, the foot processes of the epithelial cells are distinct and there is no proliferation of cells in contact with the basement membrane. (From: Spargo, McCartney and Winemiller. Arch. Path. 68: 593, 1959.)

I. *Postictal trauma:* The convulsing patient often traumatizes herself by injury to her oral cavity or extremities. A more serious complication, however, is aspiration with subsequent pneumonia.

J. *Fetal complications:* The decrease in uterine blood flow ultimately encroaches upon available placental reserve and may lead to unfortunate situations for the fetus. The placenta shows an increased number of infarcts which result in a decrease in the functional integrity of this organ and in turn results in fetal malnutrition. This intrauterine fetal malnutrition can be complete, with delivery of a stillborn; or partial, as seen in a neonatal death or a newborn that weighs less than expected for the length of gestation.

This fetal malnutrition is best illustrated by Figure 10, which shows the birth weight of babies from a group of 69 eclamptic patients, as correlated with historical weeks of gestation. The comparison is made to the birth weight curve of normal gestation from the data of Charles Hendricks [31] and illustrates that in the severe forms of toxemia of pregnancy intrauterine growth retardation occurs by as much as 400-700 gm. This decrease in fetal weight does not necessarily indicate that the fetus is not mature, but often misleads the clinician in his judgement of fetal size and viability.

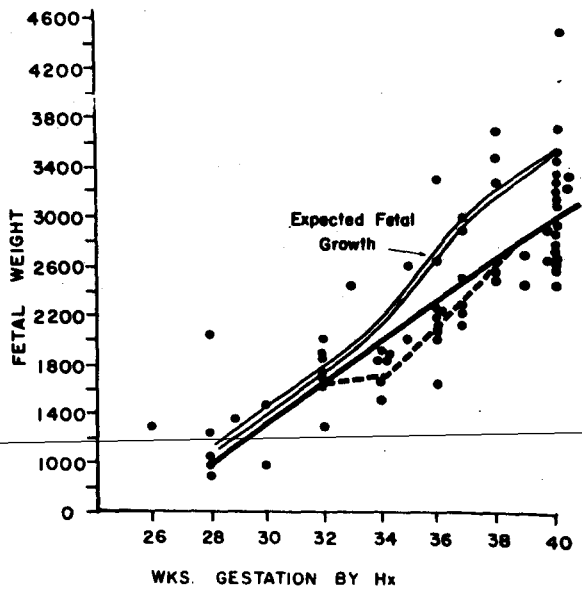

FIG. 10. Weight of fetus delivered from eclamptic patients as determined by weeks of gestation by history. (From: Zuspan, Treatment of Severe Preeclampsia and Eclampsia, Symposium on Toxemia of Pregnancy. Clin. Obstet. Gynec. 9: 971, 1966.)

Prematurity is a frequent complication of the severe forms of toxemia and to some

extent governs the rate of fetal salvage. I have not seen a live baby delivered from an eclamptic patient before 30 weeks of gestation. The toll of fetal loss, however, is evident in Figure 11, which shows that there is considerable fetal loss even up to 40 weeks gestation. This infers that factors other than prematurity are influencing fetal salvage. These factors have been previously alluded to in the discussion of abnormal placental function and diminished placental reserve. Ideally, after 32 weeks of gestation the fetus' statistical chance of death should be no greater than five per cent, rather than five to six times this figure.

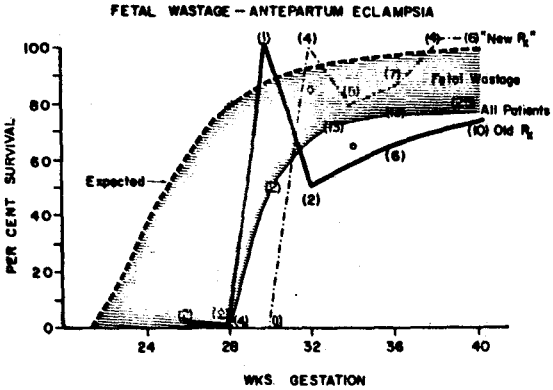

FIG. 11. Fetal survival from antepartum eclampsia. Shaded area indicates fetal loss from expected survival at various weeks of gestation. (From: Zuspan, Symposium on Toxemia of Pregnancy, Clin. Obstet. Gynec. 9: 965, 1966.)

DIAGNOSIS — INCLUDING DIFFERENTIAL DIAGNOSIS

At first glance the differential diagnosis between preeclampsia, renal disease and chronic vascular disease clinically appears rather easy. However it becomes apparent from the electronmicroscopy studies of renal biopsies that this clinical differentiation is often not valid. It must be assumed, of course, that the so-called toxemic lesion of glomerular endotheliosis, as seen under the electronmicroscope, is a valid definitive diagnosis for preeclampsia. This may not be totally correct but, at the present time, this is the most definitive means used for a differential diagnosis. Clinical diagnoses at

TABLE II.

Hypertensive disorders of pregnancy. Clinical diagnosis in the Chicago Lying-in Hospital*.

Disorder	Per cent
Preeclampsia	46.0
Eclampsia	1.7
Chronic hypertensive vascular disease	51.0
Chronic renal disease	1.1

*Number of cases: 7893.

From McCartney, C. P. Toxemia of Pregnancy — Classification, Symposium on Toxemia, Zuspan, F. P. (ed.), Clin. Obstet. & Gynec. 9:866, 1966.

the Chicago Lying-in Hospital during the past 18 years are seen in Table II [32]. It indicates that roughly half of the patients were diagnosed as chronic hypertensive vascular disease and the other half as preeclampsia, whereas chronic renal disease was diagnosed in only 1 per cent. The security of this clinical diagnosis was challenged when Spargo, McCartney and Winemiller described their electronmicroscopic findings from renal biopsies, indicating that chronic renal disease was much more prevalent than ever before suspected [30]. Table III Describes the clinical diagnosis and renal biopsy findings in the hypertensive disorders of pregnancy. It is immediately apparent that the best of clinical diagnoses in the primigravida can be correct approximately 70 per cent of the time if we assume that the toxemic lesion is indeed the *sine qua non* for preeclampsia. The situation is almost reversed in the multigravida patient, where roughly 25 per cent of the clinical diagnoses will be substantiated by the renal biopsy and 75 per cent will not. It becomes apparent, than that the clinical diagnosis of so-called "toxemia of pregnancy" often is in error. This should be kept in mind when trying to compare statistics from one institution to another, as these are dependent upon multiple factors and whether or not a renal biopsy was done on patients. It should be added that at the present time renal biopsies should be done in the immediate puerperium, i.e., approximately 72 hours or more postpartum, rather than during pregnancy. The definitive diagnosis is needed for long-term prognosis of the patient, but not for the immediate therapy of the individual patient.

TABLE III.

Hypertensive Disorders of Pregnancy

	Clinical diagnosis	Cases	Renal histology: Normal	Toxemic lesion	Toxemic lesion and arteriolar sclerosis	Arteriolar sclerosis	Chronic renal disease
Primigravida	Preeclampsia	62	3	43	1		15
Multigravida	Chronic hypertensive vascular disease with super-imposed acute toxemia	152	81	5	16	18	32

From McCartney, C. P., Toxemia of Pregnancy — Classification, Symposium on Toxemia of Pregnancy (Zuspan, F. P., ed.), Clin. Obstet. Gynec. 9:867, 1966.

The schematic drawing of Figure 12 illustrates the enigma of the differential diagnosis and the multiple factors that must be weighed in determining whether the diagnosis will be preeclampsia, renal disease or chronic vascular disease. At the present time the ultimate diagnosis is established by renal biopsy.

The convulsing pregnant patient, unless she has signs of hypertension, edema and proteinuria, should not immediately be classified as eclampsia, as other forms of

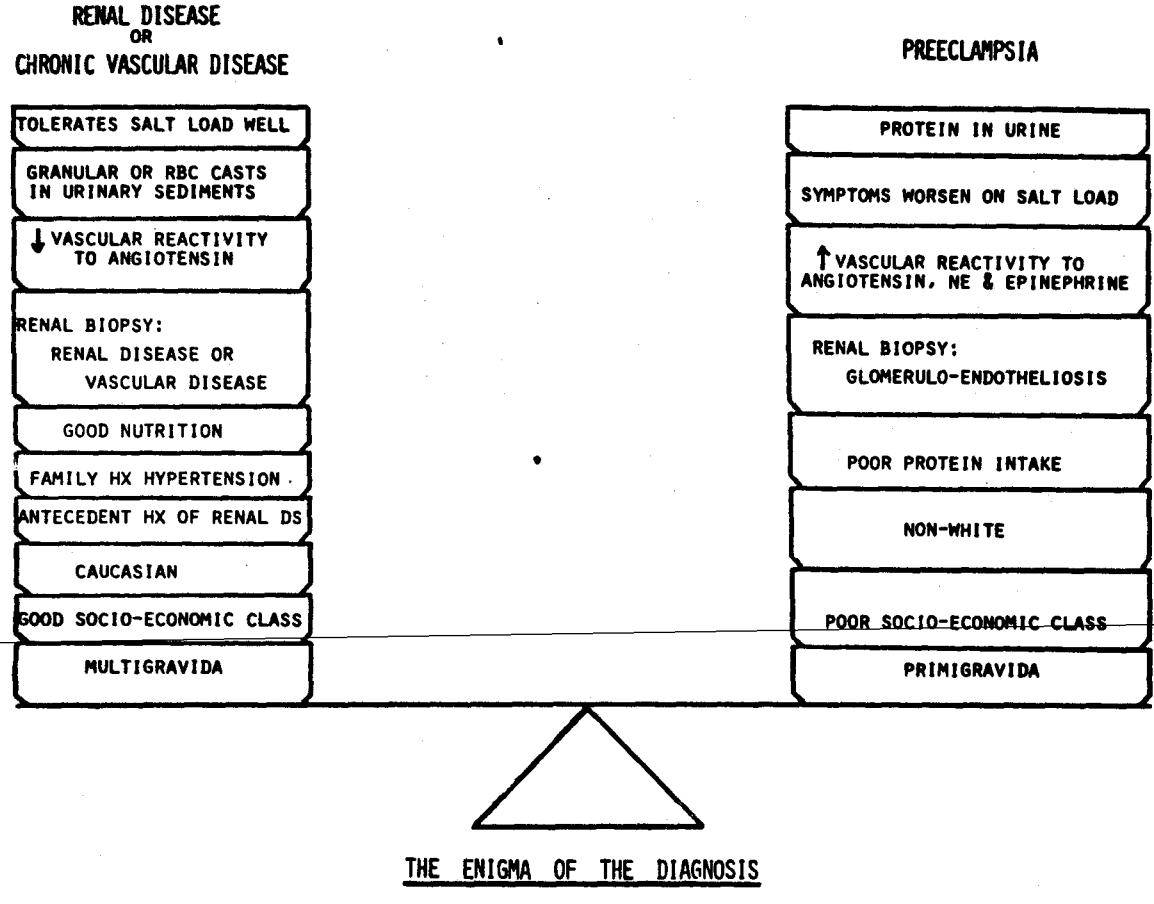

FIG. 12. The clinical differential diagnosis of hypertension in late pregnancy is based on multiple factors. (From: Zuspan. *Davis' Gynecology and Obstetrics.* Hagerstown, Md., Hoeber Medical Division, 1968.)

seizures can occur in the pregnant patient. Such things as aneurisms, epilepsy, stroke, brain tumors, etc., must be considered when examining and managing the patient.

Prevention

To prevent toxemia of pregnancy, one must prevent the ills of society such as low socio-economic groups, poor nutrition with low protein intake, overcrowding, limited antenatal care and poor reproductive education. Since this cannot be done, the main effort should be in the identification of those patients in whom toxemia of pregnancy is known to be prevalent. Figure 13 illustrates that eclampsia is a disease of teenage primigravidas. When identified early in pregnancy and seen at frequent intervals, this group can be guided in a regimen of prevention, since the severe forms of preeclampsia are preventable. Whether or not abnormal fluid retention and mild preeclampsia can be prevented remains a point of conjecture. Until physicians realize that hospitalization is mandatory once a patient progresses beyond the clinical stage of abnormal fluid retention, no headway will be made in the prevention of the severe forms of preeclampsia. This was amply demonstrated in Australia and New Zealand, as reported by Corkill [5], where the incidence of eclampsia in registered patients was reduced from 1:300 in 1947 to 1:3,786 in 1957. This was accounted for only by a change in admittance policy when early signs of toxemia were recognized. Early recognition of toxemia and its aggressive hospital management, coupled with identification of the group of patients in which this statistically should occur, may well be the greatest contribution of prenatal care.

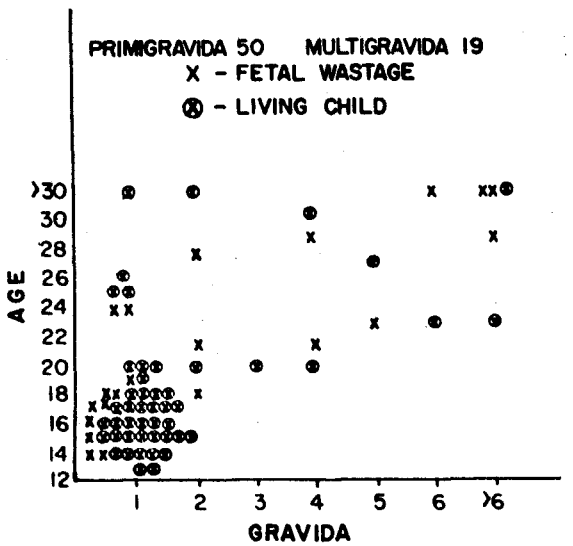

Fig. 13. Eclampsia in these 69 patients tends to point toward the disease as one of a teenage primigravida. If a multigravida, the fetal prognosis is worse. (From: Zuspan, Symposium on Toxemia of Pregnancy, Clin. Obstet Gynec. 9: 964, 1966.)

Outpatient diuretic and antihypertensive therapy only leads to procrastination and has little place in the management of preeclampsia. Vacant beds on obstetric services in this country could well be utilized by more liberal admittance policies when patients develop abnormal fluid retention. Prevention should be directed toward the mild forms of the disease.

Treatment

There are as many different types of therapy for toxemia as there are physicians in the world and each feels his own method of therapy is satisfactory. All therapy for preeclampsia-eclampsia is empiric since the basic etiologic mechanism is unknown. This does not mean that patients who have this disease cannot be intelligently managed by keeping in mind the pathophysiology, i.e., that it involves (1) disease of arterioles, (2) compromised metabolic function, (3) increased central nervous system irritability, (4) decreased renal function, (5) alterations of the vascular compartment, and (6) is a catabolic disease.

The strongest form of therapy rests in the identification of that group of patients in which it is known statistically that preeclampsia-eclampsia can develop and in institutions of appropriate antenatal care for this group. Prevention, rather than therapy, is the most important single factor in management.

Hospitals and physicians must have a

liberal admission policy for the treatment of preeclampsia. This will almost completely eliminate the severe manifestations of preeclampsia and decrease maternal and fetal mortality and morbidity.

Keeping in mind the disturbed pathophysiology, the following treatment guideposts can be used for the preeclamptic-eclamptic patient. It should be emphasized that there is no cookbook method for treating this disease and that therapy must be highly individualized.

(a) If preeclampsia is diagnosed in an outpatient, the only therapy is hospitalization. Procrastination and non-beneficial effects for the patient and her fetus result if diuretic and anti-hypertensive therapy is used. Much has been written on the use, both prophylactic and therapeutic, of diuretics during pregnancy. Most papers, however, have been weighed by the emotional impact of the medication and the fact that once a study is conducted, the patient usually behaves differently. In 1966, a paper by Kraus, Marchese and Yen [33] was published. This double-blind investigation was on the continuous prophylactic use of hydrochlorothiazide (50 mg daily) in 1,030 obstetrical patients. This is one of the best controlled studies in the literature and demonstrates that long-term prophylactic use of hydrochlorothiazide in pregnancy does not alter the incidence of preeclampsia, hypertension, prematurity, congenital anomalies, or perinatal mortality. These few lone voices which point out the fact that diuretics do not serve a purpose in the prevention of preeclampsia are usually lost in the over-enthusiasm of other investigators who feel that diuretics are the answer to the problem of toxemia of pregnancy. It is unsound physiologically to give diuretics, as these investigators point out that asymptomatic hypokalemia developed in a reasonable percentage of patients. Diuretics potentiate the pathophysiology that exists in toxemia of pregnancy. Figure 14 illustrates this point with data on four eclamptic patients. The potassium-nitrogen ratio in the balance ward studies was calculated over a total of 21 days postpartum and was higher than the normal ratios in muscle. It is felt that this may well represent an intracellular deficit of potassium.

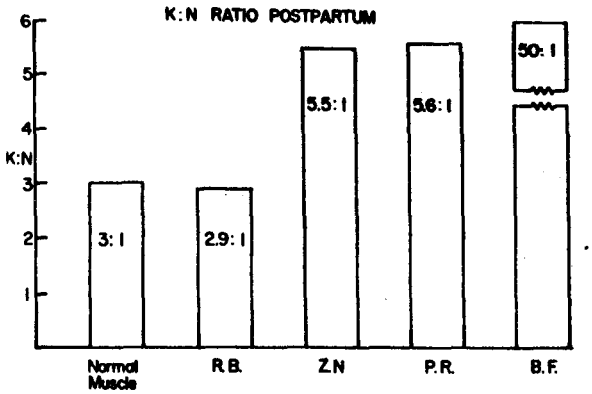

FIG. 14. The Potassium:Nitrogen ratio is compared to normal muscle. Three of the four eclamptic patients show ratios greater than normal muscle. (From: Zuspan and Goodrich, Metabolic studies in normal pregnancy: I. Nitrogen metabolism. Am. J. Obst. Gynec. In press)

(b) Patients that will probably develop preeclampsia-eclampsia should be identified and followed on a different antepartum schedule than normal pregnant patients. With careful antepartum care it is almost impossible for the severe forms of toxemia to develop. Eclampsia does not come as a bolt of lightening, but is usually insidious in onset and can be recognized. These milder forms of preeclampsia can be diagnosed and then treated by hospital admittance.

(c) Once a patient is hospitalized and under therapy, convulsions should rarely occur. If reflexes are hyperactive an intravenous infusion of magnesium sulfate is given.

(d) Neither the fetus nor the patient should be harmed by poor obstetric judgement and/or overzealous therapy. The concern in the preeclamptic patient is primarily for the fetus, since maternal mortality is rare.

(e) Residual cardiovascular damage is not caused by toxemia of pregnancy. In later life the single most important factor in the development of hypertension is a familial history of hypertension. In all probability the genetic component of hyper-

tension is the deciding factor, not the fact that the patient had preeclampsia-eclampsia. This means that the urgency for delivery is not based upon maternal indications, but is dictated by the immediate welfare of the fetus.

I. *Period of Fluid Retention:* During early antepartum care most patients can be managed by a word of caution simply stating that excessive ingestion of salt is detrimental to the mother and fetus. After the fifth month of pregnancy the patient should curtail her salt intake to some degree. However, the most important single item in antenatal management is an adequate diet of good protein. Balance ward studies have shown that 0.75-1.00 gm protein per kilogram of body weight is necessary for the development of a positive nitrogen balance in the pregnant patient [17]. This is also essential in the prevention of preeclampsia-eclampsia. If a patient develops fluid retention, it may simply be suggested that she curtail salt intake and get additional bedrest to increase renal bloodflow and promote diuresis for a period of two to three days. This, in conjunction with the liberal use of small doses of phenobarbitol, has proved a beneficial and easy way to manage most patients. It is an exception to the rule when a patient needs a diuretic medication. This patient is not considered to have preeclampsia, and can be managed on an outpatient basis. She should be seen again three days after the initial visit to determine whether or not a therapeutic response has been achieved. Daily weights are helpful in plotting the therapeutic response. If, however, the patient has gone beyond the point of simple fluid retention, hospitalization is mandatory and the patient should be diagnosed as having preeclampsia.

II. *Mild preeclampsia:* Once a diagnosis of preeclampsia has been made, even though it is mild, the patient often will have a dramatic response to hospitalization. (Fig. 15.) She is taken away from her environmental influences and placed in the hospital at complete bed rest with bathroom privileges. She is restricted to a sodium intake of less than one gram per day, coupled with a protein intake of 70 gm or more per day, and placed on mild sedatives such as phenobarbitol (30 mg four times a day). If urinary output is measured a diuresis should be achieved within 18 to 36 hours after admittance to the hospital. This is manifested by a decrease in weight. If she had a mild blood pressure rise her blood pressure usually returns to normal. Again, the mild preeclamptic patient usually does not need diuretics, assuming that she has a diuresis within 48 to 72 hours after admittance to the hospital. The disease usually improves symptomatically, and often in three to five days the patient may be discharged from the hospital under very careful outpatient care. However, if the patient is 38 weeks gestation or more she should be induced with intravenous oxytocin, after careful control, for 38 weeks or more is considered as term gestation.

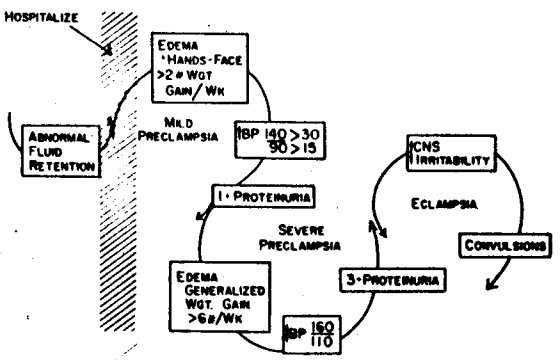

FIG. 15. Hospitalization is the only therapy once a diagnosis of preeclampsia has been made. (From: Zuspan. *Davis' Gynecology and Obstetrics.* Hagerstown, Md., Hoeber Medical Division, 1968.)

III. *Severe Preeclampsia:* The patient with severe preeclampsia is immediately identified as an individual who is seriously ill and one in which survival of the fetus is threatened. The management of this patient is not too dissimilar to that of the mild preeclamptic group. The patient requires immediate hospitalization with close supervision. She is placed at bed rest with bathroom privileges, weighed daily, intake and

output are recorded, baseline laboratory studies are done, and a 24-hour urine is collected for protein and estriol determinations. A 500 mg sodium diet that contains 70 gm of protein is given and the patient is placed on a mild sedative, such as phenobarbitol (30 mg four times a day). The blood pressure is observed every four to six hours. Diuresis can be expected to occur within the next 24 to 48 hours and the patient's condition should improve. However, some patients do not improve and their condition deteriorates in spite of hospital management. The decision for delivery must be pushed to the front for these patients. If the patient has responded with some decrease in blood pressure, but not the expected diuresis within 48 hours (by more than 2 kilograms), she will be placed on diuretics such as hydrochlorothiazide (50 mg twice a day). This rarely needs to be done however. Figure 16 shows what should be expected in the normal pregnant term patient. The data from this balance ward study shows the results of 12 normal patients near term on a restricted sodium intake. A two kilogram weight loss can be expected within two or three days after admittance to the hospital [12]. If the patient has preeclampsia the weight loss on this regimen should exceed two kilograms in three days. If weight loss does not occur in the toxemic patient then several factors may be involved: the patient's diet is improper, food is perhaps being brought in from the outside, or the disease is worsening in spite of good hospital management. The severe preeclamptic patient, if beyond 34 weeks gestation, should be delivered. Once preeclampsia develops in a given patient, in spite of the subjective and clinical response of the patient, the disease continues to smolder until the products of conception are removed from the maternal organism. The fetus may often be small for the acknowledged menstrual history, but intrauterine growth retardation is an accepted phenomenon in these hypertensive disorders of pregnancy. The reflexes may be hyperactive in the severe preeclamptic patient and need quieting by intravenous magnesium sulfate. The magnesium sulfate is administered by infusion pump or by an intravenous infusion adding 10-20 gm of magnesium sulfate to 1000 cc of 5% dextrose and water. The rate of administration is approximately 1 gm per hour. If magnesium sulfate is administered, the reflexes are monitored at frequent intervals and the urinary output should be in excess of 100 cc every four hours. Once clinical improvement ensues, the magnesium sulfate is gradually decreased and often may be discontinued. Diuretics or hypotensive agents should not be used routinely in severe preeclampsia or eclampsia. Antihypertensive therapy should be given if it appears that the patient will develop a cerebral vascular accident. A diastolic pressure in excess of 110-120 mm mercury would be an indication for hypotensive therapy. If it is used, however, it is imperative that the diastolic pressure not be reduced below 90. Intravenous apresoline has been successful in controlling acute

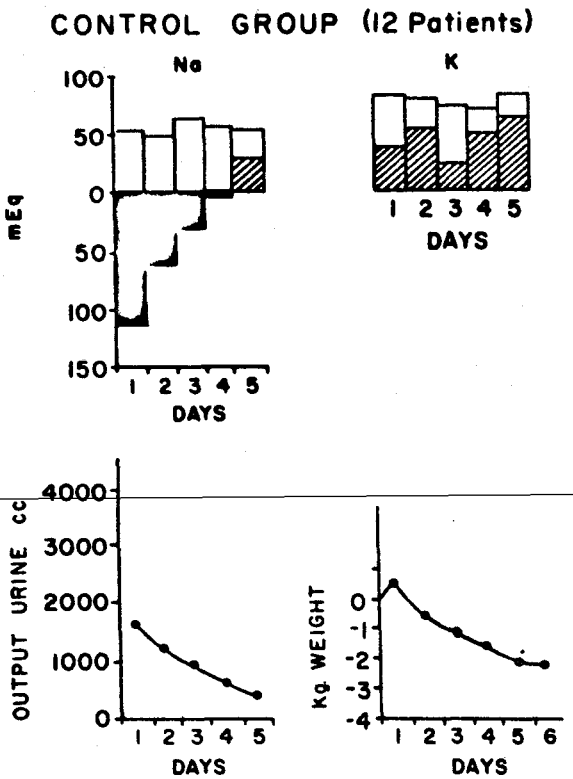

FIG. 16. Normal pregnant patients at term. The only therapy was bedrest and a restricted salt intake. (From: Zuspan and Bell, Obstet. Gynec. 18: 530, 1961.)

hypertension. The uterus should be emptied within a short period of time and clinical improvement would then be expected. Medications given intramuscularly to the severe preeclamptic patient are often poorly absorbed because of the disturbed metabolic function. Therefore it is preferable to use an intravenous medication for most forms of therapy. When induced the severe preeclamptic patient delivers rather readily, as it is known that there is increased uterine activity in the toxemic patient [34, 35, 36] Oxytocin induction is started at small dosages, such as 2-4 mU, and increased to achieve the desired uterine activity. Cesarean section should be done only for obstetric indications and not for preeclampsia-eclampsia itself.

IV. *Eclampsia:*

A. Airway — Self-injury to the oral passages should be avoided during a convulsion by use of padded tongue blades kept at the bedside and available in all labor and delivery rooms. A plastic airway is inserted following the convulsion and oxygen is administered. Adequate suctioning of the oral passages is necessary to prevent aspiration, as well as the liberal use of the Trendelenburg position. If the stomach contents are abundant they should be aspirated by a nasogastric tube.

B. Control of convulsion — Magnesium sulfate, 4-6 gm, is given intravenously over a period of five minutes to control the convulsion. If the convulsions persist in spite of this medication, then a small amount of amobarbital is administered (0.5 gm or less) as an adjunct. Usually this is not repeated. A Foley catherter is inserted and urinary output is recorded hourly; the specific gravity and albumin of the urine is determined every six to eight hours and in pooled 24-hour specimens. If the patient is not convulsing, the initial loading dose of magnesium sulfate is 2-4 gm given intravenously, followed by 1 gm per hour. This is administered by either infusion pump or intravenous drip, using 5% dextrose and water. If additional magnesium sulfate is needed because of hyperactive patellar reflexes, this can be injected into the intravenous tubing. The therapeutic dosage of magnesium acts as a mild sedative for the patient, making her much easier to manage and eliminating the use of other medications. Calcium gluconate is kept at the bedside at all times in case of a rare overdosage. Total dosage of magnesium sulfate is governed by the urinary output and patellar reflexes. Patellar reflexes should always be present and hypoactive when optimum therapy has been achieved. The urinary output should exceed 100 ml per four hours if magnesium sulfate is being administered. If it decreases below this level, the magnesium sulfate dosage should be decreased or stopped; if diuresis occurs the dosage should be increased. Once a patient is on this medication it is extremely rare for a convulsion to occur. Precautionary measures in the use of magnesium sulfate should be:

1. Check reflexes every hour; if absent, decrease or stop magnesium sulfate.
2. Urinary output, 30 cc per hour; if less, decrease dose.
3. Respiration greater than 14/minute.

Magnesium sulfate is excreted primarily by the kidney and its major action is a depression of the central nervous system activity. The clinical order of increasing levels of magnesium sulfate is first a disappearance of knee jerks, followed by a decrease in respiration and then cardiac arrest. The combined experience of Pritchard at Southwestern and the groups at the Medical College of Georgia and at Chicago Lying-in Hospital exceeds 16,000 patients treated with magnesium sulfate. In none of these patients was there ever seen a severe overdosage or serious major complication from the administration of magnesium sulfate. It is believed that most patients are *undertreated with magnesium sulfate and overtreated with other medications.*

In a group of 40 eclamptic patients treated from 1962 to 1966 (only intravenous magnesium sulfate was administered. Hypotensive and diuretic agents were not used. Fetal salvage in these 40 consecutive

eclamptic patients (admitted with live fetuses) was 89 per cent [23].

The postulated action of magnesium sulfate therapy in the eclamptic patient is shown in the following formula:

$$UBF = \frac{BP}{Ri + Re} \quad [37]$$

Uterine blood flow (UBF) is directly proportional to blood pressure (BP) and inversely proportional to the intrinsic resistance (Ri-as observed in the vasospastic vascular tree) and extrinsic resistance (Re-as seen in increased uterine tone), both of which occur in preeclampsia-eclampsia. The deciding factor for fetal salvage is the amount of placental reserve that remains to promote good fetal nutrition in preeclampsia-eclampsia. Magnesium sulfate can decrease both vascular tone (Ri) and uterine tone (Re) while changing the blood pressure (BP) very little; thus the net result would be an increase in uterine blood flow (UBF) which should promote fetal well-being.

C. Charting and laboratory studies — A simple recording system is mandatory for the critically ill eclamptic patient. Intake-output, vital signs and supplementary medications should be recorded at frequent intervals. Baseline laboratory studies consist of a hematocrit every six hours along with a specific gravity and albumin determination of the urine. Daily determinations for BUN and electrolytes. A portable chest x-ray is needed to rule out aspiration pneumonitis following a convulsion.

D. Decision for delivery—Figure 17 illustrates the general guidelines for delivery of the eclamptic patient as these relate to fetal prognosis. In the severe preeclamptic-eclamptic patient the major concern is for the fetus. The earlier the gestation, the more guarded the prognosis since a live fetus of 30 weeks gestation or less has not been delivered from an eclamptic patient. If maternal conditions permit in severe preeclampsia-eclampsia of 30-32 weeks gestation, induction of labor can be delayed until 34 weeks gestation. If the patient is 34 or more weeks of gestation she should be

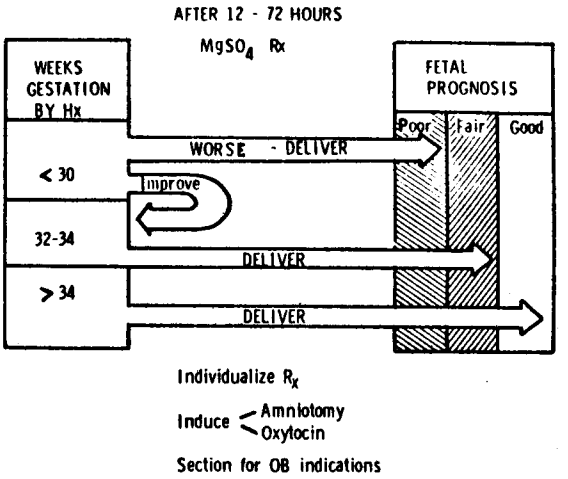

FIG. 17. Flow chart for delivery of eclamptic patient. Weeks of gestation (by history) and clinical response dictate fetal prognosis. (From: Zuspan, Symposium on Toxemia of Pregnancy, Clin. Obstet. Gynec. 9:970, 1966.)

delivered as soon as possible after good therapeutic control. Induction is by means of intravenous oxytocin and an amniotomy is done at the appropriate time. It is often necessary to "ripen" the cervix with the administration of oxytocin and this should be borne in mind in relation to when the amniotomy is contemplated. Of the three cesarean sections performed in 69 eclamptic study patients, only one was for a failed induction; the other two were done for obstetric complications [23]. It is often stated that the toxemic patient cannot be delivered because the cervix is "unripe". This is not a true statement. Figure 18 is a classification of 51 eclamptic patients according to the condition of their cervices and illustrates that most of the eclamptic patients delivered within 12 hours of induction [38]. Those patients that need induction are frequently those of less than 36 weeks gestation. The obstetrician should not be deceived by the condition of the cervix prior to an adequate trial of oxytocin, for it is felt that a higher fetal salvage is achieved if vaginal delivery is done. The increased fetal salvage of both Pritchard and Stone [25] and the Zuspan and Ward studies [8] indicate that the liberal use of magnesium sulfate in the absence of other medications, along with a

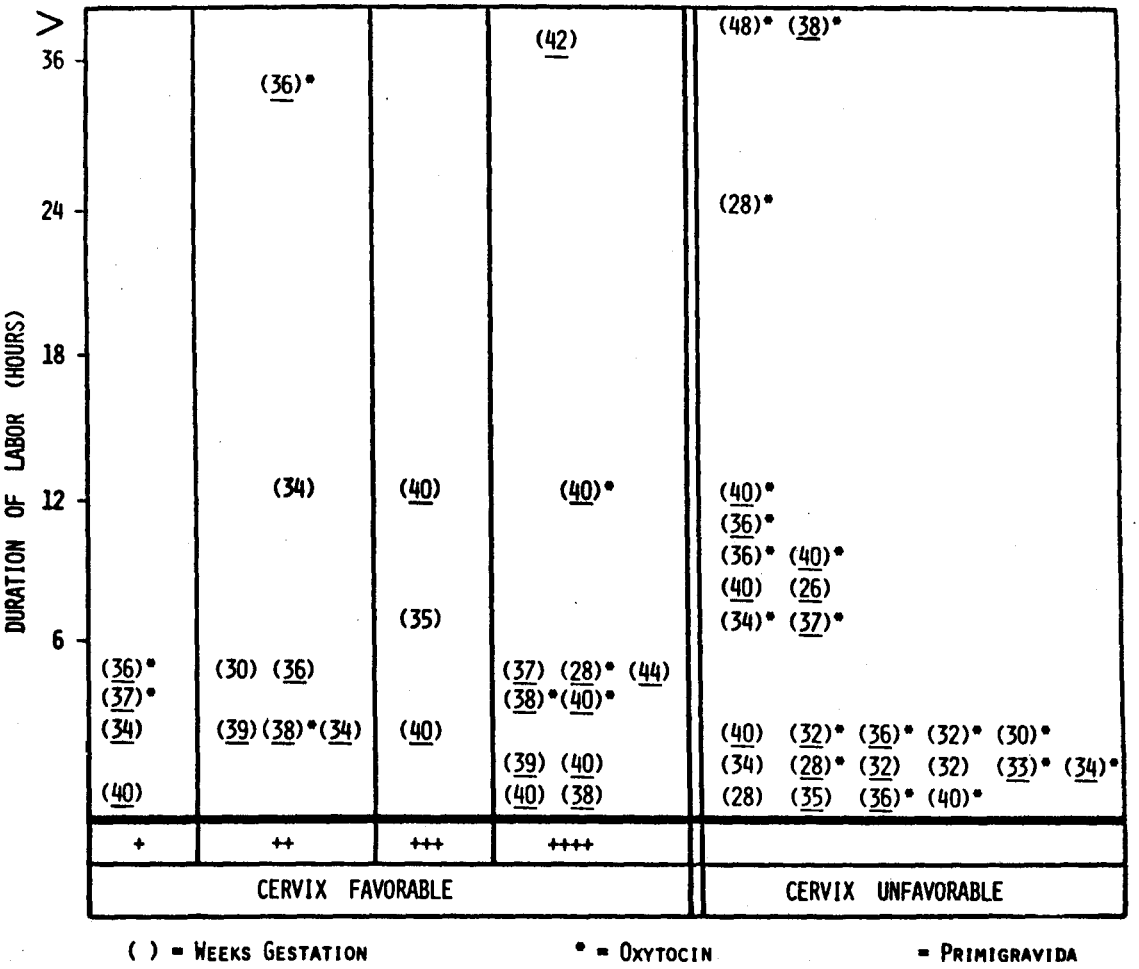

FIG. 18. Duration of labor in hours is on the ordinate and condition of cervix on the abscissa. The groups are divided into graduations of one-plus to four-plus. Weeks of gestation are within parentheses, and asterisk denotes oxytocin stimulation. (From: Zuspan and Talledo, Factors affecting delivery in eclampsia. Am. J. Obst. Gynec. 100: 672, 1968.)

prompt decision for delivery by the vaginal route, yields the highest fetal salvage.

PROGNOSIS

The immediate prognosis for the mother and fetus has been previously covered. It has been pointed out that the mild forms of preeclampsia are not associated with an increased fetal or maternal loss; however, the severe forms of preeclampsia-eclampsia are associated with an increase in fetal loss and a maternal mortality of less than 10 per cent. The association of other factors along with preeclampsia-eclampsia, such as abruptio placenta, hypofibrinogenemia and prematurity, add to the immediate prognosis of the fetus and the mother.

A more important consideration is the long-term prognosis of both mother and fetus. To date there have been no good long-term follow-up studies on infants born from toxemic mothers. Data are presently being gathered which will provide a more accurate picture of the toll taken by this disease. Studies should uncover the fact that fetal prognosis and long term morbidity is less favorable than is currently anticipated from mere counting of live fetuses.

Follow-up studies on the long-term prognosis for the mother have shown that

eclampsia does not contribute to residual cardiovascular disease [39, 40, 41]. It is the genetic predisposition of the patient that determines whether or not hypertension develops later, since those patients in the 25-year follow-up had a family history of hypertension if they had hypertension after the age of 50.

The one point that should be emphasized is that if a diagnostic problem exists in the hypertensive pregnant patient, a postpartum renal biopsy in the first five days postpartum is helpful in establishing a diagnosis of preeclampsia-eclampsia, hypertensive cardiovascular disease or latent renal disease.

The treatment of hypertensive disease and renal disease have intentionally been omitted, as attention has been focused on the diagnosis of preeclampsia-eclampsia and its therapy. The important point to make regarding vascular disease and renal disease is that an accurate diagnosis is necessary before intelligent management can be achieved and that these are often clinically confused with preeclampsia-eclampsia.

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Management of Abruptio Placentae

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Analyzing 130 cases of abruptio placentae among 26,743 deliveries (1:206), the authors conclude that effective management depends primarily on early recognition of the high-risk patient, immediate hospitalization of all women with third trimester bleeding, prompt institution of liberal whole blood replacement, and early definitive diagnosis. Except when hemorrhage can not be controlled, vaginal delivery should be expected after amniotomy and oxytocin stimulation in mild cases, or if the fetus has died in utero. In moderate and severe cases, if vaginal delivery is not imminent, the viable fetus probably will not survive unless cesarean section is performed as soon as blood replacement has been started and any existing coagulopathy has been corrected.

Despite continuous efforts to improve standards of care for the parturient and her fetus, the United States still ranks 14th among the leading countries of the world in perinatal mortality. Abruptio Placentae accounts for approximately 15% of all such deaths. Recent obstetrical literature has alluded to earlier cesarean section and more liberal replacement of whole blood as means of improving fetal salvage rates and reducing maternal morbidity and mortality from premature placental separation. In order to underscore etiologic factors, appreciate difficulties in diagnosis, and arrive at an optimal method of management of patients with premature placental separation, we have analyzed retrospectively all cases of confirmed abruptio placentae at Kaiser Foundation Hospital, San Francisco, during the nine years 1960 to 1968. Consideration was limited to those instances in which the classic clinical picture was presented, inspection of the placenta revealed retroplacental cloth with depression or disruption of the maternal cotyledons, or both; and in which fetal birth weight was at least 1,000 gm (2.2 lb).

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Incidence; Severity

The frequency of abruptio placentae has been variously reported to be from 0.4% to 3.5% of all deliveries.[1-4] At this hospital during the period reviewed, abruption after the 28th week of pregnancy was diagnosed in 130 (0.48%) of 26,743 deliveries, an incidence of 1:206.

The 130 cases were classified according to severity by the criteria of Page et al[5]: mild (external bleeding only, or mild tetany, no shock), 72 cases; moderate (tetany, uterine tenderness, possibly external bleeding, fetal distress or death, no shock), 35 cases; severe (maternal shock or coagulation defect, tetany, intra-uterine death), 23 cases.

Maternal Survival. – There were no maternal deaths.

Fetal Rate.–Ninety-one (70%) of 130 infants survived. The corrected fetal survival rates for the subgroups were; mild: 61 of 72 which is an 87.1% survival rate, corrected by omission of two infants with congenital anomalies incompatible with life; moderate: 27 of 35 which is 82% survival rate, corrected by elimination of two infants in which fetal heart tones were absent on admission; severe: 3 of 23 which is 30% survival rate, corrected by omission of 13 infants in whom fetal heart tones were absent on admission.

Maternal Age. – (Table 1). The incidence of abruption did not differ significantly according to year of life until age 30 years, after which it rose gradually, until in women older than 40 years the incidence was more than double the frequency in women younger than 30 years.

Parity.–(Table 2). There was a small but nonsig-

nificant positive correlation between increasing parity and frequency of abruption. However, in fifth or later pregnancies, the frequency was more than three times that in first and second pregnancies, and approximately double that in third or fourth.

Gestational Age.—We did not determine the relative incidence of abruption at various gestational ages, but found no statistical correlation between gestational age and severity of abruption. The life risk attendant upon abruption clearly diminished with increasing fetal size, or gestational age, or both. Of the 130 infants, 66 (51.6%) were premature. Only 33 premature infants (50%) survived the neonatal period, while 58 (90%) of the mature infants lived. Our 11.5% incidence of breech presentation (15 infants) reflects the high prematurity rate.

Recurrent Abruption.—Five (7%) of women with mild abruption had previously experienced abruption; three (9%) of the 35 with moderate abruption, and four (16.6%) of the 23 with severe abruption had such a history—a frequency similar to the 17% five times the expected incidence) noted in severe cases by de Valera[6] and by Hibbard and Jeffcoate.[2]

Of more importance, recurrent separation tended to be more severe than the initial episode. One must seriously consider cesarean section for any patient with recurrent abruptio placentae.

Toxemia and Hypertension.—While some authors[3,6,7] have observed toxemia in 16% to 60% of patients with abruption, Hibbard and Jeffcoate[2] recently reported the much lower incidence of 6%. They suggested that toxemia is not important etiologically, but that hypertension may precipitate the accident if placental attachment is insecure for other reasons.

Preeclampsia as determined by hypertension, proteinuria, and hyperreflexia was present in 26 (20%) of our 130 patients. Five (3.8%) had chronic hypertension, diagnosed antenatally. The incidence of toxemia rose from 13.9% in mild cases to 25.7% in moderate and 52.1% in severe cases.

Folic Acid Deficiency.—It has been postulated that folate deficiency early in pregnancy is a common cause of abruption; however, there have been conflicting reports as to its role in this regard.[2,4,6]

Only 26 (20%) of the women in our series were anemic (hematocrit value <34%) on admission; among these, the hematocrit reading was less than 30% in eight patients (6.1%). During 1963 to 1966, when folic acid was not given at our hospitals, there were 57 cases of premature placental separation among 10,669 deliveries; this 0.53% incidence is comparable to the lowest reported in any large series whether or not folic acid was used, and not statistically different from the overall incidence of abruption in our series.

Table 1.—Relationship Between Maternal Age and Incidence of Abruptio Placentae

Age (yr)	Patients (No.)	Abruptio Placentae: Mild	Moderate	Severe	Total	Incidence	%
<20	3,346	6	7	1	14	1:239	0.42
20-24	7,953	25	5	4	34	1:234	0.43
25-29	8,254	21	11	3	35	1:236	0.39
30-34	4,596	10	8	10	28	1:164	0.60
35-39	2,095	7	2	5	14	1:149	0.67
40-44	499	3	1	1	5	1:100	1.00

Table 2.—Relationship Between Parity and Incidence of Abruptio Placentae

Parity	Patients (No.)	Abruption (No.)	Incidence	%
1	9,032	34	1:266	0.37
2	7,068	25	1:283	0.35
3	4,338	24	1:181	0.55
4	2,957	13	1:227	0.44
5	3,348	34	1:98	1.01

Methods of Management and Outcome

Our policies with respect to management of abruptio placentae are presented in approximately chronologic sequence of specific factors: prophylaxis, diagnosis, amniotomy, supportive therapy, mode of delivery, and management of complications.

Prophylaxis.—Until the role of folic acid in the etiology of abruptio placentae is clarified, we feel that folic acid should be included in the daily prenatal vitamin supplement, and that it should be started as early in pregnancy as possible. The 1.0 mg dose contained in most prenatal vitamin compounds is probably sufficient to prevent megaloblastic anemia of pregnancy, but may cause the hematologic picture of pernicious anemia to return to normal while the neurologic features remain progressive.

Diagnosis.—All patients with third trimester bleeding are hospitalized immediately. The sudden onset of severe, continuous abdominal pain, low back pain, or both, followed by vaginal bleeding is considered virtually diagnostic of moderate or severe placental separation.

Careful abdominal palpation may aid in diagnosis before definitive diagnostic procedures are carried out: the uterus is usually tender or irritable, and may be board-like and enlarging. Fetal heart tones may be inaudible, and fetal palpation may be difficult or impossible.

Painless vaginal bleeding without uterine tenderness or irritability does not exclude the diagnosis of abruption. In three of our patients with mild, four

with moderate, and two with severe abruption, painless vaginal bleeding and an admission diagnosis of placenta previa led to delay in aggressive management. This situation was particularly deceptive when gestation was less than 36 weeks and symptoms were so minimal that prolongation of pregnancy in the hope of improving chances of fetal survival was an important consideration. In the group with premature fetus and painless bleeding, close observation for changing signs and symptoms, hematologic evaluation, and placental localization studies (such as scintillation scanning with technetium Tc 99m) are essential for early diagnosis of abruption. Mild placental separation with minimal bleeding and uterine tenderness is often an exclusion diagnosis and is frequently recognized retrospectively.

Definitive diagnosis is made by double set-up examination with anesthesia ready, operative pack open, blood on hand, and the doctors scrubbed.

Amniotomy.—Amniotomy is performed as soon as possible after exclusion of placenta praevia, to decrease intra-amniotic tension and reduce extravasation of blood into the myometrium. This procedure often gives a clue to the presence of intra-amniotic bleeding when external bleeding is absent. If satisfactory labor does not soon follow amniotomy, oxytocin is given intravenously to institute or increase uterine contractions.

Amniotomy was performed in 67 (63%) of the 106 patients with presumed intact membranes. In one, cord prolapse immediately followed the procedure at 8 cm dilatation. A live, 1,304 gm (2 lb 14 oz) infant was delivered vaginally. Amniotomy was supplemented by intravenously administered oxytocin in 40 (30%) of the 130 patients.

Supportive Therapy.—As soon as possible after admission of a patient with abruption, it is our policy to start the intravenous administration of fluids, and to draw blood for typing, crossmatching, and clot observation. If bleeding is profuse, rapid infusion of large amounts of Ringer's solution or dextran 70 often helps to stabilize vital signs until blood is available.

Blood replacement must be rapid, and often massive. It must far exceed estimated blood loss, especially when abruption is moderate or severe, in order to maintain adequate visceral perfusion and to prevent shock and renal damage. It is fallacious to balance blood replacement against estimated blood loss before delivery, because external bleeding may be minimal while retroplacental bleeding may be copious.

In our study, the mild and moderate groups did not differ significantly with respect to estimated blood loss; but estimated blood loss was greatly increased in severe abruption (Table 3). Of the five patients with mild abruption who received blood by transfusion, three were admitted with a diagnosis of placenta praevia and were treated by expectant observation and blood replacement, receiving 500, 1,500 and 2,000 cc of whole blood before the diagnosis of abruption was made. Only 30% of the patients with severe abruption had estimated blood loss as low as 250 to 750 ml; 56% were estimated to have lost between 900 and 1,500 ml of blood.

The term "obstetric shock," implying shock out of proportion to blood loss, is a misnomer. Shock occurring during obstetric hemorrhage is the result of profound hypovolemia due to grossly underestimated blood loss. To determine the efficiency of blood replacement, we compared hematocrit values before delivery and approximately 48 hours post partum, when cardiovascular hemodynamics had stabilized and readjustments in blood volume and hematocrit value had occurred (Table 4). A significant positive correlation was seen between severity of placental separation and underestimation of blood loss and replacement. There was no instance of overhydration or overtransfusion in our series; but many patients received undertransfusions, especially those patients with severe abruption.

During shock, peripheral venous pressure is not an accurate indicator of blood volume, partly because compensatory vasoconstriction maintains blood pressure until this mechanism can no longer adjust the circulatory system to its critically reduced volume. Indeed, this protective mechanism is potentially lethal, leading at the extreme to renal and uterine ischemia, oliguria, fetal anoxia, and pituitary necrosis. Certainly, vasoconstrictors are contraindicated in the management of abruption.

Quantitative determination of blood replacement is best assessed by monitoring the central venous pressure (CVP). While not accurately reflecting blood volume, CVP indicates cardiac competence to accept and expel venous return, and is therefore, an excellent guide to safe blood volume replacement[4] An abrupt rise in CVP over 13 to 15 cm of saline solution warns of impending myocardial failure (a possible result of circulatory collapse), or of acute pulmonary edema, or both. Progressive decline in CVP on serial measurements suggests continuing blood loss. Fluids and drugs should not be infused through the CVP catheter, as they may have a bolus effect on the heart, leading to cardiac arrhythmia or arrest.

Management of Coagulation Failure.—Coagulation failure is usually due to hypofibrinogenemia, but may also be associated with abnormalities in platelet function.[9] Prompt delivery unaccompanied by trauma usually limits progression of the coagulopathy and eliminates the need for further therapy, assuming uterine contraction is maintained. If

coagulation failure does not occur within eight hours after the onset of placental separation, it probably will not occur at all.[10]

Cesarean section performed before correction of hypofibrinogenemia almost always causes persistent bleeding from all cut surfaces. Fibrinogen should be replaced to approximately 150 mg per 100 ml before cesarean section[10]; this requires approximately 4 to 6 gm.

Hypofibrinogenemia was clinically apparent in six (26%) of our 23 patients with severe placental separation. This compares favorably with the 38% incidence reported by Pritchard and Brekken[10] in 141 patients with severe abruption. Four of our patients received 2 to 6 gm of fibrinogen. Two who delivered vaginally shortly after admission did not receive fibrinogen, and stopped bleeding just after delivery. Severe thrombocytopenia, with normal fibrinogen levels, developed in one patient who was toxemic (blood pressure 200/100 mm Hg) during labor. She was delivered vaginally 2½ hours after admission, following amniotomy, intravenously administered oxytocin, and magnesium sulfate therapy. Immediately post partum, the patient continued to bleed from the uterus and from a large vaginal hematoma, went into shock, and rapidly became oliguric, comatose, and decerebrate, either from an unrecognized eclamptic seizure or from intracranial hemorrhage. Her blood loss was estimated at 2,500 ml; she received 6,000 ml of whole blood and 8 units of platelets. After 13 days of unresponsiveness, the patient's condition improved and she was discharged on the 20th hospital day.

Antifibrinolytic Agents.—The indications for use of antifibrinolytic agents such as epsilon-aminocaproic acid (EACA) are fetal death in utero with profuse generalized bleeding, and fibrinogenolysis, as determined by rapid clot lysis. Epsilon-aminocaproic acid should not be used while the fetus is alive, as it crosses the placenta and may cause fetal coagulopathy.

Recently, the use of heparin sodium in the treatment of intravascular coagulation has been suggested.[11,12] Antifibrinolytic agents should probably be used only in combination with heparin because of the risk that they may maintain intravascular thrombi in patients with continuing intravascular clotting even though the fibrinolytic system has been activated.[12] We have not had occasion to use EACA or heparin in any of our patients.

Delivery.—Mild placental separation is managed by amniotomy and stimulation of the uterus through the intravenous administration of oxytocin, in the anticipation of vaginal delivery. If symptoms progress or fetal heart tones become irregular, we are prepared to perform cesarean section immediately. In moderate or severe abruption, if fetal heart tones are audible the combination of rapid blood replacement, correction of coagulation failure, and cesarean section offers the best chance of fetal salvage. Many authors have achieved high salvage rates (79%,[5] 81.5%,[13] and 91%[14]) by early cesarean section, before the onset of shock and coagulation difficulties.

If the fetus dies in utero, the patient's condition should be managed conservatively by blood replacement, correction of blood coagulopathy, and induction or stimulation of labor in the anticipation of vaginal delivery. Cesarean section is indicated only if hemorrhage persists after stabilization or if labor does not progress after three or four hours; it then becomes important, as continued intra-uterine bleeding and defibrination may lead to irreversible shock and maternal death.

Of our 29 cesarean sections, 25 were performed in attempts to salvage live infants; 23 infants (92%) survived. Sixty-eight (67.3%) of the 101 babies delivered vaginally survived. Five of the 72 women with mild abruption were delivered by cesarean section all for reasons other than abruptio placentae. In the moderate group, all 15 infants delivered by cesarean section survived. Among 20 babies delivered vaginally, six died during the neonatal period. Nine of the 23 women with severe abruption were delivered by cesarean section. In five cases, in which the procedure was an attempt to salvage a live infant, three infants survived. In these three instances, cesarean section was performed within 90 minutes after diagnosis. The other four cesarean sections were performed in the inter-

Table 3.—Estimated Blood Loss and Blood Replacement

	Abruptio Placentae					
	Mild		Moderate		Severe	
Estimated Blood Loss (ml)	No.	%	No.	%	No.	%
<2500	9	12	5	15	0	0
250-750	53	74	23	65	7	30
>750	10	14	7	20	16	70
Blood Replaced (ml)						
500	1		1		2	
1,000	1		4		5	
1,500	2		3		6	
2,000	1		1		1	
2,500	...		1		1	
6,000	...		...		1	
6,500	...		...		1	

Table 4.—Comparison Between Predelivery and Postdelivery Hematocrit Values

	Abruptio Placentae					
	Mild		Moderate		Severe	
Difference %	No.	%	No.	%	No.	%
≦ 5	55	76	20	57	7	31
6-9	14	20	11	32	6	26
≧10	3	4	4	11	10	43

Table 5.—Diagnosis-Delivery (D-D) Interval Compared to Corrected Fetal Survival

D-D Interval (hr)	Abruptio Placentae			Corrected Fetal Survival					
				Mild*		Moderate*		Severe*	
	Mild	Moderate	Severe	No.	%	No.	%	No.	%
<2	44	20	10	42	95	19	95	3	30
2-4	15	7	5	10	67	5	71	...	...
4-6	7	3	2	6	86	2	67	...	...
6-8	1	0	3	...	...	...	...	...	...
>8	3	3	3	3	100	1	33	...	...
Totals	70	33	23	61	87.1	27	82	3	30

*Degree of abruptio placentae. Total survival rate: 70%.

est of a mother in shock, with hypofibrinogenemia, or both, whose condition had been stabilized, and who failed to progres in labor after amniotomy and intravenous oxytocin stimulation.

Postpartum Complications.—Six (26%) of the patients with severe abruption were in shock on admission or during labor. Three became oliguric secondary to hypovolemic shock. There were no instances of renal cortical necrosis. Prompt whole blood replacement, in amounts determined to be adequate by CVP monitoring, maintained visceral circulation and prevented serious renal damage. The postpartum patient who is oliguric after adequate blood replacement may be given a trial of mannitol therapy. If there is no increase in urine output, mannitol is stopped and the usual therapy for acute tubular necrosis is instituted.

Once the immediate problem is corrected and the infant delivered, the incidence of puerperal complications is small. Only 15% of our patients had postpartum complications other than oliguria: endometritis (nine patients), urinary tract infection (three patients), postpartum hemorrhage (4 patients), and one instance each of septicemia and ruptured uterus. Three patients with severe abruption were found on cesarean section to have Couvelaire uterus. In none of these was hysterectomy performed. In one woman with moderate abruption, cesarean hysterectomy was done because of large leiomyomata.

Fetal Outcome

Diagnosis-Delivery Interval.—The diagnosis-delivery (D-D) interval is related to corrected fetal survival in Table 5. The D-D interval was less than four hours in 59 (84%) of the 72 patients with mild abruption. In this group there was no correlation between fetal salvage rate and D-D interval. Two infants with congenital anomalies incompatible with life were eliminated from the analysis. Two potentially preventable deaths occurred: A 1,449 gm (3 lb 5 oz) fetus was delivered six hours after diagnosis of abruptio placentae; the fetal heart tones had ceased three hours before delivery. A 2,467 gm (5 lb 7 oz) baby, born 2½ hours after diagnosis, died of respiratory distress syndrome and pulmonary hemorrhage. The remaining five infants who died weighed under 1,619 gm (3 lb 11 oz); in two of these, the diagnosis of abruption was made at the time of delivery. The D-D interval was under four hours in 27 (82%) of the 35 patients with moderate abruption. Of the six neonatal deaths, only two were potentially preventable. The other four babies who died weighed under 1,162 gm (2 lb 9 oz); all were delivered within six hours of diagnosis. The three surviving infants of the severe group were delivered within 90 minutes of diagnosis. In two patients, fetal heart tones disappeared within 30 minutes of admission, before delivery could be effected. There were three neonatal deaths, none of which was considered preventable; each of the three infants weighed under 1,591 gm (3 lb 10 oz). In the moderate and severe groups, there was a significant inverse correlation between fetal survival and D-D interval: the shorter the D-D interval, the greater the fetal survival rate.

Among the fetal complications of abruptio placentae are premature birth, hyperbilirubinemia, anemia, and hyaline membrane disease. Irrespective of maturity, the fetus is more likely to be depressed at birth if abruption has occurred than if delivery has been normal. Neurologic evaluation during the first year of life has demonstrated that the premature infant born after placental separation is at considerable disadvantage.[13] Birth weight and Apgar score appear to be reliable indices of fetal prognosis in abruptio placentae.

Pulmonary hemorrhage as a complication of placental disruption deserves comment. The fetus lives in a basically hypoxic environment[15]; and fetal hypoxia, which is intensified by premature placental separation, has been considered to be a major cause of neonatal pulmonary hemorrhage, especially in the premature infant with diminished respiratory movements. In fact, pulmonary hemorrhage may represent a more severe form of the pathologic process responsible for hyaline membrane disease.[16] A bleeding tendency has been noted in newborns with respiratory distress.[16,17] It is interesting to postulate an association between subclinical hypofibrinogenemia and hypoxia of abruption, and the development of hemorrhagic complications in the newborn. Perhaps earlier delivery by cesarean section in all but the mildest cases of abruption would avert this event in the neonate.

Of the 130 babies in our series, 111 (85.3%) were born alive, and 91 (70%) survived the neonatal period. Fifteen (11.6%) had died in utero before admission. Four others (3.1%) died after admission, but before delivery. Twenty died during

the neonatal period; 19 of these weighed less than 2,500 gm 5.5 lb).

Hyperbilirubinemia was noted in seven babies, six of whom were premature. Two exchange transfusions were performed, both in premature infants.

Hemoglobin levels were below 17 gm/100 ml in 4% of the neonates—approximately twice the expected incidence of fetal anemia. Although low fetal hemoglobin values are rarely encountered, awareness of this possibility may prevent further fetal morbidity.

Respiratory distress syndrome occurred in five premature infants, and was the cause of death in four.

Conclusions and Summary

Part of the problem of management of abruptio placentae is recognition of high-risk patients. In this group we include women of high parity and advanced age; and those with chronic hypertension, preeclampsia, poor nutritional status, or a history of previous placental separation.

All patients with third trimester bleeding are hospitalized immediately. Management is aggressive, with rapid and liberal whole blood replacement and correction of coagulation failure.

The diagnosis of abruption should be made as early as possible, preferably by "double set-up" examination. Blood loss and replacement, which are often grossly underestimated, should be monitored by CVP, especially in severe cases. In mild cases, vaginal delivery should be anticipated after amniotomy and stimulation with intravenously administered oxytocin. In moderate and severe cases, if fetal heart tones are audible, unless vaginal delivery is imminent within minutes, cesarean section should be performed as soon as blood replacement has been started and coagulopathy (if it exists) has been corrected. Cesarean section is not usually indicated if the fetus has died in utero, unless hemorrhage continues after adequate blood replacement and correction of coagulation failure, or unless labor fails to progress after amniotomy and intravenous oxytocin stimulation.

It is only by the liberal and early use of whole blood transfusions and rapid cesarean section at an early stage of the abruption process, that we will allay the onset of severe and potentially irreversible maternal complications, and can hope to increase the incidence of fetal survival.

Nonproprietary and Trade Names of Drugs

Technetium Tc 99m—*Neimotec, Perteg-99m, Technekow Generator, Technekow-CS Generator, Technetope.*
Oxytocin—*Pitocin, Syntocinon, Uteracon.*
Dextran 70—*Macrodex.*

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THE PREGNANT DIABETIC

No other patient in obstetrical practice requires the expert liaison between internist, obstetrician and eventually the pediatrician for care than the pregnant diabetic. Diabetes mellitus complicates about one in every 325 pregnancies, and latent or gestational diabetes may have an equal incidence. Maternal mortality is only slightly increased, but morbidity is considerably worsened. Perinatal mortality is still about 10-25% and the incidence of anomalies in the offspring of diabetic patients is five times the average.

Before the advent of insulin in 1921 most women with diabetes were too ill to conceive. The exact cause of the infertility in diabetic women during the preinsulin era is not clear, but amenorrhea was common, the incidence being as high as 50%. In the infrequent cases in which pregnancy did occur, about one-fourth of the mothers and one-half of the fetuses and infants died.

With the advent of insulin, and increased knowledge concerning causes and treatment of diabetes, pregnancy in diabetic women became more common. The outcome of the diabetic pregnancy depends on the seriousness of the disease and the management the patient receives during the gestational period.

For purposes of identification, diabetic patients are classified under a system defined by Dr. Patricia White. They are as follows:

Class A - Chemical diabetes only

Class B - Diabetes with adult onset (after age 20 years)

Class C - Diabetes of long duration (10 to 19 years)

Class D - Diabetes more than 20 years, or onset before age 10, or with vascular lesions, benign retinopathy, or calcified leg arteries.

Class E - Diabetes with calcification of iliac or uterine arteries

Class F - Diabetes with nephropathy

It is apparent from many studies that maternal prognosis depends on the classification of the diabetes and good medical and obstetrical care throughout

the pregnancy and puerperium. Today, almost all maternal deaths from diabetes occur because of less than optimal care.

The prognosis for the fetus, although vastly better than in the pre-insulin era, is still guarded. It depends to a considerable degree on the intensity of the diabetes, its duration, the presence of maternal renal, or vascular disease and the development of complications during the pregnancy.

Some of the more complex problems that medical personnel must deal with in the pregnant diabetic are: (1) nausea and vomiting in early pregnancy, a common complaint, may lead to insulin shock, or if prolonged and severe (hyperemesis) could lead to ketosis. (2) Infection is more prevalent in diabetics, and commonly results in insulin resistance and ketoacidosis unless promptly recognized and treated. (3) There is usually a changing insulin requirement as the pregnancy progresses, requiring close monitoring by the patient and her physician. (4) The normal lowering of the renal glucose threshold during pregnancy can cause confusion to the diabetic patient as she checks her urine. (5) Close supervision and education of the patient to gain a cooperative attitude from her is essential.

There are several Obstetrical complications that are common in pregnant diabetics. They are: (1) Pre-eclampsia and eclampsia occurence is 3-4 times greater. (2) Congenital anomalies occur 5 times more often, (3) macrosomia - the large size of the fetus often leads to problems in labor and delivery. (4) Higher incidence of infection, and greater severity of infection. (5) Intrauterine fetal demise after 37th week of gestation (6) Polyhydramnios (7) Post-partum hemorrhage.

Although increased knowledge and diagnostic tests have helped to decrease the maternal and perinatal mortality and morbidity rates, it is felt that there is room for further intensive investigations. The management of the pregnant diabetic should include consideration of the following: A select group of physicians

skilled in the treatment of diabetes and in obstetrics should assume primary responsibility for care throughout the pregnancy. At the time of delivery, an anesthesiologist cognizant of the problems of the diabetic mother and her fetus, is a desirable addition, and a pediatrician, skilled in the care of high-risk infants, should immediately assume the responsibility for the care of the infant. Ideally, the patient will be seen early in her pregnancy, and follow-up appointments made every two weeks during the first and second trimesters, and weekly during the last trimester. Education for the mother as to the close checking of her urine and what and when she should report to her physician is of utmost importance. Hospitalization may be needed if problems of diabetic control arise. These may be for brief periods from time to time until control is re-established, and the patient well-educated to carry on at home.

Adjustments in insulin dosage must be supervised closely. Most physicians follow the blood plasma glucose levels rather than the urine, since the lowered renal glucose tolerance level tends to distort somewhat the significance of the glucosuria. Another accepted practice of better control is the splitting of the insulin dosages so the patient does not receive one large dose in the morning and none the rest of the day. Dr. Joseph Brown of the University of Iowa Department of Internal Medicine advocates the insulin be regulated in this manner if the patient is taking over 40 units of insulin per day:

AM dosage: 50% of requirement in NPH Insulin
15% of requirement in Regular Insulin

PM dosage: 35% of requirement in NPH Insulin

The patient is also given a mid-morning, mid-afternoon, and evening snack, so is able to carry through the 24 hour period with more even levels both in insulin and nourishment. He also advocates some salt restriction -- a "no-added" salt diet is usually recommended.[1]

measurements of the amount of glucose administered IV must be kept, and a liter of 5% Glucose in water is counted as a replacement for each missed meal. After delivery the insulin requirement usually decreases markedly, and it may fluctuate for a few days. It is usually felt that the patient will stabilize at her pre-pregnancy requirements after about 72 hours.

The patient who is classified as a Class A (chemical) diabetic may also become a challenge during pregnancy. These patients need close supervision, and should be given small amounts of insulin if the need arises. The use of an oral hypoglycemic agent (Orinase) is contraindicated in pregnancy, because of its tendency to produce severe hypoglycemia in the infant. These patients usually can be allowed to progress to term in their pregnancies, providing they are closely supervised by the physician and remain free of obstetrical complications.

The diabetic mother ideally should be delivered in a medical center where all facilities are provided. The availability of an anesthesiologist, immediate availability of blood for transfusion, rapid Cesarean section capabilities, electronic monitoring of labor, and an intensive care neonatal care nursery to receive the infant are of the utmost importance in the successful outcome of these pregnancies.

Birth control information should be readily available to the diabetic mother, either by permanent means if desired, or by reversible practices.

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Prognostic index for vaginal delivery in breech presentation at term

Prospective study

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The obstetric decision of allowing the vaginal delivery of a term fetus in breech presentation is fraught with danger. In our present state of knowledge, the choice of abdominal versus vaginal delivery in these cases resides mostly in the obstetrician's experience. It is unfortunate that this decision is the wrong one on far too many occasions. Retrospective analysis of term breech presentation has led us to the development of a Breech Scoring Index. The present study, a prospective one, emphasizes the usefulness of the Index in selecting those patients whose labor should be terminated by the abdominal route. Experience with the Breech Index demonstrates that approximately 20 per cent of patients with a term fetus in breech presentation will require cesarean section. The morbidity and mortality associated with breech presentation will be significantly decreased by the use of the Breech Index.

IN THE Collaborative Study of Cerebral Palsy,[1] it was shown that abnormal neurologic development and retarded motor development occurred twice as often when the infant was delivered as a breech presentation. Morgan and Kane,[2] in their cooperative hospital survey of 16,327 breech births, demonstrated that the fetal mortality, when the breech presents, is three and one-half times as high with a cephalic presentation, even after correction for prematurity. Many large surveys[3, 4, 5] have shown that the underlying cause for this increase in fetal mortality and morbidity with breech delivery at term is unrecognized fetopelvic disproportion.

Pelvic diameters measured radiologically may be adequate for a cephalic presentation, but inadequate for the same infant in breech presentation. This is so because the aftercoming head has no opportunity to mold. Further, a small or large degree of fetocervical extension and rotation inherent in every breech delivery may introduce diameters larger than the biparietal diameter.

The use of ultrasound techniques for determining the actual biparietal diameter is extremely accurate. However, the same criticism applies to this diagnostic modality as stated above for the use of radiologic techniques.

The decision of the obstetrician to allow the vaginal delivery of a breech presentation must be dependent upon many factors. In a previous paper,[6] we analyzed these pertinent factors and established in retrospect the validity of a Breech Index to select those patients who probably should be delivered by cesarean section.

Table I depicts the pertinent data required and the points ascribed to each factor. The total score may range from 0 to 11. Worthy of notation is the fact that some period of labor must have existed prior to admission

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Table I. Criteria for scoring

	Points		
	0	*1*	*2*
Parity	Primigravida	Multipara	
Gestational age	39 weeks or more	38 weeks	37 weeks or less
Estimated fetal weight	Over 8 pounds (3,630 grams)	7 to 7 pounds, 15 ounces (3,629-3,176 grams)	Less than 7 pounds (< 3,175 grams)
Previous breech*	None	One	Two or more
Dilatation†	2 cm.	3 cm.	4 cm. or more
Station†	−3 or higher	−2	−1 or lower

*Greater than 2,500 grams.

†Determined by vaginal examination on admission.

to the hospital. The scoring system cannot be applied to those patients scheduled for elective induction.

We have shown that the complications of labor and delivery in breech presentation at term occurred almost exclusively in those patients who had a score of 3 or less. All patients delivered by cesarean section were in the low-score group. In addition, many low-score patients who were delivered vaginally had severely traumatized infants. Four deaths occurred in the low-score group, all directly related to difficulty in extracting the after-coming head.

Of the 137 patients with high scores (4 or more), only one infant required endotracheal resuscitation. There was no fetal mortality or morbidity related to vaginal delivery in this entire group.

With these retrospective results in mind, we stated that all patients who had a score of 3 or less should be delivered abdominally. When the score is 4, careful re-evaluation of the patient and the size of the fetus should be accomplished. If this evaluation remains the same, then vaginal delivery should be safe. When the score is 5 or more, there should be no difficulty with breech delivery by the vaginal route.

Further, when the score is high and abnormal labor develops, oxytocic stimulation may be used to advantage. Oxytocic stimulation in the low-score group is to be avoided, as in almost all instances the background for the low score is fetopelvic disproportion.

The prospective study has been done to properly evaluate the Breech Index. In this study no direct attempt was made to influence the management of any patient. The factors involved in the scoring system were indicated on work sheets provided to the staff upon admission of the patient to the labor suite. Follow-up of each patient was then accomplished by the authors.

Table II. Type of breech delivery

Type	*No.*	*%*
Spontaneous	7	5.0
Partial extraction	76	54.7
Complete extraction	32	23.1
Cesarean section	24	17.2
Total	139	100.0

Materials

This report covers the period from Sept. 1, 1963, to April 30, 1966. During this time there were 6,117 deliveries at Temple University Health Sciences Center, of which 218 (3.4 per cent) were breech presentations. Upon eliminating premature births, cases of severe congenital anomalies, prolapsed cord cases, and bleeding placental problems, 139 cases are available for analysis.

Primigravidas constituted 37 per cent (51 patients) of the series. The ages for all patients ranged from 14 to 43 years, with 16 patients over the age of 35.

Results

Table II shows the types of delivery utilized by the staff and residents. Table III demonstrates the distribution of scores and the correlation of outcome. Table IV is a

Table III. Score distribution and correlation of outcome

	Points									
	Low score				*High score*					
	0	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*
Total patients	1	8	13	8	10	42	28	17	8	4
Abnormal labor	1	7	11	4	2	5	3	2	0	0
Cesarean section	1	7	9	6	0	1	0	0	0	0
Vaginal delivery (complicated)	0	1	3	2	1	0	0	0	0	0
Corrected breech mortality	0	0	1*	0	0	0	0	0	0	0
Neonatal morbidity	0	1†	2‡	2§	0	0	0	0	0	0

*Anoxia, convulsions, intracerebral hemorrhage.

†Brachial palsy.

‡Anoxia, pneumonia, pneumothorax.

§VII nerve palsy, apneic episodes, convulsions.

Table IV. Duration of labor (vaginal delivery)

	Breech index	
	Low score (0-3)	*High score (4-11)*
Nulliparas		
First stage	10 hr., 34 min.	7 hr., 52 min.
Second stage	1 hr., 29 min.	41 min.
Multiparas		
First stage	8 hr., 10 min.	5 hr., 46 min.
Second stage	48 min.	23 min.

comparison of the duration of labor of the high-score versus the low-score group.

When the infants' weights were analyzed, it was found that 76 per cent of the low-score group weighed in excess of 3,500 grams as compared with only 21 per cent of the high score group.

Table V shows the results of oxytocic stimulation when abnormal labor occurred in both groups.

Comment

The cesarean section rate in the retrospective study was 6 per cent. Had all low-score patients been delivered abdominally, the section rate would have been 21 per cent. This would have avoided 4 infant deaths and many instances of severe anoxia, infant and maternal trauma, and perhaps the later sequelae of cerebral palsy and retardation in general.

In the prospective study, the cesarean section rate was 17.2 per cent (24 patients). There was one case of fetal death and 5 cases of severe neonatal morbidity. Had all patients with a low score been delivered abdominally, the section rate would have been 21.5 per cent (30 patients). The mortality and morbidity occurred only in those infants who had a low score and who were delivered by the vaginal route.

When the duration of labor was analyzed, it was found that labor was definitely prolonged in the low-score group, regardless of parity. Further, the infant weights were significantly higher in the low-score group. The neonatal morbidity indicates that the low-score fetus has a poor chance of being safely delivered vaginally.

The use of oxytocin is not warranted in those patients with abnormal labor and low score. Seventeen patients in the low-score group were given oxytocic stimulation because of abnormal labor. Thirteen were subsequently delivered abdominally without fetal morbidity. Four infants were delivered vaginally and, in all cases, severe morbidity resulted.

In 12 high-score patients with abnormal labor, 11 were given oxytocin. All were delivered vaginally without morbidity.

Many[3, 4] have suggested that a reasonable cesarean section rate in term breech presentation would be about 20 per cent. The vagueness of *which* 20 per cent has con-

Table V. Oxytocic stimulation in abnormal labor

Breech index	*No.*	*Oxytocic stimulation*		*Cesarean section*	*Vaginal delivery*	*Morbidity*
Low score (0-3)	23	Oxytocic	17	13	4	4
		No oxytocic	6	5	1	0
High score (4-11)	12	Oxytocic	11	0	11	0
		No oxytocic	1	1	0	0

tinually perplexed obstetricians. The use of the Breech Index will indicate those patients who should best be delivered abdominally. The Breech Index may also be used to advantage in abnormal labor situations when oxytocic stimulation is being considered.

We have found the Breech Index to be a simple device which represents sound clinical judgment. Its use should immediately alert the obstetrician of an impending difficult breech delivery and thereby save the patient and her physician hours of fruitless procrastination or, even worse, an abnormal or dead baby.

Summary

A prospective study to validate the use of the Breech Index has been accomplished.

The results confirm the value of the Breech Index as a means of selecting those patients who should be delivered abdominally.

The proper use of the Breech Index will probably give a cesarean section rate of approximately 20 per cent.

With proper use of the Breech Index, the high fetal mortality and morbidity associated with breech presentation at term can be significantly reduced.

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OBSTETRICS

Management of breech presentation at term

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In a retrospective analysis of 2,145 cases of singleton term breech presentation, the corrected perinatal mortality rate was 3 times higher and the neonatal traumatic morbidity rate was 12 times higher than in singleton term vertex presentation. The poor pregnancy outcome was concentrated among infants delivered vaginally; there was no excess perinatal mortality rate among singleton term breech infants delivered by cesarean section, although neonatal traumatic morbidity was 5 times higher than for vertex presentation. Based on this experience, a series of recommendations are derived, designed to improve pregnancy outcome in term breech presentation: (1) Indications for cesarean section should be liberalized, but routine elective cesarean section for term breech presentation is not justified; (2) vaginal delivery should be considered if maternal pelvic measurements are known and are at least of mean normal dimensions and the estimated fetal weight is not excessive; (3) breech labor and delivery should be supervised by the most experienced obstetrician available and effected with the least obstetric manipulation possible; and (4) constant monitoring of the fetal heart is essential to prevent asphyxic perinatal death.

UNIQUE problems confront the obstetrician in the management of term breech presentation. To delineate the problems pertaining specifically to pregnancy outcome and to evaluate the applicability of modern diagnostic techniques to their alleviation or solution, a retrospective analysis was made of breech presentation at term at The Mount Sinai Hospital, New York, New York, over an interval of 18 years (1953 to 1970).

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Study population

Between 1953 and 1970, inclusively, there were 86,812 deliveries at The Mount Sinai Hospital, New York. Breech presentation at delivery was encountered in 3,594 patients, an incidence of 4.1 per cent. Overall perinatal deaths in breech presentation were 159 per 1,000 deliveries (/M), approximately 8 times higher than the gross perinatal mortality rate in vertex presentation (19.5/M).

Since breech presentation occurs more fre-

Table I. Perinatal deaths associated with breech presentation at term

Vaginal delivery			
Antepartum deaths			17
Maternal diabetes		4	
Erythroblastosis		3	
Congenital anomaly		2	
Abruptio placentae		1	
Cord accident		1	
Cause undetermined		6	
Intrapartum deaths			28
During labor		18	
Congenital anomaly	9		
Prolapsed cord	6		
Abruptio placentae	1		
Cause undetermined	2		
During delivery		10	
Intracranial hemorrhage	3		
Fetal distress	2		
Ruptured uterus	1		
Bilateral nuchal arms	1		
Cause undetermined	3		
Neonatal deaths			18
Immediate (24 hr. or less)		12	
Congenital anomaly	7		
Erythroblastosis	2		
Traumatic delivery	2		
Respiratory distress syndrome	1		
Late (over 24 hr.)		6	
Congenital anomaly	3		
Pneumonitis/atelectasis	2		
Traumatic delivery	1		
Cesarean section			
Intrapartum deaths			2
Abruptio placentae	1		
True knot in cord	1		
Neonatal deaths			3
Congenital anomaly	1		
Abruptio placentae	1		
Erythroblastosis	1		
Total perinatal deaths			68

Table II. Perinatal deaths related to breech presentation at term

Related to presentation alone			13
During labor		8	
Prolapsed cord	6		
Cause undetermined	2		
During delivery		5	
Fetal distress	2		
Cause undetermined	3		
Related to obstetric delivery			11
During delivery		5	
Intracranial hemorrhage	3		
Ruptured uterus	1		
Bilateral nuchal arms	1		
Neonatal deaths		6	
Traumatic delivery	3		
Pulmonary complications	3		
Total causally related perinatal deaths			24

quently with increasing prematurity, which carries its own high perinatal mortality rate, and because the mechanical problems of delivery differ greatly, the analysis focused on the delivery of singleton infants with breech presentation at term; for this purpose, "term" was defined as the delivery of an infant weighing 2,500 grams or more.

During the study interval, there were 2,145 singleton breech deliveries at term, which constituted 59.8 per cent of all breech deliveries. The incidence of singleton breech presentation at term was only 2.7 per cent and that of breech presentation at premature delivery was 18.5 per cent. Over-all perinatal mortality rate associated with singleton breech presentation at term was 31.7/M, approximately 4 times higher than the gross perinatal mortality rate for vertex presentation at term (8.4/M).

Immediate pregnancy outcome

There were 68 perinatal deaths (31.7/M) among the 2,145 cases of singleton breech presentation at term. The proximate causes of these deaths are detailed in Table I. Of the 68 deaths, 44 (64.7 per cent) were either unrelated to or possibly the cause of the breech presentation. The incidence of major or lethal congenital malformations noted in the immediate neonatal period was 2.1 per cent in term breech presentation versus 0.8 per cent in term vertex presentation. When adjusted for fetal congenital anomalies and perinatal deaths resulting from maternal disease, the perinatal mortality rate causally unrelated to term breech presentation and/or delivery (5.6/M) was identical to that encountered in term vertex presentation.

There were 24 perinatal deaths (35.3 per cent) in which breech presentation and/or delivery were possible etiologic factors (Table II). In this series, there were no antepartum fetal deaths causally related to breech presentation. Intrauterine asphyxia was the cause of 13 fetal deaths: In 6 cases, the etiology (prolapse of the umbilical cord)

was established; in 2, there was clinical evidence of fetal distress before delivery, but the specific cause remained undetermined; and in 5, no etiologic factor was apparent. Injuries incurred during traumatic delivery or undue delay in delivery resulting from mechanical dystocia accounted for 8 perinatal deaths. Three neonatal deaths resulted from pulmonary complications (pneumonitis/atelectasis, 2; respiratory distress syndrome, 1). The incidence of neonatal death from pulmonary complications (1.4/M) did not differ significantly from that associated with vertex delivery at term (1.1/M). Although it is possible that pulmonary complications may have resulted from aspiration during breech delivery, this etiologic factor represented no excess risk of perinatal death and was not considered in subsequent analyses.

There were no perinatal deaths attributable to either presentation or delivery in singleton term infants with breech presentation and delivered by cesarean section.

This left a residual perinatal mortality rate in breech presentation at term and associated with labor and vaginal delivery which exceeded the perinatal mortality rate associated with term vertex delivery by 12.3/M. Excess perinatal mortality rate (EPM) associated with breech presentation alone was 7.6/M; EPM resulting from obstetric delivery was 4.7/M. This was a statistically very significant increase in perinatal mortality rate ($p < 0.0001$) for term breech vaginal delivery (17.9/M) as compared to term vertex vaginal delivery (5.6/M), approximately a threefold increase in risk, and became the subject of additional analyses.

Another significant difference between breech and vertex vaginal delivery at term was neonatal morbidity. Traumatic morbidity, which included fractures, dislocations, and peripheral nerve injuries diagnosed during the immediate neonatal hospital stay, occurred with an incidence of 1.2 per cent following term breech vaginal delivery, as opposed to 0.1 per cent after term vertex vaginal delivery. Neonatal morbidity caused by pneumonitis, atelectasis, and/or respiratory distress syndrome had an incidence of 0.9 per cent after term breech vaginal delivery and 1.0 per cent after term vertex vaginal delivery. The twelvefold increase in traumatic morbidity presented a significant problem for analysis; the unaltered neonatal pulmonary morbidity (as in the case of neonatal death caused by pulmonary complications) did not.

Definition of terms

Imprecision in the use of obstetric terminology has contributed to misinterpretation of data concerning breech delivery. Therefore, a definition of terms employed in this report and in the obstetric records of the institution during the study interval is stipulated.

Breech presentation. This is a longitudinal fetal lie in which the fetal pelvis is the leading pole.

Frank breech. This is a breech presentation in which the fetal lower extremities are flexed at the hips and extended at the knees.

Complete breech. This is a breech presentation in which the fetal lower extremities are flexed at both the hips and the knees. (In this report, the term "complete breech" includes the "footling breech," in which one or both fetal lower extremities are extended and one or both feet are presenting. There is some ambiguity in these terms: A fetus reaching term as a complete breech may at times convert into a footling breech during labor or with rupture of membranes, while a fetus at term with one or both feet presenting sometimes evolves during labor into a complete breech.)

Spontaneous breech delivery. The entire delivery of the infant is produced by the natural forces of the mother, with no assistance other than support of the baby as it is being born. (This includes the Bracht and Mauriceau-Smellie-Veit maneuvers.)

Assisted breech delivery. The infant is delivered by maternal natural forces as far as the umbilicus; the remainder of the baby is extracted by the attendant.

Breech extraction. The entire body of the infant is extracted by the attendant. (This

includes traction on an extended or partially flexed lower extremity in a complete breech, and digital traction in the groins to deliver the buttocks in a frank breech.)

Breech decomposition. This is the intrauterine conversion of a frank into a footling breech by flexing the fetal knee(s) and extending the hips prior to extraction. (This includes the Pinard maneuver.)

Complications of breech vaginal delivery

Perinatal death and morbidity resulting purely from breech vaginal delivery were due entirely to trauma. These complications are presented and analyzed in terms of possible etiologic factors in Table III.

Presentation. Frank breech presentation was encountered in three fourths of the cases of singleton term breech vaginal delivery. There was no statistically significant difference in either traumatic EPM or in traumatic morbidity between frank and complete breech presentation.

Operation for delivery. The method of delivery influenced traumatic EPM and traumatic morbidity. The absence of traumatic EPM in spontaneous breech delivery statistically was very significant; the observed differences in traumatic EPM between assisted breech delivery and breech extraction and the absence of traumatic EPM among the few cases of breech decomposition and extraction were not. A separate analysis revealed a very significant preponderance of infants in the 2,500 to 2,999 grams birth weight range in the spontaneous breech delivery group. Traumatic morbidity was progressively and significantly higher with each step of increasing manipulation to effect delivery.

Forceps to the after-coming head (FACH). In almost one third of term breech vaginal deliveries, forceps were employed to extract the after-coming head. The use of FACH was not associated with a significant increase in traumatic EPM, but was associated with a highly significant increase in traumatic morbidity. A separate analysis showed that the use of FACH was concentrated among infants whose birth weights exceeded 3,500 grams, and included all infants with birth weights of 4,000 grams or more.

Parity. Patients in this series were divided evenly between primigravidas and multiparas. Maternal parity had no statistically significant influence on either traumatic EPM or traumatic morbidity.

Birth weight. Both traumatic EPM and traumatic morbidity were very significantly lower in the 2,500 to 2,999 gram birth weight range, and very significantly increased when the birth weight was 4,000 grams or more. Birth weights in the middle ranges (3,000 to 3,999 grams) had no significant effect.

Use of oxytocin. Oxytocin was administered for induction or stimulation of labor or both to one third of the patients who were delivered of a term infant with breech presentation vaginally. The use of oxytocin bore no significant relationship to either traumatic EPM or traumatic morbidity.

Premature rupture of membranes. Premature rupture of membranes, duration of latent period, and duration of ruptured membranes to delivery had no significant influence on traumatic EPM or traumatic morbidity.

Duration of labor. Duration of labor up to 18 hours had no significant influence on either traumatic EPM or traumatic morbidity, but both increased very significantly when labor lasted 19 hours or longer. Separate analysis indicated that prolongation of labor beyond 18 hours involved a significant preponderance of infants weighing in excess of 3,500 grams at birth.

Maternal prepregnancy weight. Maternal prepregnancy weight bore no significant relationship to traumatic EPM. Traumatic morbidity was increased significantly when maternal prepregnancy weight was 180 pounds or more. The 3 infants involved were in the lower birth weight range ($< 3{,}499$ grams).

Attendant at delivery. One third of the patients in this series were service cases, and two thirds had private obstetricians. Both traumatic EPM and traumatic morbidity were significantly higher in the hands of the less experienced attendants.

Table III. Traumatic EPM and morbidity in breech vaginal delivery

Parameter	Cases		Deaths		Morbidity	
	No.	%	No.	/M	No.	%
Presentation						
Frank breech	1,289	74.9	6	4.7	15	1.2
Complete breech	398	23.1	2	5.0	7	1.8
Unknown	33	2.0	0	0.0	0	0.0
Total	1,720	100.0	8	4.7	22	1.3
Operation for delivery						
Spontaneous breech	93	5.4	0	0.0	0	0.0
Assisted breech	1,080	62.8	6	5.6	9	0.8
Breech extraction	511	29.7	2	3.9	10	2.0
Decomposition and extraction	36	2.1	0	0.0	3	8.3
FACH						
Used	534	31.0	3	5.6	18	3.4
Not used	1,186	69.0	5	4.2	4	0.3
Parity						
Primigravidas	853	49.6	4	4.7	9	1.1
Multiparas	867	50.4	4	4.6	13	1.5
Birth weight (grams)						
2,500 - 2,999	599	34.8	1	1.7	3	0.5
3,000 - 3,499	739	43.0	4	5.4	10	1.4
3,500 - 3,999	311	18.1	1	3.2	5	1.6
4,000 and over	71	4.1	2	28.2	4	5.6
Use of oxytocin						
Oxytocin	287	16.7	0	0.0	3	1.0
No oxytocin	1,433	83.3	8	5.6	19	1.3
Premature rupture of membranes						
Present	286	16.6	1	3.5	2	0.7
Not present	1,434	83.4	7	4.9	20	1.4
Duration of labor (hr.)						
1 - 6	678	39.4	1	1.5	8	1.2
7 - 12	706	41.0	4	5.7	8	1.1
13 - 18	249	14.5	1	4.0	3	1.2
19 and over	87	5.1	2	23.0	3	3.4
Maternal prepregnancy weight (pounds)						
to 119	580	33.7	3	5.2	7	1.2
120 - 139	710	41.3	3	4.2	8	1.1
140 - 159	288	16.7	2	6.9	3	1.1
160 - 179	96	5.6	0	0.0	1	1.0
180 and over	46	2.7	0	0.0	3	6.5
Attendant at delivery						
Service	659	38.3	5	7.6	13	2.0
Private	1,061	61.7	3	2.8	9	0.8

Estimation of pelvic capacity. The utilization of x-ray pelvimetry in this series is detailed in Table IV. The incidence of traumatic EPM was very significantly higher among patients who had x-ray pelvimetry as compared to patients who had none. Among patients who had x-ray pelvimetry, traumatic EPM was significantly higher in multiparas than in primigravidas. In patients without pelvimetry, traumatic EPM was higher in primigravidas, but the increase fell short of statistical significance ($0.05 < p < 0.10$). Whether pelvimetry was obtained bore no significant relationship to traumatic morbidity, and there was no significant difference in this regard between primigradivas and multiparas. These seemingly paradoxical results will be discussed later.

Complications of breech presentation alone

The perinatal complications resulting from breech presentation alone already have been defined; only EPM secondary to acute fetal

Table IV. X-ray pelvimetry in term breech vaginal delivery

X-ray pelvimetry	Cases		Deaths		Morbidity	
	No.	%	No.	/M	No.	%
Pelvimetry						
Primigravidas	388	–	2	5.2	7	1.8
Multiparas	232	–	4	17.2	5	2.2
Total	620	36.0	6	9.7	12	1.9
No pelvimetry						
Primigravidas	438	–	2	4.6	3	0.7
Multiparas	662	–	0	0.0	7	1.1
Total	1,100	64.0	2	1.8	10	0.9

asphyxia proved to be a significant problem. This included fetal death by intrauterine asphyxia during labor (prolapsed cord in 6, cause undetermined in 2) and during urgent delivery following clinical detection of fetal distress (cause undetermined at delivery in 2) and 3 fetal deaths which occurred during routine delivery with no prior evidence of fetal distress. The data pertaining to asphyxic complications are presented and analyzed with respect to possible etiologic factors in Table V.

Presentation. The incidence of asphyxic EPM was very significantly greater in complete breech presentation, 50 per cent occurring in cases categorized as "footling" breech. The specific problems of prolapse of the umbilical cord and of fetal distress of undetermined etiology are discussed in greater detail below.

Operation for delivery. The manner of obstetric delivery did not influence asphyxic EPM significantly. The absence of aspyhxic EPM in spontaneous breech delivery and in breech decomposition and extraction was not significant.

FACH. The use of forceps to deliver the aftercoming head bore no relationship to asphyxic EPM.

Parity. Asphyxic EPM was significantly greater in multiparas than in primigravidas.

Birth weight. Birth weights of 3,000 grams or more bore no relationship to asphyxic EPM. In the birth weight range of 2,500 to 2,999 grams, asphyxic EPM was increased very significantly.

Use of oxytocin. The preponderance of asphyxic EPM among patients who did not receive oxytocin was very significant.

Premature rupture of membranes. Ruptured membranes were related very directly to asphyxic complications of all types. Premature rupture of membranes, duration of latent period, and duration of ruptured membranes to delivery did not influence asphyxic EPM.

Duration of labor. There was no significant relationship between duration of labor and asphyxic EPM.

Attendant at delivery. There was no significant difference in asphyxic EPM between service and private cases.

Prolapse of the umbilical cord. Data pertaining to overt prolapse of the umbilical cord in singleton term breech presentation are presented in Table VI.

During the study interval, the incidence of cord prolapse in term vertex presentation was 0.5 per cent, and the associated number of perinatal deaths was 200/M. The incidence of overt prolapse of the umbilical cord in term breech presentation was increased eightfold: a threefold increase in frank breech and a twentyfold increase in complete breech presentation. The perinatal loss associated with this complication was significantly lower (reduced by 62.5 per cent) in breech than in vertex presentation and in complete than in frank breech. In 48 cases (57.8 per cent), prolapse of the umbilical cord occurred sufficiently late in the course of labor to permit immediate vaginal delivery of a surviving infant. In 29 cases (34.9 per cent), prompt diagnosis of umbilical cord

Table V. Asphyxic EPM in term breech presentation

Parameter	Cases		Deaths	
	No.	%	No.	/M
Presentation				
Frank breech	1,289	74.9	6	4.7
Complete breech	398	23.1	6	15.1
Unknown	33	2.0	1	30.3
Total	1,720	100.0	13	7.6
Operation for delivery				
Spontaneous breech	93	5.4	0	0.0
Assisted breech	1,080	62.8	9	8.3
Breech extraction	511	29.7	4	7.8
Decomposition and extraction	36	2.1	0	0.0
FACH				
Used	534	31.0	5	9.4
Not used	1,186	69.0	8	6.8
Parity				
Primigravidas	853	49.6	3	3.5
Multiparas	867	50.4	10	11.5
Birth weight (grams)				
2,500-2,999	599	34.8	7	11.7
3,000-3,499	739	43.0	4	5.4
3,500-3,999	311	18.1	1	3.2
4,000 and over	71	4.1	0	0.0
Use of oxytocin				
Oxytocin	287	16.7	1	3.5
No oxytocin	1,433	83.3	12	8.4
Premature rupture of membranes				
Present	286	16.6	3	10.5
Not present	1,434	83.4	10	7.0
Duration of labor (hr.)				
1- 6	678	39.4	6	8.9
7-12	706	41.0	6	8.5
13-18	249	14.5	1	4.0
19 and over	87	5.1	0	0.0
Attendant at delivery				
Service	659	38.3	6	9.1
Private	1,016	61.7	7	6.9

prolapse permitted delivery by cesarean section without fetal death. In 6 cases (7.3 per cent), overt prolapse of the umbilical cord and the consequent clinical evidence of fetal distress went undetected until intrauterine fetal death had occurred; vaginal delivery then was permitted to ensue. In each case, a significant interval (> 30 minutes) had elapsed between the last recorded auscultation of the fetal heart rate and the detection of fetal death. In 2 instances, membranes ruptured spontaneously and the umbilical cord prolapsed (to be found later lying outside of the introitus) in sedated, unattended multiparas otherwise in normal labor; in the other 4 instances, "internal" cord prolapse occurred in patients in whom membranes had ruptured previously and in whom neither vaginal examination nor fetal heart rate ausculation was performed for 40 to 60 minutes, although attendants were present constantly.

Fetal distress. Data concerning acute intrauterine fetal asphyxia of undetermined cause in singleton term infants with breech presentation are presented in Table VI. In this series, fetal distress was defined by changes in fetal heart rate: tachycardia above 160 per minute, bradycardia below 120 per minute, or marked cardiac rate irregularity. The presence of meconium in the amniotic fluid, alone, was not considered an indication of fetal distress in breech presentation in labor. Meconium, in association with a

Table VI. Fetal asphyxia in term breech presentation

Mode of delivery	Deliveries		Overt cord prolapse				Fetal distress				Total			
			Cases		Deaths		Cases		Deaths		Cases		Deaths	
	No.	%	No.	%	No.	/M	No.	%	No.	/M	No.	%	No.	/M
Vaginal														
Frank	1,289	74.9	17	1.5	3	176.5	77	6.0	3	39.0	94	7.4	6	63.8
Complete	431	25.1	37	8.6	3	81.1	25	5.8	4	160.0	62	14.4	7	143.9
Total	1,720	100.0	54	3.1	6	111.1	102	5.9	7	68.6	156	9.1	13	83.3
Cesarean section														
Frank	343	80.7	10	2.9	0	0.0	29	8.5	0	0.0	39	11.4	0	0.0
Complete	82	19.3	19	23.2	0	0.0	6	7.3	0	0.0	25	30.5	0	0.0
Total	425	100.0	29	6.8	0	0.0	35	8.2	0	0.0	64	15.1	0	0.0
All breeches														
Frank	1,632	76.1	27	1.7	3	111.1	106	6.5	3	28.3	133	8.2	6	45.1
Complete	513	23.9	56	10.9	3	53.6	31	6.0	4	129.0	87	17.0	7	80.5
Total	1,730	100.0	83	3.9	6	72.3	137	6.4	7	51.1	220	10.3	13	59.1

regular fetal heart rate, was present during labor in 11.7 per cent of all breech presentations, occurring with the same incidence in both frank and complete breech. There was no fetal death among these cases and no neonatal evidence of fetal hypoxia or acidosis. Extrusion of meconium during labor in breech presentation, without other concomitant evidence of fetal distress, results from compression of the fetal abdomen as the breech enters the pelvis. The presence of meconium in the amniotic fluid prior to the onset of labor would have an entirely different significance; there were no such cases in this series.

Without continuous fetal heart rate monitoring, the true incidence of fetal distress cannot be known. Continuous monitoring was not employed routinely at this institution during the study interval. Subject to the error inherent in intermittent auscultation of the fetal heart rate, the recorded incidence of fetal distress of undetermined cause in term vertex presentation in labor was 0.8 per cent, and the associated perinatal deaths were 150/M. The incidence of fetal distress of undetermined cause in term breech presentation (6.4 per cent) was 8 times greater than in term vertex presentation and did not vary significantly between frank and complete breech. The associated perinatal mortality rate was one third that in term vertex presentation and was 4 times higher in complete than in frank breech presentation. In 95 cases (69.3 per cent), fetal distress was detected sufficiently late in the course of labor to permit immediate vaginal delivery of a surviving infant. In 35 cases (25.5 per cent), emergency cesarean section was performed without fetal death. In 4 cases (3.0 per cent), the detection of very severe fetal distress was followed so promptly by fetal death that palliative measures were useless, and neither abdominal nor vaginal delivery could be effected in time to salvage the infants; in each case, the detection of severe fetal distress followed unduly long intervals (> 30 minutes) during which fetal heart rate monitoring was not recorded. In 3 cases (2.2 per cent), fetal death occurred during the actual delivery. The labors had been normal and progressive, and the fetal heart rates were regular when the patients were placed on the delivery tables. In each instance (2 frank breech, one complete breech presentation), the deliveries took between 15 and 20 minutes (all assisted breech deliveries, one with FACH), although there were no dystocic problems; the fetal heart rates had not been monitored during the delivery process. The infants were stillborn and could not be resuscitated.

Breech delivery by cesarean section

Experience with delivery of term infants with breech presentation by cesarean section was very satisfactory with regard to pregnancy outcome. There was no excess peri-

Table VII. Term breech delivery by cesarean section

	No.	*%*
Cesarean section rate		
1953-1960	155	17.0
1961-1970	270	22.1
Total	425	19.8
Presentation		
Frank breech	343	80.7
Complete breech	67	15.8
Unknown	15	3.5
Parity		
Primigravidas	237	55.8
Multiparas	188	44.2
Birth weight (grams)		
2,500-2,999	113	26.6
3,000-3,499	167	39.3
3,500-3,999	104	24.5
4,000 and over	41	9.6

Table VIII. Indications for cesarean section in term breech presentation

	No.	*%*
Cephalopelvic disproportion	204	48.0
Previous cesarean section	100	23.5
Prolapsed cord	29	6.8
Fetal distress	14	3.3
Breech	12	2.8
Placenta previa	12	2.8
Uterine dysfunction	12	2.8
Previous myomectomy	12	2.8
Diabetes mellitus	7	1.6
Elderly primigravida	5	1.2
Abruptio placentae	5	1.2
Tumor previa	5	1.2
Other indications (each less than 1%)	8	2.0
Total	425	100.0

natal mortality rate related to breech presentation or delivery among infants delivered abdominally. Two fractures were attributed to difficult extractions of the infants from the uterus, a traumatic morbidity of 0.5 per cent. During the study interval, the rate of delivery of term infants with breech presentation by cesarean section increased steadily and throughout was approximately twice the cesarean section rate for term infants with vertex presentation (Table VII).

The primary indications for delivery of term infants with breech presentation by cesarean section are listed in Table VIII. The ratio of primary to repeat cesarean sections in term breech presentation was 3:1, as compared to a ratio of 2:1 in term vertex presentation. The incidences of indications related to fetopelvic disproportion and of elective cesarean section without labor were almost double those in vertex presentation. This represents not a concentration of patients with small pelves but rather an increasingly liberal interpretation of "fetopelvic disproportion."

Comment

The statistical data in this review are in general agreement with data reported from other large series of breech presentations. Although absolute comparisons are difficult because patient populations, methods of recording, and even obstetric definitions differ between series, some general comparisons are expressed in Table IX. It is evident that breech presentation at term presents similar problems everywhere.[1-5]

The main purpose of the present analysis was to define, if possible, the etiologic or associated factors in excess perinatal deaths and morbidity for term singleton breech delivery reported in every series. A summary of the relationships found to be significant statistically in term breech vaginal delivery at this institution is presented in Table X.

Obstetric trauma. The incidences of traumatic EPM and traumatic morbidity in term breech vaginal delivery were related to 3 factors: maternal pelvic capacity, size of the fetus, and manipulation employed to effect delivery. Some factors, such as type of breech presentation, maternal parity, use of oxytocin, and premature rupture of the membranes (as well as duration of latent period and total duration of ruptured membranes) were not related at all to traumatic complications of breech delivery. Other less peripheral factors expressed their influence in terms of the 3 basic factors: Increased traumatic morbidity among babies delivered with FACH was related to the preponderance in this group of infants weighing in excess of 3,500 grams, and the inclusion of all infants weighing 4,000 grams or more; the traumas

Table IX. Comparison of selected series of breech deliveries*

Parameters	*Series A*	*Series B*	*Series C*	*Series D*	*Series E*
Incidence of breech presentation (%)	4.1	4.0	3.2	4.3	–
Over-all perinatal deaths (/M)	159	159	123	–	–
Incidence of singleton breech presentation at term (%)	2.7	2.7	2.4	–	–
Perinatal deaths at term (/M)	31.7	30.1	40.0	44.1	–
Perinatal loss at term, corrected†	17.9	–	22.0	19.1	16.0

*Series A: The Mount Sinai Hospital, New York (1953 to 1970), breech presentation in singleton pregnancy. Series B: Morgan and Kane[1] (1964), breech presentation, including multiple gestations. Series C: Hall and associates[2] (1965), breech presentation in singleton pregnancy, corrected for birth weights below 1,000 grams. Series D: Fischer Rasmussen and Trolle[3] (1967), breech presentation in singleton pregnancy at term. Series E: Johnson[4] (1970), breech presentation in singleton pregnancy at term.

†Corrected for antepartum deaths, congenital malformations, and maternal complications.

Table X. Factors related to excess perinatal deaths and morbidity*

Factor	*Traumatic deaths*	*Traumatic morbidity*	*Asphyxic deaths*
Presentation	0	0	↑ (Complete)
Operation for delivery			
Spontaneous breech	↓	↓	0
Assisted breech	0	↑	0
Breech extraction	0	↑	0
Decomposition and extraction	Ins.	↑↑	0
FACH	0	↑	0
Maternal parity	0	0	↑ (Multiparas)
Birth weight (grams)			
2,500-2,999	↓	↓	↑
4,000 and over	↑	↑	0
Use of oxytocin	0	0	↓
Premature rupture of membranes	0	0	0
Duration of labor (19 hr. and over)	↑	↑	0
Maternal prepregnancy weight (180 pounds and over)	0	↑	0
X-ray pelvimetry	↑	0	0
Experience of attendant	↓	↓	0

*Only statistically significant relationships are depicted. Ins. = insufficient cases for significance.

incurred were not forceps injuries. Increased traumatic EPM and traumatic morbidity among infants born after labors lasting longer than 18 hours also were related to a preponderance in this category of infants weighing in excess of 3,500 grams. Increased traumatic morbidity among babies born to mothers whose prepregnancy weight was 180 pounds or more apparently was related to a decrease in available pelvic capacity. While it has been demonstrated that maternal prepregnancy weight is related directly to the birth weight of the infant,[6, 7] the traumatized infants in this group all were in the birth weight range of 2,500 to 3,499 grams; this suggests that pelvic capacity does not depend only on the maternal bony pelvis but may be reduced by soft (adipose) tissue encroachment. Finally, the decrease in traumatic EPM and traumatic morbidity in infants delivered by the more experienced physicians possibly reflects better evaluation of pelvic capacity, more accurate estimation of fetal weight, and better judgment in borderline situations and certainly is a measure of greater skill in obstetric manipulation.

The 3 basic factors which influence traumatic EPM and traumatic morbidity directly in term breech vaginal delivery can stand alone for purposes of discussion but often are interrelated clinically.

Fetal birth weight. The influence of fetal birth weight on pregnancy outcome in term breech vaginal delivery long has been recog-

nized[8, 9] and was manifest in several ways. All other factors being equal, there were significantly less traumatic EPM and traumatic morbidity in the small fetus (< 3,000 grams). The small breech infant tended to be delivered with less manipulation and did not require the use of FACH. Conversely, all other factors being equal, the large breech infant (> 4,000 grams) was associated with significantly increased traumatic EPM and traumatic morbidity. The large breech infant often endured prolonged labor and required more obstetric manipulation and the use of FACH to effect delivery.

Operation for delivery. The deleterious effects of obstetric manipulation on the fetus were obvious and were manifest more readily in traumatic morbidity than in traumatic EPM. Spontaneous breech delivery was associated with no traumatic complications. Increasing degrees of obstetric manipulation corresponded to progressive elevations of the traumatic morbidity rate. The highest rate (8.3 per cent) occurred with breech decomposition and extraction—10 times the rate for assisted breech delivery and 4 times the rate for breech extraction. The relationship between obstetric manipulation and fetal birth weight already has been discussed.

Maternal pelvic capacity. The influence of maternal pelvic capacity on traumatic EPM and traumatic death in term breech vaginal delivery has been documented repeatedly.[10] The results in this series seem paradoxical in that greater traumatic EPM was encountered in patients who had x-ray pelvimetry than in patients who did not. Obviously, taking x-ray pelvimetry does not cause traumatic EPM. Also, patients with clinically large pelves more frequently may be permitted to avoid x-ray exposure. Review of traumatic EPM in patients with x-ray pelvimetry showed that all of these fetal deaths were avoidable and occurred because of 3 errors in clinical judgment: application to the breech of concepts of adequacy of pelvic capacity derived from vaginal delivery of vertex presentation (i.e., "compensatory room" in the platypelloid pelvis, pelvic indices, and optimal accommodation of the largest diameter of the fetal vertex to the largest diameter of the maternal pelvis); underestimation of fetal weight; and reliance on clinical evidence of pelvic capacity (i.e., birth weight of largest infant previously delivered vaginally, albeit as a vertex, and "trial of labor"). The conclusions to be drawn, therefore, are the converse of initial impressions derived from the data. Parity, with prior clinical testing of pelvic capacity, did not influence the incidence of traumatic complications. Evaluation of pelvic capacity by clinical trial of labor has some advocates[11, 12] in the management of term breech delivery but often has been found to be unreliable.[10, 13] In this series, increased duration of labor (mainly, slow progress in cervical dilatation) did not influence traumatic EPM or traumatic morbidity until more than 18 hours had elapsed and even then was related to the large size of the fetus. Neither was descent of the breech a critical parameter; in 6 of the 8 cases of traumatic EPM, the duration of the second stage was 30 minutes or less, and spontaneous delivery of the breech infant to the umbilicus occurred in 5. It seems clear that for optimal results objective mensuration of pelvic dimensions and accurate knowledge of the pelvic architecture are essential in each case of term breech vaginal delivery. At present, this can be accomplished only with x-ray pelvimetry.

Asphyxic complications. Excess perinatal deaths secondary to acute fetal asphyxia are a problem of equal magnitude to obstetric trauma, accounting for 54 per cent of the excess pregnancy wastage in singleton term breech presentation. In some reports,[2, 4, 5] much has been made of the increased incidence of prolapse of the umbilical cord in breech as compared to vertex presentation, of the more frequent occurrence of prolapsed cord in the complete rather than frank breech, and of the higher perinatal mortality rate associated with this complication in the frank breech. The data in this series corroborate these statements (Table VI). However, 54 per cent of cases of asphyxic EPM in this series were associated with acute intrapartum fetal asphyxia without overt cord

prolapse and in which the etiologic diagnosis remained undetermined. To evaluate the problem of fetal asphyxia more accurately, the total picture (i.e., overt cord prolapse plus fetal distress for undetermined etiology) must be considered (Table VI).

When the 2 components of fetal asphyxia were combined, different relationships became apparent. Some degree of intrauterine asphyxia occurred in 10.3 per cent of all term breech presentations, and this complication was twice as frequent in the complete breech (17.0 per cent) as in the frank breech (8.2 per cent). The associated perinatal mortality rate was 59.1/M, the rate in the complete breech (80.5/M) being almost twice that in the frank breech (45.1/M). The data suggest that any complacency about the less lethal consequences of this complication in the complete breech as compared to the frank breech is unjustified.

Obstetric factors which proved to be unrelated to asphyxic EPM included type of operation for delivery, use of FACH, premature rupture of membranes (as well as duration of latent period and total duration of ruptured membranes), duration of labor, and experience of the attendant physician. The significant increase in asphyxic EPM in small infants with birth weights between 2,500 and 2,999 grams may result from the fact that the small breech infant leaves more space in the maternal birth canal for either overt or covert prolapse of the umbilical cord to occur.

The 2 most significant factors again at first glance seem to be paradoxical. Three fourths of the cases of asphyxic EPM occurred in multiparas, and 92 per cent occurred in patients not receiving oxytocin. At the same time, the incidence of asphyxic complications per se was distributed evenly by parity and by use of oxytocin. The determinant in this disparity was vigilant observation of the patient in labor and during delivery. The primigravida with a term breech presentation tended to be observed and monitored in labor more carefully and regularly; for the patient receiving oxytocin intrapartum, constant monitoring was required. Close observation ensured early detection of cord prolapse and/or fetal distress and permitted the infants to be salvaged. Similar close intrapartum attention certainly would have reduced and possibly eliminated entirely all fetal deaths caused by acute intrapartum asphyxia.

Mode of delivery. Significant problems are associated with labor and vaginal delivery in term breech presentation. This retrospective review could consider only the immediate perinatal complications. A number of reports[14-18] have suggested that there also may be very significant deleterious long-term major and minor neurological abnormalities among infants born of breech vaginal delivery, including increased incidences of cerebral palsy, epilepsy, mental retardation, athetoid and spastic hemiplegias, and functional brain damage, which may not be recognized in the immediate neonatal period or be included in the usual obstetric statistics. However, these reports are of little help in assessing the safety of breech vaginal delivery. Frequently, infants of all birth weights have been considered together; more searching analysis has shown that the deleterious etiologic influence of prematurity overwhelms that of breech delivery and that the difference in neurological complications between breech and vertex vaginal deliveries in term-sized infants is very small.[19] Moreover, the role played by intrapartum asphyxic episodes in many of the affected infants is difficult to evaluate.[20]

More recently, reports had suggested that normal labor and vaginal delivery may impose a burden of intrauterine hypoxia on the fetus in breech presentation.[21,22] Studies of umbilical cord blood lactate/pyruvate ratios at birth, a measure of anaerobic metabolism in the fetus secondary to hypoxia (when corrected for the maternal biochemical state),[23,24] seemed to show that uncomplicated breech vaginal delivery carried an increased risk of fetal hypoxia as compared to vertex vaginal delivery and that oxytocin stimulation of breech labor and delivery apparently eliminated this excess risk, presumably by reducing the time interval during which the fetus

was at risk. The immediate problem was whether some fetuses in breech presentation, without manifesting the usual clinical indications of fetal distress, become more vulnerable to the documented vicissitudes of labor and vaginal delivery or suffer significant central nervous system impairment resulting in late neurological sequelae because of subclinical hypoxia. Fortunately, the same investigators[25] have demonstrated that infants monitored during labor and delivery whose fetal heart rates were normal throughout always showed a normal biochemical status at birth, although the clinical state of the infant as measured by the Apgar score showed some variability. While this report must be confirmed in larger series of patients and specifically in breech presentation, it emphasizes the value of fetal heart monitoring and the significance of clinical signs of fetal distress and tends to refute the specter of undetectable, subclinical fetal hypoxia.

More obvious poor obstetric results (perinatal death and morbidity) in breech presentation always have been associated overwhelmingly with vaginal delivery. In this series, there were no excess perinatal deaths associated with delivery by cesarean section. The incidence of traumatic morbidity at cesarean section was 0.5 per cent, 5 times higher than in term vertex delivery but less than half that associated with term breech vaginal delivery. Similar observations have been made by others[1] and have evoked the recommendation that all patients reaching 35+ weeks of gestation with a fetus in breech presentation should be delivered routinely by elective cesarean section for fetal indications.[26] While the benefits for the term fetus in breech presentation of delivery by elective cesarean section are incontrovertible, they must be weighed against the maternal consequences, which include not only immediate death and morbidity but also risks during a subsequent obstetric career such as elective repeat cesarean section, vaginal delivery after previous cesarean section, or antepartum/intrapartum uterine scar dehiscence, as well as possible late sequelae of abdominal operations such as intestinal obstruction or incisional hernia. The maternal risks in otherwise uncomplicated elective cesarean section in the modern surgical and anesthetic eras remain to be evaluated. Unless and until elective cesarean section is shown to be as safe for the mother as vaginal delivery, routine elective cesarean section remains an unsatisfactory solution to the problems posed by term breech vaginal delivery.

In an effort to improve perinatal salvage in breech presentation without resorting to cesarean section, some have utilized routine antepartum external cephalic version.[27-29] In one series,[27] the incidence of breech presentation at term was reduced by this approach from 3.15 to 2.05 per cent. The fetal loss associated with external cephalic version was 3.8/M; with vertex delivery, 10.5/M; with breech delivery, 31.5/M; and with vertex delivery after successful external cephalic version, 14.3/M. External cephalic version may be difficult to accomplish and contributes its own inherent risks for both mother and fetus; version under general anesthesia is particularly hazardous.[29] In terms of rate of successful version, number of cesarean sections avoided (keeping in mind that the leading cause of emergency cesarean section after successful version was fetal distress, probably secondary to cord entanglement),[28] and the number of infants salvaged who otherwise would have died (from the aforementioned perinatal death rates, approximately 2 per 1,000 cases of breech presentation), routine external cephalic version also seems to be an unsatisfactory solution to the problems posed by breech vaginal delivery.

It is the consensus[2-5] that indications for cesarean section should be liberalized in breech presentation but that the majority of breech infants still can be delivered safely vaginally. The choice between vaginal and abdominal delivery thus becomes a crucial determinant of fetal salvage. Unfortunately, the suggested criteria upon which this decision should be based usually have been distressingly vague and subjective, as, for example, ". . . vaginal delivery should be planned when there is absolutely no argument against it. . . ."[3] One attempt to quantify these

Table XI. Mean normal dimensions of the adult female pelvis

Pelvic dimension	*Berman[32] (1956)*	*Thoms and Greulich[33] (1940)*	*Young and Ince[34] (1940)*
Anteroposterior diameter of inlet (obstetric true conjugate)	12.0 cm.	11.9 cm.	11.8 cm.
Transverse diameter of inlet	13.5 cm.	12.8 cm.	13.1 cm.
Interspinous diameter	10.5 cm.	10.4 cm.	9.9 cm.
Intertuberous diameter	10.5 cm.	–	10.1 cm.
Subpubic angle	85.0°	–	93.5°

criteria[10] has proposed a prognostic scoring index to be employed when the patient with a breech presentation is admitted in labor. While application of this index has been deemed useful,[30, 31] several problems remain unresolved: First, permitting the patient to go into labor exposes the fetus to the risk of asphyxic complications which elective cesarean section might avoid. Second, from the data in this report, some of the criteria used to construct the index have little or no direct relationship to pregnancy outcome, i.e., parity, gestational age, cervical dilatation, and station of the presenting part. Objective criteria which would permit decisions about the feasibility of vaginal delivery in term breech presentation to be made before the onset of labor would obviate these problems.

Recommendations

Based on the data and analyses in this report, a number of recommendations for objective criteria can be made, which would permit the obstetrician to anticipate or avoid unnecessary risks to the fetus from breech vaginal delivery.

X-ray pelvimetry. Accurate and objective information about maternal pelvic dimensions and configuration must be available. X-ray pelvimetry should be performed prior to the onset of labor, to permit elective cesarean section if indicated, in each patient with a breech presentation at term regardless of maternal parity, size of infants previously delivered vaginally, or estimated fetal weight in the incumbent pregnancy. If not included routinely with x-ray pelvimetry, a single x-ray examination of the entire maternal abdomen will define precisely the fetal presentation and the attitude of the fetal vertex and may alert the physician to the possible presence of congenital anomalies.

Maternal pelvic capacity. Gauging pelvic capacity by criteria derived from experience with vaginal delivery and vertex presentation is of little value in the breech. Critical "lower limits of normal" and calculated pelvic indices must be ignored. For vaginal delivery in breech presentation to be feasible without undue trauma to mother or infant, all pelvic measurements must be at least mean normal values. The principal emphasis in most recent obstetric texts is on defining borderline pelvic capacity; to learn mean normal pelvic dimensions, the older literature must be consulted[32-34] (Table XI).

Given at least average pelvic dimensions, pelvic configuration becomes of secondary importance. In this series, a contracted anteroposterior diameter of the pelvic inlet, as in a platypelloid pelvis, contributed most to dystocia in breech delivery. The dimensions of the pelvic inlet were of paramount importance; no case of significant mid-pelvic or outlet contraction was encountered when dimensions of the pelvic inlet were adequate. There remains some room for the exercise of clinical judgment. A very small breech may be delivered through a slightly smaller pelvis. A very obese patient may require a larger bony pelvis to compensate for soft tissue encroachment. With few exceptions, however, a maternal bony pelvis which fails to meet the criteria outlined above should be considered an indication for delivery of a term breech infant by elective cesarean section.

Fetal weight. Estimation of fetal weight by palpation of the maternal abdomen notoriously is inaccurate, particularly in

other than vertex presentation and with the very small or very large fetus. In term breech presentation, the biparietal diameter of the fetal vertex should be determined routinely by ultrasonography, if available, not for comparison with maternal pelvic dimensions (a fallacy carried over from vertex presentation) but to obtain within the limits of error inherent in the technique and calculations the most accurate antenatal estimate of fetal weight possible at this time.[35-38] A fetus in breech presentation whose estimated fetal weight by ultrasonography is 4,000 grams or more should be delivered by elective cesarean section no matter what the maternal pelvic dimensions may be. If ultrasonography is not available and reliance must be put on estimation of fetal weight by palpation only, the margin of safety must be increased because of greater potential for error, and any fetus estimated to weigh more than 3,600 grams (approximately 8 pounds) should be delivered by elective cesarean section.

Operation for delivery. Given an adequate maternal pelvis and a fetus of suitable estimated weight and other obstetric factors being equal, breech delivery should be accomplished with the least manipulation possible. Spontaneous breech delivery is highly preferable. There is no contraindication to the use of forceps to deliver the aftercoming head. Assisted breech delivery and breech extraction both involve a change in motive power for delivery, substituting traction from below for pressure from above and increasing the probability of occurrence of unilateral or bilateral nuchal arms or extension of the fetal vertex; expert assistance with abdominal fundal pressure during traction may minimize these difficulties. Decomposition and extraction of a frank breech infant carries a significantly increased fetal risk and imposes additional maternal hazards because of intrauterine manipulation (uterine rupture) and the need for deep general anesthesia (anesthetic complications, uterine atony, postpartum hemorrhage). Further, the opportunity to develop adequate technical facility with this mode of delivery now is very limited; in this series, extending over 2 decades, the incidence of breech decomposition and extraction was only 2.1 per cent. In view of these problems, this operation probably should be abandoned for singleton term breech infant delivery.

Obstetric experience. Breech delivery should be conducted or at least personally supervised by the most experienced accoucheur available. Often, this experience may be more valuable when providing assistance with abdominal pressure than in actual delivery of the breech itself.

Fetal monitoring. The fetus in breech presentation is at high risk for asphyxic complications, regardless of type of presentation. This is especially true for the small breech infant ($< 3{,}000$ grams). Accordingly, the fetus must be monitored constantly for clinical evidence of fetal distress and/or prolapse of the umbilical cord. This is best accomplished in an obstetric "intensive care" labor room with continuous automatic electronic or ultrasonic monitoring of the fetal heart rate. If this is not available, the fetal heart rate must be monitored by auscultation during and after each uterine contraction. Vaginal examination must be performed immediately upon rupture of the fetal membranes to detect possible overt cord prolapse. In view of the very significant association between ruptured fetal membranes and asphyxic complications in breech presentation, amniotomy should be deferred. With evidence of significant fetal heart rate alteration, immediate upward displacement of the presenting breech by manual pressure from the vagina almost always will relieve umbilical cord compression temporarily, restore the fetal heart rate to normal, and provide a short interval in which to effect immediate vaginal or abdominal delivery, as dictated by the obstetric situation.

Fetal heart rate monitoring must be continued throughout the delivery itself. Although other factors, such as vagal stimulation and possibly delayed venous return to the fetal heart secondary to chest compression, are operative at this point, the potential for umbilical cord obstruction is high. Fetal bradycardia during delivery correlates

positively with poor clinical condition and depressed Apgar score of the newborn infant, and prolongation of abnormal cardiac rates into the neonatal period in such cases is related directly to protracted delivery times.[39] Fetal bradycardia on the delivery table therefore is an indication for most expeditious delivery—vaginally if obstetrically feasible or abdominally even at this late stage if not.

These recommendations are derived from a retrospective analysis of 2,145 singleton term breech presentations and deliveries. Had they been followed during the accumulation of this series of cases, excess perinatal deaths would have been eliminated entirely and traumatic morbidity in breech vaginal delivery would have been reduced to a level (0.52 per cent) equivalent to that accompanying term breech delivery by cesarean section (0.50 per cent). It was not possible in this retrospective study to determine what the incidence of term breech delivery by cesarean section would have been under the altered circumstances. Whether the recommendations are weighed too heavily in favor of the fetus at the expense of the mother will have to be determined by prospective studies of resultant cesarean section rates which, with maternal risk figures for uncomplicated abdominal delivery, form the maternal side of the perennial obstetric equation.

Conclusions

The perinatal mortality rate, corrected for congenital anomalies and maternal disease states, is approximately 3 times higher in singleton term breech presentation than in term vertex presentation, and traumatic morbidity is 12 times higher.

Despite the excellent fetal outcome when a term infant with breech presentation is delivered abdominally, routine elective cesarean section for this indication alone to date is not acceptable in terms of increased maternal risk. Routine external cephalic version also has not provided a satisfactory alternative method of management.

Based on an analysis of 2,145 cases of singleton breech presentation at term, a series of recommendations are made concerning maternal pelvic capacity, fetal weight, fetal monitoring during labor and delivery, and experience of the accoucheur which are designed to reduce perinatal deaths in term breech vaginal delivery to the level encountered in vertex vaginal delivery and traumatic morbidity to that accompanying term breech delivery by cesarean section.

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Pathology of Pregnancy and Labor in Adolescent Patients

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SUMMARY

A study done on 1,033 pregnant teen-agers whose babies were delivered at Chicago Lying-in Hospital between January 1, 1965 and June 30, 1968 indicated that the incidence of pregnancy in girls 17 years and younger is increasing. The most common complications during pregnancy in the patients studied were anemia, premature labor, vaginal infection, and pre-eclampsia. Uterine hypertony as a form of dystocia was a relatively common problem during labor, especially in patients who had had insufficient or no prenatal care. The most common postpartum complications were urinary tract infection and puerperal endometritis. The psychological trauma of such early pregnancy may interfere with the maturing personality.

Additional key words: high risk pregnancy; postnatal complications; prematurity.

Although there is no common doctrine concerning the best age for the delivery of the first baby, from physiologic, obstetric, and psychologic points of view, the ideal age is between eighteen and thirty years.

The adolescent period is difficult to define. It usually begins at approximately 12 years of age and ends when the transition of early adulthood occurs, that is, after 17 years [6, 20].

Adolescence is a transitional phase in the developmental process. The goal of this period is to prepare an individual to take his place as an adult in his own family and in the larger community. Adolescence is also a period when the young person is exceedingly malleable and vulnerable to psychic stress. Most of the psychiatric disorders of adolescents reflect an imbalance between various internalized familial and social forces [17, 27].

Although pregnancy is a physiological state it can represent a psychological trauma to a person who is in a developmental phase [27].

More than 30 papers have been published in the world literature in the last four decades concerning pregnancy and labor in the adolescent period, but classical obstetrical textbooks [8] barely mention the topic.

Harris [14] in 1922 was the first to publish a paper about this problem. With perhaps a bit too much optimism, he concluded that 16 years of age represents "optimum time for the first labor". This conclusion could perhaps be accepted with regard to the physical and biological development of many 16-year-old girls but not with regard to their psychic maturity. Emotional and intellectual development is, as a rule, slower than physical and biological. A one or two year difference in age during the adolescent phase means a great deal with regard to physical development of a person and even more with regard to the degree of her psychic maturity.

Pakter and his associates [21] have performed psychological testing on more than 250 pregnant girls between the ages of 12 and 17. They found that approximately one-third were mentally retarded (I.Q. less than 75); another one-third were subnormal (I.Q. 75 to 90); and the last one-third were classified as normal (I.Q. 90 or more). We were not able to test systematically the psychological development of our patients. However, while studying their other problems, we have noted that their average intellectual development was significantly below that of the average adult.

Seldom are teen-age pregnancies planned. The pregnancy, labor, and care of the newborn are often a burden and stress for very young mothers as well as their parents. Very few of these patients are married and in many cases the marriages are forced and unstable.

Teen-age parents represent a high risk group in any community not only from health point of view but from psychological, educational, social and others, too [29].

Several authors [15, 16, 22, 23] believe that weight gain, toxemia, behavioral changes, prematurity, and perinatal mortality are more common in teen-age pregnancies than in pregnancies of older girls. Hassan and Falls [13] noted in addition an increased percentage of cervical lacerations, prolonged labors, and abdominal sections in patients 12 to 14 years of age.

The following analysis uses age 17 as the arbitrary end point of the adolescent period and includes data from patients 17 years or younger. Patients older than 17 years will be used as a control group in the study.

It is important to note in the following data that incidence of complications and other figures are not always comparable from author to author since there is no uniformity of agreement concerning the age at which adolescence ends.

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Materials, Methods, and Results

All patients studied delivered at the Chicago Lying-in Hospital. The total deliveries during this three and one-half year study period (January 1, 1965 to June 30, 1968) was 9,536. The adolescent group numbered 1,033 (10.8 per cent) and were between 11 and 17 years of age.

The percentage of teen-age pregnancies is steadily increasing in the United States. According to Zackler and associates [30], in 1945 there were 3,573 births to mothers under 14 or 13.1 births per 10,000 live births. In 1955 there were 5,883 births or 14.5 per 10,000 live births. By 1963 this figure had risen to 7,594 or 18.5 births per 10,000 live births. Table I shows a similarly increasing percentage in Chicago Lying-in Hospital.

Age

The most common age at delivery was 16 years (41.5 per cent). Our youngest patient was 11 years old (see Table II). She delivered a live male infant in cephalic presentation who weighed 3465 gm. Her labor lasted 22 hours and was ended by application of outlet forceps.

Parity

The number of previous pregnancies in our cases was as follows: para 1, 848 or 82 per cent of patients; para 2, 159 or 15.5 per cent; para 3, 23 or 2.2 per cent; and para 4, three patients or 0.3 per cent.

Race and socio-economic class

The great majority of patients (1,022 or 98.94 per cent) were Negro. Only eleven (1.06 per cent) were white. Nearly all belonged to lower socio-economic classes and many were referred from the Chicago Board of Health Maternity and Infant Care Project as high risk pregnancies. The data from several other authors [2, 5, 15, 16, 18, 22, 28] are different (Table III).

Prenatal Care

Hassan and Falls [15] point out the important inverse relationship between quality of prenatal care and the incidence of complication before, during, and after delivery including toxemia, excessive weight gain, prematurity, and perinatal mortality. We are of the same opinion and believe that adequate prenatal care for teen-age gravidas is the only way to guarantee a decrease in the incidence of the above complications.

The young pregnant patients at the Chicago Lying-in Hospital are routinely advised regular check-ups at least every three weeks or more until the 28th week of pregnancy. After 28 weeks the patients are seen more often. The majority of our obstetrical patients 15 years or younger were sent to our special High Risk Clinic and treated as problem pregnancies. Special group therapy by the Social Service Unit was held for our unmarried teen-agers at regular intervals. Adequate prenatal care was given to 1,002 patients (97 per cent). Thirty-one patients were unregistered and had little or no care prior to admission for delivery. Ten of these 31 were patients of private physicians and 21 were examined for the first time during labor.

Prenatal complications

The most frequent problem found in our patients was vaginal infection (445 cases or 40.9 per cent). However, only those girls with symptomatic vaginitis were treated. Flagyl® and Mycostatin® were most frequently used drugs (depending on the specific infection) with good results. In the control group the incidence of vaginal infection was 27 per cent.

Anemia was the second most frequent problem and occurred in 145 patients (14 per cent). All patients with hemoglobin lower than 10 gm per 100 ml and hematocrit lower than 32 per cent were classified as anemic. Iron deficiency anemia was diagnosed in 128 patients. All cases improved following treatment with iron-containing drugs. In the control group, 9.3 per cent of the patients had anemia.

TABLE I

The Incidence of Teen-age Pregnancies at Chicago Lying-in Hospital by Years (January 1, 1965 to June 30, 1968)

Year	Number of Cases	Total Number of Deliveries	Percentages
1965	250	2622	9.5
1966	300	2753	10.9
1967	346	2814	12.3
1/1 - 6/30/68	137	1347	10.1

TABLE II

Ages of Pregnant Teen-age Patients at Chicago Lying-in Hospital, Between January 1965 and June 1968

Years	11	12	13	14	15	16	17
No. of Cases	1	1	21	91	233	431	254
Percentages	0.09	0.09	2.03	8.8	22.5	41.7	24.5

TABLE III

RACE OF PATIENTS IN THIS STUDY AND THOSE REPORTED BY VARIOUS AUTHORS

Authors	Per Cent White	Per Cent Negro
Hassan and Falls	72.3	27.7
Morrison	5.2	94.8
Poliakoff	27.8	72.2
Aznar and Bennett	10	90
Clough	44.6	55.4
Van der Ahe and Bach	44	56
Marchetti and Menaker	1	99
Chicago Lying-in Hospital	1.1	98.9

TABLE V

CASES OF PRE-ECLAMPSIA AMONG PREGNANT TEEN-AGE PATIENTS, JANUARY 1965 THROUGH JUNE 1968

Age	NUMBER OF CASES: Mild Pre-Eclampsia	Severe Pre-Eclampsia	Total	Percentage
11	0	0	0	0
12	0	0	0	0
13	2	1	3	14.2
14	7	3	10	10.9
15	16	2	18	7.7
16	33	5	38	8.3
17	6	1	7	2.7

Table IV lists the prenatal complications found in this group of pregnant teen-agers.

Pre-eclampsia was diagnosed in 76 patients (7.3 per cent). Sixty-three were classified as mild and 12 as severe pre-eclampsia (Table V). Excessive weight gain was found without other signs of pre-eclampsia in 44 patients (4.2 per cent) who were not included in the pre-eclamptic group. There was one case of eclampsia (0.09 per cent) in the study group. This patient was a 14-year-old girl, gravida 1, para 1, who developed eclamptic seizures during a prolonged induced labor. Labor was ended by abdominal section due to fetal distress.

The incidence of toxemia in the control group was 5.1 per cent.

Some authors [15, 22] have pointed out that toxemia among teen-age mothers is more common in Negroes than in whites. We cannot make a similar valid comparison because of the poverty of white patients.

It has long been noted that the incidence of pre-eclampsia is higher among teen-age patients than among older pregnant patients [1, 10, 15, 16]. It also seems that the incidence rises as the age decreases, as can be seen, to some extent, from Table V.

Less frequent complications include placenta previa, diabetes mellitus, and cephalopelvic disproportion.

TABLE IV

PRENATAL COMPLICATIONS AMONG PREGNANT TEEN-AGE PATIENTS, CHICAGO LYING-IN HOSPITAL, JANUARY, 1965 THROUGH JUNE 1968

Trichomonas vaginalis	248	
Moniliasis	136	
Combined infection (trichomonas + monilia)	47	445
Gonorrhea	14	
Iron deficiency anemia	128	
Sickle cell trait	15	145
Sickle cell anemia	2	
Rh negative	41	
Allergy to penicillin	12	
Rheumatic heart disease	9	
Contracted pelvis	7	
Diabetes mellitus	3	
Placenta previa	2	
Lues	1	

Duration of pregnancy

The pregnancy lasted between 280 and 294 days in 916 (88.8 per cent) patients. In 105 patients (10.1 per cent), it was shorter than 280 days, and in 12 patients it was (1.1 per cent) it was longer than 294 days (postmaturity). In these last the labor was induced. Postmaturity in our control group was 3.3 per cent.

Duration of labor

Various authors [2, 14, 15, 16, 22, 23, 28] report different durations of labor in patients younger than 17. The average length reported varies from approximately 9 hours and 40 minutes to 16 hours and 32 minutes. Clough [5], who reports the shortest average of 9 hours and 40 minutes, partially explains his statistics by the frequent use of operative ending of labor with forceps application and delivery. In the majority of our cases (955 or 92.7 per cent) the labor lasted between six and 24 hours. In 56 patients (5.2 per cent) it was only three to five hours but in 22 patients (2.1 per cent) it was longer than 24 hours. The labor in two patients (0.19 per cent) was longer than 40 hours, but in three (0.29 per cent) less than three hours, ending in precipitate delivery. Precipitate delivery in the control group occurred in 5.8 per cent of the patients.

Prolonged labor

There is not yet a uniform opinion with regard to the definition of prolonged labor in primiparas. According to some authors [3, 7, 16] normal labor in primiparas lasts up to 30 hours, but others [2, 10, 18] allow only 24 hours. Greenhill and Clark [4, 11] think that 18 to 20 hours

should be the upper limit of duration of labor in primiparas under normal circumstances.

In our clinical material every labor in a primipara lasting longer than twenty-four hours was classified as prolonged. In twenty-two patients (2.1 per cent) from the study group the duration of labor was prolonged. The incidence of prolonged labor in the control group was 2.3 per cent.

Fetal presentation and position

The incidence of breech (24 or 2.3 per cent), shoulder (2 or 0.2 per cent), occipito-posterior (21 or two per cent) and occipito-transverse (12 or 1.4 per cent) positions was less in our studied clinical material than in the adult group of patients where the incidence was 3.2, 0.3, 2.7 and 2.1 per cent respectively.

Type of deliveries

Delivery in the majority of our cases was ended by low forceps or spontaneous delivery. Abdominal section was done in 34 patients (3.24 per cent). The incidence of section in the control group was 5.1 per cent. Cephalopelvic disproportion was recorded in only seven cases (0.68 per cent). The most frequent indications for abdominal section were: repeat section, fetal distress, and cephalopelvic disproportion (Table VI).

Complications intrapartum

Complications during labor were not numerous. The most common complication was perineal laceration (23 or 2.2 per cent). All lacerations were extensions of episiotomies.

Intrapartum bleeding occurred in 21 patients (2.03 per cent). The causes of bleeding were various: premature separation of the placenta, placenta adherence, cervical and vaginal lacerations. There were no cases of uterine rupture, placenta accreta, increta, or percreta. The incidence of intrapartum bleeding in our control group was 3.2 per cent.

TABLE VI

INDICATIONS FOR ABDOMINAL SECTION IN 34 TEEN-AGE PATIENTS

Indications	Number of Patients
Repeat section	10
Fetal distress with abruptio placentae	7
Cephalo-pelvic disproportion	7
Fetal distress with severe toxemia	4
Prolonged labor with fetal distress	2
Placenta previa	2
Pelvic mass (ovarian cyst)	1
Prolapsed cord	1

Complications postpartum

The most frequent postpartum complications in our patients were urinary tract infections (33 or 3.1 per cent) and endometritis (30 or 2.9 per cent). No puerperal sepsis, embolism, thrombophlebitis, or mastitis was noted.

There was one maternal death which will be briefly described. The patient was 16 years old, Negro, gravida one, para zero, in the 36th week of gestation. She was admitted to the hospital at 3:45 in the morning for observation and treatment for severe headaches. On admission she was conscious, well oriented in space and time, and not in labor. Vital signs were: blood pressure 170/110; pulse 100. She had no pedal edema and her urine was negative for proteins. Her hematocrit was 32 per cent. Examination of the eyes showed a mydriasis and fixed left pupil with a small and reactive right pupil. Because of the abnormal findings in the eyes, a lumbar puncture was done in the emergency room which showed a pressure of 370 mmHg. Very soon after admission (around 3:55 a.m.) the patient became comatous and at 4:00 a.m. developed pulmonary edema. She was examined by a neurosurgeon at that time and the diagnosis of subarachnoid bleeding was made. At 5:00 a.m. section was performed and a female baby weighing 1900 gm was born. The baby was in good condition but soon showed signs of respiratory distress (hyaline membrane disease).

The patient expired at 6:30 a.m. about two hours and forty-five minutes after admission. The autopsy report was as follows: "Intracerebral hemorrhage (left parietal region) with parasagital cortical necrosis and small recent subdural hematoma". The infant survived respiratory distress and was discharged from the hospital in good condition.

Twin pregnancy

Only five of our 1,033 analyzed cases delivered twins for an incidence of one in 206 deliveries. This is far less than a general accepted ratio in this country of one to 93.3 found by Guttmacher [12].

The incidence of twins in our comparable group was one in 89.8 deliveries.

Babies' weight

Of the 1,038 babies born, 165 or 15.9 per cent were classified as premature since their weights were below 2500 gm. Although the prenatal care, in our opinion, was generally adequate, the percentage of prematurity in the analyzed group of teen-age mothers was very high if compared with that of the general population which is approximately 9.8 per cent [5]. The majority of babies from these patients (399) weighed between 3001 and

3500 gm. Prematurity in the control group of patients was found in 7.3 per cent.

Birth injuries

Birth injuries in the babies included: three fractures of the clavicle; one left facial paralysis; eleven cephalhematomas, and eight cases with intracranial bleeding. Of these eight cases of intracranial bleeding, three cases had traumatic injuries of the central nervous system (one of these babies died and the other two survived). In the other five cases, ventricular and subarachnoidal bleeding was found at autopsy, probably due to severe anoxia according to the patho-histological report.

Fetal malformations

The incidence of fetal malformations in the general population is approximately two to three per cent [8]. In babies of our teen-age patients, malformations were seen in only eleven (1.06 per cent) cases, but in babies of mothers older than the age of seventeen delivered during the same period of time, fetal anomalies were found in three per cent.

Fetal and neonatal mortality rate

There were 21 deaths among the 1,038 babies of our adolescent patients (perinatal death rate of 20.2 per 1,000 live births). Fourteen babies were stillborn (five stillborn mature and nine stillborn premature). The cause of death for all mature stillborn babies was intrauterine asphyxia (patho-histological report). Seven babies died during the first seven days after birth (two mature and five premature). One mature baby died of intracranial bleeding and the other of bronchopneumonia. This last was born by section; the mother had intrapartum eclampsia. Three premature babies died of hyaline membrane disease, but the other two died of prematurity since no definite causes were found on autopsy. The incidence of perinatal mortality in babies of patients from our control group was 18.1 per 1,000 live births.

Discussion

The 10.8 per cent incidence of adolescent pregnancy in this study is higher than that reported in all previously published studies. For example, Marchetti and Menaker [16] report an incidence of 6.3 per cent; Hacker and associates [13] 9.7 per cent; Von der Ahe and Bach [28] 5.4 per cent; Hassan and Falls [15] 8.3 per cent, and Aznar and Bennett [2] 3.6 per cent.

Up to ten years ago gravidas younger than 17 years were in a sense neglected with regard to prenatal control, mainly because of their unmarried state and low socioeconomic status. The great majority of such patients came for delivery without previous medical care as well as with no psychological preparation for such an important event. As a sequela of this state, the courses of pregnancy, labor, and the postpartum period were followed by common complications including toxemia, prolonged labor, stillbirth, postpartum hemorrhage, infection, etc. Today, however, much less shame is associated with the unwed pregnant state, and married and unmarried young gravidas come regularly for prenatal control and receive equal if not more medical attention. The young patients who were without any prenatal care could not escape tremendous fear, worry, and unhappiness. These strong emotions might have been causative factors in uterine dysfunctions which in turn led to an increased incidence of prolonged labor, particularly in the latent phase.

We noticed that, in the very young gravida who had incomplete or no prenatal care, there was a predisposition to hypertonic uterine contractions during the latent phase or the beginning of labor. In these cases the uterus is not relaxed between contractions but tonic and tender on palpation. This observation remains to be confirmed by more authors in the future. We believe that regular prenatal care and skillfully conducted psycho-prophylaxis of labor would probably minimize this labor problem.

The incidence of toxemia of pregnancy in teen-age patients varies considerably in the literature, i.e. from 4.3 per cent to 23.5 per cent. The lowest percentage of 4.3 per cent has been reported by Arnot and Nelson [1] and the highest or 23.5 per cent by Seland [25]. Mussio [19] reported the highest incidence of toxemia (28 per cent) in the 12 and 13 year old. The critical age with the highest incidence of toxemia at the Chicago Lying-in Hospital was 13 and 14 years.

In our clinical material the incidence of toxemia reached 7.3 per cent. Eclampsia resulted between 0.13 per cent and 3.2 per cent in patients analyzed and observed by various authors [1, 2, 15, 16, 29, 31]. Its incidence of 0.09 per cent in our patients is the lowest thus far reported.

Excessive weight gain (over 20 or 25 pounds) during pregnancy in patients between 11 and 17 years of age with no other signs and symptoms of toxemia should not cause major concern for their physicians. Girls at this age have an average physiological non-pregnant weight gain of five to ten pounds per year [15].

Complications intra and postpartum were no more frequent than in the other older patients. We emphasize that thrombophlebitis, puerperal sepsis, mastitis, and embolism were not noticed in any of our cases.

The incidence of premature labor of 15.9 per cent in our adolescent mothers is similar to that reported by other authors [2, 9, 15, 16, 22, 23] i.e. 10.4 per cent to 18.7 per cent, but in our opinion is very high in comparison

› patients from the control group in whom the incidence f prematurity was 7.3 per cent.

Perinatal mortality for all infants with a birth weight ver 1000 gm reported by Potter and Davis [24] from Chicago Lying-in Hospital fell from 36.0 per 1,000 live births n 1931-1941 to 20.7 per 1,000 live births in 1951-1956 nd stood at 19.6 per 1,000 live births in 1961-1966. Our erinatal mortality figure is 20.2 per 1,000 live births.

Conclusions

1. Pregnancy and labor in persons seventeen years old nd younger is increasing at the Chicago Lying-in Hosital and the incidence in the last several years is approximately 10.8 per cent.

2. The most common complications during pregnancy n our patients were anemia, premature labor, vaginal nfection, and to some extent pre-eclampsia.

3. Uterine hypertony as a form of dystocia seems to be relatively common problem during labor mainly in dolescent patients who had insufficient or no prenatal are with psychological support.

4. The most common postpartum complications were: rinary tract infection and puerperal endometritis.

5. Pregnancy and motherhood in girls seventeen years nd younger is a serious and sometimes disastrous problem. It frequently disturbs the education of the patient, revents complete formation of an independent and nature personality, and generates misunderstandings and esentment in the family circle. The psychological trauma nay leave an indelible scar on the patients' impressionble personality. They are not sufficiently mature for complete understanding and acceptance of their new roles nd duties as mothers [17, 26, 27].

The high incidence of pregnancy in adolescent girls nd the resulting complications can be decreased by improving socio-economic conditions as well as the education of adolescent girls and their parents.

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INDUCTION OF LABOR

INDUCTION OF LABOR

The safest method of medical induction of labor is by administration of dilute intravenous oxytocin.[1-3]

The use of intramuscular injections of oxytocin in the prenatal period for the induction or stimulation of labor should be discontinued.[2]

Buccal Pitocin, recently gaining in popularity, has been found to be as potentially harmful as intramuscular injections, in that the exact dosage is difficult to titrate to the patient's needs. In a report of a long-term evaluation, Chalmers and Moorehouse[4] now suggest the use of a total of only 500 units of oxytocin to achieve induction instead of their original 4000 units. Recent literature records instances of uterine rupture during buccal pitocin administration[5], which points up the potential dangers of the drug, and the need for close and constant monitoring of the patients who are receiving it.

Sparteine sulfate administration has been shown, in a controlled double-blind study, to have a high incidence of tumultuous labor, hypertonus, poor uterine relaxation, and fetal bradycardia.[6] Obviously the disadvantages of this drug far outweigh the advantages, and suggest that it is not suitable for induction of labor.

The revised oxytocic standards (March 1972) of the American College of Obstetricians and Gynecologists are:[7]

a. Induction or stimulation of labor: Oxytocic agents should be used only when qualified personnel, as determined by the hospital staff and administration, can attend the patient closely. It is recommended that:
   1. The attending physician evaluate the patient for induction or stimulation especially with regard to indications.
   2. The person starting the oxytocin be familiar with its effects and complications, and qualified to identify both maternal complications and fetal distress.
   3. A qualified physician be immediately available to manage any complications.
   4. When oxytocin is being administered it is recommended that an

infusion pump, or other device for accurate control of rate of flow, be used. Also, the following should be recorded every 10 minutes: fetal heart rate, frequency and character of contractions, rate of flow of the oxytocin, and maternal blood pressures.

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Chapter 75

Induction of Labor

John J. Bonica, M.D., and John S. McDonald, M.D.

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BASIC CONSIDERATIONS

Artificial rupture of the membranes, the oldest technique for the induction of labor, was reportedly performed by Soranus of Greece in 100 A.D., but it was not until 1906 that the effect of posterior pituitary extract was documented by Dale.[15] In the early 1900's numerous mechanical and medical means of dealing with uterine dystocia existed, but all produced variable and inconsistent effects. Consequently, Hofbauer's[30] published account in 1911 of six successful inductions with pituitary extract aroused tremendous interest. A few successful case histories were recorded but abuse soon produced reports of serious maternal and fetal morbidity and mortality. Watson's criteria, illustrative of the misuse of pituitary extract, included administration in instances of pelvic contracture to obviate the need for forceps and in cases of placenta praevia to minimize hemorrhage.[57] Physicians then claimed that use of pituitary extract obviated forceps delivery in hundreds of instances.[28, 37, 56] In 1918 DeLee[17] introduced an era of moderation and advised extreme caution in using the drug. He developed guidelines still used today, such as careful administration, close monitoring of the contractions, and avoidance of the technique in patients with pelvic contracture and abnormal fetal positions.[17] Williams,[58] Adair,[1] Kosmak,[36] Davis,[16] and other eminent obstetricians, endorsed this conservative attitude and believed the use of pituitary extract before the delivery of the fetus was extremely dangerous. In 1928 a major contribution was made by Kamm,[32] who developed a technique for separating the pressor from the oxytocic fraction of pituitrin. Finally, by 1948, intravenous administration of dilute oxytocic solutions became common practice. During the past two decades the use of this technique has steadily increased. It is probable that in the future induction of labor will be used even more widely as scientific advances will permit the elimination of all risks to mother and fetus inherent in the method, and will actually enable us to provide the parturient and her offspring with the potential benefits of scheduled delivery.

Indications

Labor may be induced electively for the convenience of patient and physician or medically

for an indicated therapeutic reason. Previously, the most common induction was the therapeutic one, but today in many centers the most common reason for elective induction is patient convenience and physician conservation. Causes of therapeutic induction include toxemia, premature rupture of the membranes and suspected postdatism. Another common reason for induction in some areas arises when a multiparous patient with a history of short labors lives far from the hospital.[21] Other factors responsible for induction are outlined in Table 75-1. Each of these conditions is considered in detail in Sections 9, 10, and 11.

Table 75-1. Indications for Induction of Labor

MEDICAL

Maternal Disease
- Diabetes
- Hypertension
- Renal disorders
- Tuberculosis
- Other medical disorders (Section 9)

Obstetric Disorders
- Toxemias of pregnancy
- Marginal placenta praevia
- Abruptio placentae
- Hydramnios
- Other obstetric problems (Section 10)

Fetal Disorders
- Erythroblastosis
- Intrauterine death of fetus
- Truly prolonged pregnancy (postmaturity)
- Habitual death of viable fetus in utero before term
- Other fetal disorders (Section 11)

ELECTIVE INDUCTION

Prerequisites

Careful selection of patients based upon a complete and comprehensive examination of history, fetal size, pelvic and cervical conditions must precede any attempt at induction:

(a) There should be no cephalopelvic disproportion, and the cervix should be dilated 2 cm. and effaced at least 20 per cent;

(b) The fetus should be normal and weigh an estimated 2,800 Gm.;

(c) The presenting part should be fixed in the pelvis.

Failure to adhere to these prerequisites results in prematurity, prolonged labor, and induction failures. An elaboration of other criteria is found in Table 75-2. Naturally, a therapeutic induction for toxemia, diabetes or abruptio placentae need not have the cervical requirements as outlined since these conditions are emergent ones and since dilation usually proceeds rapidly regardless of the initial status of the cervix. The Bishop score was introduced in 1964 to aid the physician in his selection of suitable induction candidates (Table 75-3).[4] Friedman's evaluation of this scoring index revealed several interesting features.[23] First, he found cervical dilation often four times greater and consistency two times greater in importance than cervical position in predicting the course of the first stage of labor. Secondly, he concluded that cervical position played very little role in predicting anything. Thirdly, he found an over-all induction success of 94 per cent with an index score of five or greater. Finally, he discovered a score of nine or more was associated with a 100 per cent suc-

Table 75-2. Criteria for Induction of Labor

Gestational
- Last menstrual period
- Quickening
- Expected date of confinement based upon 1 and 2 above
- Previous gestation lengths and newborn weights

Fetal
- Size greater than 2,800 Gm. by clinical estimate
- Vertex presentation
- Absence of fetal malposition
- Single intrauterine gestation

Maternal
- Pelvis of adequate size and configuration
- Fixed fetal head
- Cervical dilation of 2 cm. and effacement of 20 per cent
- Absence of uterine scarring
- Complete acquiescence

Table 75-3. Bishop Induction Score*

Factor	*0*	*1*	*2*	*3*
Dilation	Closed	1 – 2	3 – 4	5
Effacement	0 – 30	40 – 50	60 – 70	80
Station	–3	–2	–1, 0	+1, +2
Consistency	Firm	Medium	Soft	–
Position	Posterior	Midposition	Anterior	–

*From Bishop.[4]

cess rate, five to eight with a 95 per cent rate, and less than four still with an 80 per cent success rate. Unfortunately, a score of 9 is found in less than 25 per cent of all multiparous patients — those with a cervical effacement of 80 per cent and dilation of 5 cm., and the fetal head engaged. This is one reason elective induction of labor has not become more popular in private practice. Usually physicians simply do not have the time required for careful evaluation, monitoring of contractions, and stimulation of the unsuccessful induction. Some therapeutic inductions require preliminary oxytocic preparation of the unripe cervix as advocated by Caldeyro-Barcia.[9] Reid,[47] in contradistinction to Friedman, believes that the state of ripeness of the internal os is a more favorable index for elective induction than the degree of dilation.

Fetal Maturity. Since prematurity is the single greatest hazard of induction of labor, every effort must be made to avoid this risk by accurately determining the age and development of the fetus. Traditionally, fetal weight estimation has been correlated with fetal maturity and the weight of 2,500 Gm. is well recognized as the arbitrary separation between prematurity and maturity. This concept, while under scrutiny at the present time, still appears to be a reasonable clinical yardstick. Careful examination of the history and palpation of the fetal parts through the uterine wall form the usual clinical subjective estimation of fetal size, with an error of ± 400 Gm. as an accepted possibility.

Radiologic techniques enhance this crude clinical criterion by detection of certain ossification centers.[38] The distal femoral epiphyseal center appears around 36 weeks as ill-defined clusters of calcium. If these are present, the fetus will be mature 96 per cent of the time. They are a valuable indicator but only if visible, since at 36 weeks only 80 per cent will be seen. Proximal tibial epiphyseal centers are visible in 75 per cent of mature newborns, and if they measure 5 mm. maturity is almost certain. The cuboid is present in about 30 per cent of mature newborns and serves as another reference. Developed cortical bone in the skull and long bones is also found in advanced fetal age, but alone it is not reliable. Visibility of a fetal fat line, with other more confirmatory signs as noted above, tends to support the impression of maturity.[48] An extensive study of the correlation between fetal head diameter and days until delivery and fetal weight was made by Jacobs.[31] However, fetal skull diameters are technically impossible to measure one third of the time and even under ideal circumstances there is a wide variation in predicted and real dates and weights. In two-thirds of the cases the delivery date can be ± 7 to 10 days and the weights ± 400 Gm.[34]

Ultrasonic techniques have also been used to determine the diameter of the fetal head. Cephalometry is performed by use of the A scope ultrasonic technique which has been described to possess an accuracy of ± 1 to 3 mm.[19, 52] The basic technique consists of directing an ultrasonic beam through the maternal pelvis in the region of the fetal head until a characteristic biparietal somogram is obtained which depicts the distance between the two outer tables of bone in the parietal area of the fetal skull.[50, 53, 59] Proponents of this technique state measurements can be performed in a harmless but extremely accurate fashion. Taylor[52] believes a biparietal diameter of greater than 8.5 cm. means the fetus will weigh more than 2,500 Gm. 91 per cent of the time, but a diameter of greater than 9 cm. signifies weight greater than 2,500 Gm. about 97 per cent of the time.

Laboratory methods for estimation of fetal maturity recently introduced offer a means of supplementing the techniques outlined. If the creatinine present in amniotic fluid is 2 mg. per cent or more the gestation is 37 weeks.[44] The disappearance of bilirubin pigments in the 450 mμ peak of nonsensitized Rh individuals is indicative of advancing fetal age. If the 450 mμ peak is greater than 0.01 the gestation is estimated to be less than 35 weeks, but if less than 0.01 it is believed greater than 36 weeks. This is based on the assumption that after 36 weeks the fetal liver is capable of conjugating bilirubin and excreting it via the placenta.[38] A Nile blue stain of amniotic fluid for identification of orange cells is believed related to fetal maturity of sebaceous glands.[6] Brosens[6] summarized these data (Table 75-4). More experience must be reported before these techniques can be viewed with confidence.

Table 75-4.

Gestation	*Per cent of Orange Cells*
Less than 34 weeks	Less than 1
34–38 weeks	1–10
38–40 weeks	10–50
Greater than 40 weeks	Greater than 50

Contraindications

Induction of labor is contraindicated in such conditions as: (1) malposition; (2) birth canal obstruction; (3) cephalopelvic disproportion; (4) lack of engagement; (5) total placenta praevia; (6) multiparity; and (7) previous cesarean section.

Physiopathology

The major hazards associated with induction of labor are prematurity, prolonged or tumultuous labor, and cord prolapse. The hazards of prematurity are real, sometimes unavoidable, often frustrating when unexpected, and frequently tragic. Prematurity still rates as the number one cause of perinatal mortality and, in spite of meticulous attention to gestational data and correlation of clinical findings of cervical ripeness with estimated fetal weight, a definite rate of prematurity exists. In two large series of elective inductions both Keettel and Hesseltine found a prematurity rate of 3.1 per cent.[29, 34] Prolonged labor is often associated with endometritis, which increases maternal morbidity, and with amnionitis and increased risk of neonatal pneumonia, both of which contribute to increased fetal morbidity. Tumultuous labor is caused by frequent and very intense uterine contractions which markedly impair intervillous space perfusion and result in fetal hypoxia and acidosis.[9, 26, 47] Uterine rupture terminating in both fetal and maternal death is an additional consequence of tumultuous labor.[34, 40] Prolapsed cord is often due to amniotomy performed at a high station or to elevation of the fetal head coincident with amniotomy, or as a consequence of an unexpected compound presentation.

Perinatal morbidity is increased if one or more of these complications are present. D'Esopo and associates[18] found a greater incidence of low Apgar scores among neonates born of mothers delivered with induced labor than among those delivered with spontaneous labor. Their assumption that this was due to rapid labors in the induced group suggests either that their selection was not optimal (i.e., poor cervical ripeness) or that there was overuse of oxytocin resulting in true precipitate labors. It seems that if the aforementioned prerequisites are fulfilled, there is no increase in neonatal morbidity. In a retrospective study of the developmental status of 4-year-old children, Niswander and associates[41] found no differences between term infants delivered through elective induction of labor and those infants delivered through spontaneous labor. Since prematurity is associated with an increased incidence of neurologic sequelae, inclusion of premature infants might have influenced the results. At least this study does indicate that among term infants the risk of induction is no greater than with spontaneous delivery all other things being equal. This is supported by data published by Keettel[33, 34] and Guttmacher.[27] Keettel and associates[34] did a retrospective study of 6,860 parturients who had had labor electively induced at term and found an uncorrected perinatal mortality of 1.4 per cent—a rate lower than that which occurred with spontaneous labor. The fact that 43 per cent of the perinatal deaths were related to the induction of labor (prematurity, cord prolapse, infection, precipitate labor) suggested that perinatal mortality could be decreased further by better selection and management of patients. This is supported by the results obtained subsequently in a group of 735 patients who had been more rigidly selected and whose perinatal mortality was 0.1 per cent.[33]

Maternal mortality is usually a direct result of uterine rupture. In Keettel's series of over 6,000 inductions by artificial rupture of membranes there was only one maternal death, but a maternal morbidity of 7.3 per cent.[34] This compared with 3 per cent in spontaneous vaginal delivery. In Guttmacher's series of 1,000 intravenous oxytocic inductions there were three uterine ruptures but no maternal mortality and a very low morbidity of 0.7 per cent.[27]

TECHNICAL CONSIDERATIONS

Labor may be initiated by medical or mechanical means, or both. Mechanical induction of labor by the use of a bougie or intrauterine catheter is outmoded. Castor oil and hot enemas alone give poor results, and the danger of quinine administration outweighs its limited usefulness. Today, in the majority of obstetric centers, induction of labor is being effected by (a) artificial rupture of the membranes; (b) stripping of the membranes; (c) artificial rupture of the membranes combined with the use of an oxytocic drug if needed; and (d) induction started with oxytocic drug followed by artificial rupture of the membranes sometime during the acceleration phase of labor. Of the oxytocic drugs

currently available, oxytocin administered by continuous intravenous infusion is the most frequently used.

Intravenous Oxytocin Infusion

There is a vast amount of evidence which suggests that the intravenous infusion of dilute oxytocin solution is the most accurate, safe, and efficient way to induce labor.[10, 12, 18, 21, 23] The oxytocin infusion should be administered in physiologic doses sufficient to produce contractions of normal intensity and frequency without significantly raising the resting tonus.[9, 10, 46] The work of Caldeyro-Barcia and associates[11] shows that uterine response to continuous intravenous infusion of oxytocin almost reaches its maximum value at 36 weeks and shows no significant changes between the 36th and 40th weeks and during labor (p. 312). At term they have found that infusion rates of 4 to 6 milliunits of oxytocin per minute are enough to raise uterine activity to a degree similar to that of spontaneous labor. Moreover, the intensity and pattern of uterine contractions are similar to those of spontaneous labor.[9–11, 22, 46] This suggests that induction of labor may be successfully carried out during this period if the drug is properly administered. Of course, the condition of the cervix is important, for Caldeyro-Barcia and associates[8, 10, 11] have noted that when the cervix is "ripe" less than 100 contractions of 40 mm. Hg or more intensity are needed to effect full dilation, as compared with 200 to 300 such contractions when the uterine cervix is unripe. Intramuscular administration of oxytocin has been advocated in the past along with the topical application of oxytocin to the nasal and buccal mucosa. However, these methods have not been shown to produce a stable level of uterine activity and have been found to be unreliable and difficult to control.

Technique of Administration of Oxytocin. The patient is admitted to the hospital the night before the proposed induction and is prepared in the usual manner. Nothing should be allowed by mouth after midnight. Physical examination on admission should be carried out. The perineum is "prepped" and the patient is given an enema. After the anesthesiologist has been informed and has seen the patient, the oxytocin infusion is started.

The best technique of administering oxytocin by infusion involves the use of a tandem arrangement (Fig. 75-1). Infusion from the larger

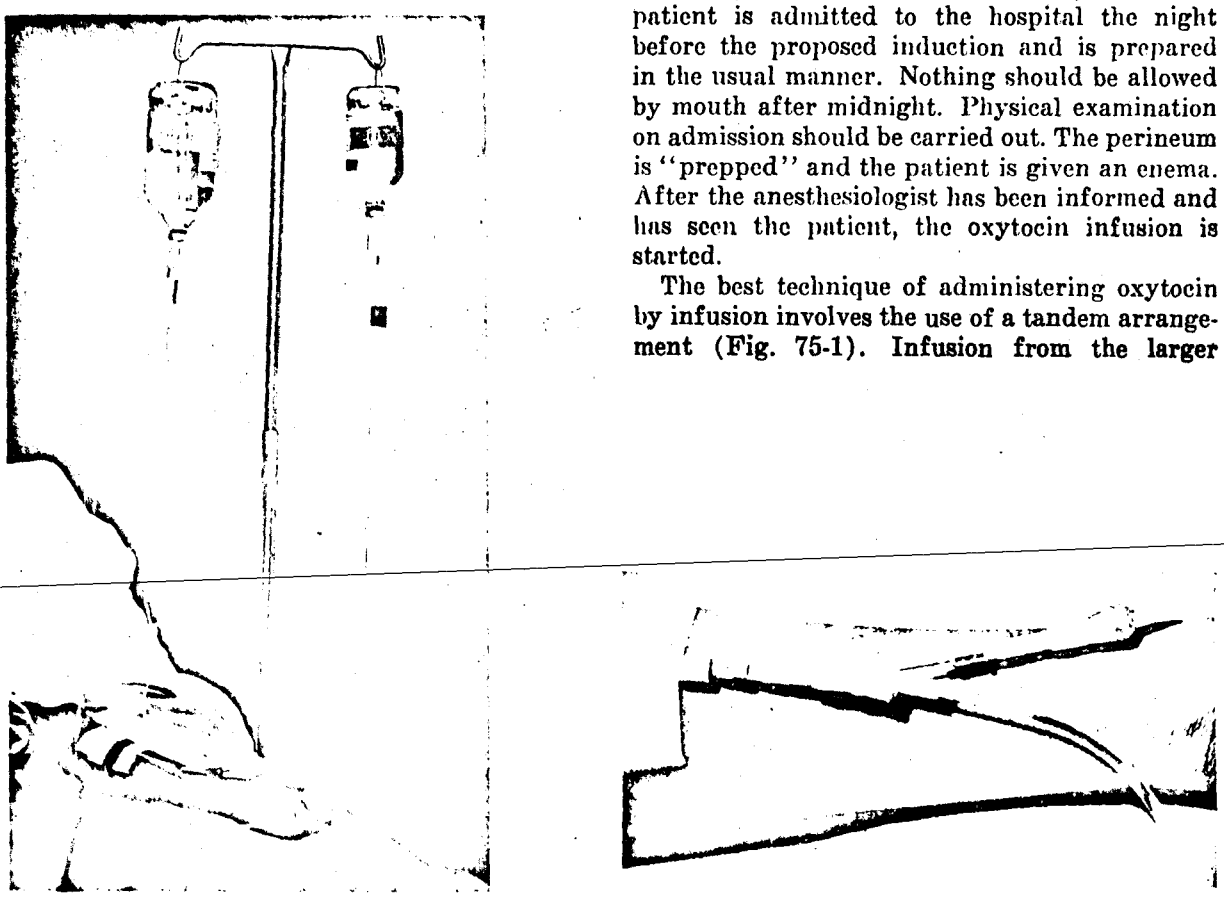

FIG. 75-1. Oxytocin infusion. *Left,* Two bottle set up with oxytocin bottle clearly marked and tubing flagged. *Right,* Close-up showing oxytocin infusion needle inserted into rubber tubing of glucose infusion.

(1,000 ml.) bottle containing plain 5 per cent dextrose in distilled water is started by inserting a 20-gauge needle in the forearm or the back of the hand. Once this infusion is established and is running well, infusion from the smaller (500 ml.) bottle, containing 5 units of oxytocin, is joined to the first infusion by inserting the needle of the second bottle into the tubing of the first bottle near its distal end. The objective is gradually to produce effective uterine contractions of good intensity, lasting 40 to 50 seconds and occurring every 2 to 3 minutes. Caldeyro-Barcia and his associates[9-11] use an infusion pump* which enables the obstetrician to administer oxytocin at exact and constant rates for an unlimited period of time.

Many clinicians prefer to rupture the membranes as soon as regular contractions are established. As labor progresses and a good contraction pattern is established, the rate of flow of oxytocin is reduced and then eliminated. The infusion is allowed to run for at least 30 to 40 minutes after delivery or as long as necessary to effect uterine hemostasis.

Other Medical Techniques

Sparteine. Sparteine sulfate administration of 150 mg. every hour until labor is effectively established is said to be simple and safe, and to require little physician attendance.[14, 45] However, in a controlled double blind study Van Voorhis and associates[55] found a high incidence of tumultuous labor, hypertonus, poor uterine relaxation and fetal bradycardia. Moreover, Pedrosian and Gamble[2] have also reported occurrence of uterine tetany and fetal distress, and Boyson[5] has reported 1 case of rupture of the uterus. Obviously the disadvantages of this technique far outweigh the advantages and suggest that this drug is not suitable for induction of labor.

Buccal Oxytocin. Experiences with buccal oxytocin have been similar to those obtained with sparteine sulfate. In a report of a long-term evaluation, Chalmers and Moorehouse[13] now suggest the use of a total of only 500 units of oxytocin to achieve induction instead of their original 1,000 units. The recent literature records six instances of uterine rupture during buccal oxytocin administration[11] data which suggest that this method of administration is dangerous and should be discarded.

Intra-amniotic Solutions. Intra-amniotic injection of a variety of solutions has now become an important adjunct in inducing labor in instances of missed abortion, therapeutic abortion, and intrauterine fetal death.[3, 25, 60, 61] These situations usually arise during the second or early third trimester and present the physician with the choice of either a protracted medical induction using high concentrations of oxytocin or a hysterotomy often associated with profuse hemorrhage.

The clinical efficacy of this technique has been well documented in over 400 cases reported in the recent world literature.[15] A number of chemical substances when injected into the amniotic space through the vaginal cul-de-sac or transabdominally will produce uterine contractions of sufficient force and number to effect either abortion or labor with delivery of the dead fetus. Wood et al.[61] successfully induced labor in all of their patients by the intra-amniotic injection of 50 per cent glucose solution. Bengtsson and Csapo[3] demonstrated the use of 20 per cent sodium chloride solution, which is the solution most widely used in this country for intra-amniotic injection. Patients undergoing therapeutic abortion after the twelfth week of gestation for such conditions as psychiatric disease and first trimester rubella infection, and patients with a dead fetus in utero, from whatever cause, are readily managed by this technique. The failure rate is greatest when the length of gestation is less than 15 weeks and the latent period likewise is longer. The technique is also somewhat more difficult because of the small size of the amniotic cavity.

The transabdominal method is generally used. Under aseptic conditions 200 ml. or more of amniotic fluid are withdrawn and replaced by hypertonic saline. The time from injection to delivery varies greatly but averages about 36 hours in most reported series. The earlier in pregnancy it is done the longer the delay in most instances. Complications have been relatively few but maternal deaths have been reported from accidental intravascular injection of hypertonic saline as well as from infections in those patients receiving 50 per cent glucose solution. Oxytocin for supplemental stimulation may be added.

*This pump may be obtained from the Harvard Apparatus Company, Cambridge, Massachusetts. The rate of infusion may be easily and accurately changed by moving a lever on the pump.

In most instances this method obviates the necessity for hysterotomy in cases of therapeutic abortion. In patients with the dead fetus syndrome the danger of fibrinogen deficiency may be circumvented by utilizing this technique and effecting rather prompt delivery. Failures are few. After the fifteenth week of gestation success has been reported in over 95 per cent of cases. In event of failure with the first attempt it may be repeated successfully.

Mechanical Techniques

Induction by mechanical means is still a very popular method because it demands less physician time. Artificial rupture of the membranes (Fig. 75-2) has been the most popular and dependable mechanical technique for inducing labor. Analysis of Keettel's series of over 6,000 inductions reveals that 75 per cent of the patients were in labor within 3 hours and that only 5 per cent experienced a prolonged latent phase. If labor does not ensue after 6 to 12 hours, oxytocin is administered; however, oxytocin administered immediately after artificial rupture of the membranes is regarded as stimulation superimposed upon induction. In most centers this is considered an unnecessary and dangerous practice.

Membrane stripping, another mechanical technique of inducing labor, consists of a digital separation of the membranes from the lower uterine segment. Many physicians use this method but never record their results or even that it was performed during a sterile vaginal examination. It is therefore difficult to estimate its efficiency, but Swann[51] reported one control study and summarized his experience by stating that 70 per cent of the patients whose membranes were stripped and 30 per cent of the control patients went into labor. Therefore it would appear to have some merit, but certainly not for a patient who must be delivered within a certain period of time. There does appear to be an increased maternal morbidity (10 per cent as opposed to less than 5 per cent for spontaneous deliveries) and there is always danger of manipulation of an unrecognized placenta praevia or risk of artificial rupture of the membranes.[51]

Maternal and Fetal Monitoring

The necessity for some type of continuous, accurate monitoring of mother and fetus is obvious. In the past monitoring consisted of manual palpation of the uterine wall, measurements of maternal blood pressure and pulse rate, and frequent periodic auscultation of the fetal heart rate, usually during uterine relaxation. It is now clear that to avoid all risks to the mother and fetus it is essential to monitor the various parameters continuously. New electronic techniques which permit this are available and should be considered essential for optimal intrapartum care in all instances, but especially during induction of labor when usually there is already a high-risk fetus. Continuous monitoring of the amniotic fluid pressure (p. 53) provides the only accurate measurement of uterine contractility. With this aid, the obstetrician can titrate the oxytocin infusion to the response of the uterus and thus minimize the risk of uterine hyperactivity and hypertonicity that usually results from a too-rapid administration of oxy-

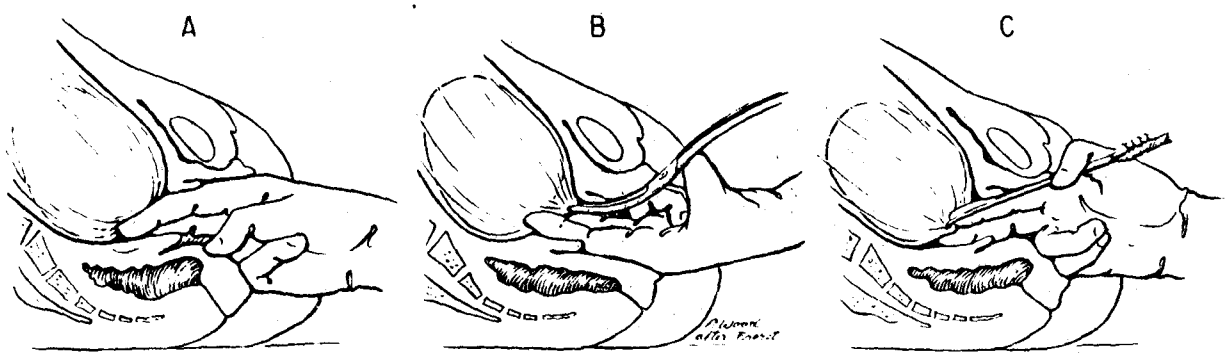

FIG. 75-2. Manual rupture of membranes. *A*, Separation of membranes from uterine wall. Membranes being ruptured with Bozeman forceps *(B)*, with membrane hook *(C)*. (Modified from Douglas, R. G., and Stromme, W. B.: Operative Obstetrics. Ed. 2, New York, Appleton-Century-Crofts, Inc., 1965.)

tocin. Although external tocography reflects trends along these lines, it is less reliable quantitatively than intra amniotic fluid pressure measurements. Fetal electrocardiography and fetal pH monitoring are equally important in improving the chances for delivery of the fetus in the best possible condition. These techniques are discussed in detail in Chapter 72.

The use of accurate monitoring, together with the scientific advances that will permit more accurate estimation of fetal maturity and refinement in our methods of inducing labor, will eliminate the hazards inhering in induction of labor. When such an objective has been achieved, a number of professional and sociologic forces will probably encourage an even greater use of elective induction of labor. The steady decrease in general practitioners who currently manage most deliveries will contribute to the obstetrician's load; this will necessitate changes in practice, including development of regional obstetric centers for the more efficient use of obstetrician, anesthesiologist and pediatrician. Some type of scheduled delivery under the constant vigilance of such a team may result in reduction of perinatal mortality and morbidity. Finally, the idea of a planned, orderly, well managed delivery appeals to most gravidas and their husbands.

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Sparteine sulfate: A potent, capricious oxytocic

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MEDICAL HISTORY repeats itself. Once again the physicians' desire for safe but effective pharmaceuticals has led to the widespread use and misuse of a new drug. This time it is sparteine sulfate.

This compound, isolated in 1851, was not used as an oxytocic until 1939.[13] Since then, sparteine sulfate has been a popular oxytocic in European countries. The pharmacology and use of this drug abroad were reviewed by Gray and Plentl,[13] in 1958. This article introduced sparteine sulfate into American obstetrical literature. Three years later, Plentl, Friedman, and Gray[19] described their experience with sparteine sulfate in a series of 1,208 patients, the first review of its type in the western hemisphere. Additional reports,[20, 21, 23] in 1962 and 1963, increased to over 1,700 the number of obstetrical patients treated with sparteine sulfate in this country.

These analyses and others[13, 25] suggested that sparteine sulfate was such a safe drug that it could be given without the constant attendance of the obstetrician, thus giving it a great advantage over oxytocin. This assumption has been found to be false, after thorough testing by American obstetricians in large clinics and small practices. Since 1963, reports[2, 4, 14, 17] of the dangers of sparteine sulfate have appeared.

Our purpose is to assess previous complications with sparteine sulfate; to record additional episodes of fetal distress, uterine tetany, and placental abruption following its use; and to demonstrate and discuss the unpredictable nature of this drug.

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Materials and methods

Sparteine sulfate was used at Kapiolani Maternity and Gynecological Hospital, Honolulu, Hawaii for the first time in October, 1961. From that time to August 1, 1963, a total of 322 pregnant patients received one or more intramuscular injections of sparteine sulfate. The obstetrical records of these patients were carefully reviewed, and all aspects of the use and effects of sparteine sulfate were evaluated.

There are 228 multiparous patients, and 94 nulliparas in this series. They received 1,064 injections of sparteine sulfate, the usual dosage schedule being an initial injection of 75 mg. followed by 150 mg. injections repeated at hourly intervals until a total of 600 mg. was given.

There were 193 attempted inductions of labor and 129 attempted augmentations of labor, one half of which were elective. Almost all the remaining inductions or augmentations were performed for premature rupture of membranes.

Results

Tetany is one of the more dangerous undesirable effects of sparteine sulfate. Four patients in this series developed tetanic uterine

contractions, as shown in Table I. In these patients, the time from the last injection of sparteine sulfate to the onset of tetany varied from 8 minutes to 4 hours. The duration of tetany was several minutes in each case, and fetal bradycardia was present in each. The fetal distress ended with the cessation of tetany in all patients except one. In Patient 63-617, the fetal heart could not be heard at the onset of tetany or thereafter, and the baby was stillborn.

It is of interest that three of these four episodes of uterine tetany occurred after the first injection of sparteine sulfate. In addition, the last and presumably the tetany-causing dose of sparteine sulfate was 150 mg. in 3 of these patients. Three fetuses subjected to tetanic contractions recovered and apparently were normal at delivery. The stillborn infant was autopsied, and cerebral changes indicative of anoxia, such as petechial hemorrhages, were found.

Four placental abruptions are recorded in this series. Two of these resulted in fetal compromise and are listed in Table II. In Patient 62-3917, the abruption presumably occurred one hour after the last dose of sparteine sulfate, but fetal distress was transient and an apparently healthy infant was delivered. The second patient (63-221) was given sparteine sulfate to induce labor after premature rupture of the membranes. However, she did not respond even after five injections. Normal fetal heart tones were heard, and she was transferred to the antenatal ward. Thirteen hours later, during which time she did not complain of any discomfort, the patient was re-examined and the

Table I. Four patients with uterine tetany following sparteine sulfate injection

Patient No.	*Total dose sparteine sulfate (mg.)*	*Last dose sparteine sulfate (mg.)*	*Onset of tetany after last sparteine sulfate (min.)*	*Duration of tetany (min.)*	*Fetal heart rate*	*Duration of bradycardia*	*Other complications*	*Time to delivery after tetany*	*Condition of infant*
61-6176	150	150	15	10	80/min.	10 Min.	Breech 3 cm. to 10 cm. in 10 min.	5 min.	Normal
61-6620	150	150	8	4	60/min.	4 Min.	4 cm. to 10 cm. in 30 min.	4¼ hr.	Normal
63-617	600	150	240	Several minutes	0/min.	Remainder of labor	5 cm. to 10 cm. in 30 min.	4½ hr.	Stillborn
63-1757	75	75	20	Several minutes	60/min.	Several minutes	None	5 hr.	Normal

Table II. Placental abruption with fetal distress

Patient No.	*Total dose sparteine sulfate (mg.)*	*Last dose sparteine sulfate (mg.)*	*Onset of fetal distress after last sparteine sulfate (min.)*	*Fetal heart rate*	*Duration of bradycardia*	*Placenta*	*Other complications*	*Time to delivery after fetal distress (hr.)*	*Condition of infant*
62-3947	675	150	60	100/min.	15 min.	Partial abruption	None	2	Normal
63-221	450	150	13 hr.	0/min.	24 hr.	Total abruption	Breech	24	Stillborn

fetal heartbeat could not be heard. The infant was delivered stillborn 24 hours later, and the placenta had sustained total abruption. Autopsy was not permitted, but there were no gross abnormalities.

A failure of sparteine sulfate was defined as a requirement for amniotomy, cesarean section, or oxytocin to effect delivery. Table III categorizes the sparteine sulfate failures in this series. The gross failure rate was 22 per cent: 32 per cent in nulliparas and 19 per cent in multiparas. Of particular interest is the marked difference between nulliparas and multiparas in response to attempted augmentation of labor with sparteine sulfate, in which the failure rate was four times higher in nulliparas.

Although they represent sparteine sulfate failures, 60 per cent of these patients reacted favorably to the initial dose of sparteine sulfate; there was no difference in the time to onset of sparteine sulfate effect (1½ to 2 hours) between these patients and those who completed labor without other treatment. In addition, the condition of the cervix was not related to the outcome of sparteine sulfate treatment.

The success or failure of sparteine sulfate was correlated with its rate of administration. Table IV indicates the dosage rates for all patients in this series, grouped according to the result of treatment. All multiparas who were successfully induced were given sparteine sulfate at a rate over 120 mg. per hour. In contrast, those patients who did not achieve delivery after sparteine sulfate stimulation were given less than 90 mg. per hour. Analysis of the successful inductions of nulliparas revealed that only 50 mg. per hour was needed to effect delivery without amni-

Table III. Sparteine sulfate failures

Use of sparteine sulfate	*No. of patients*		*No. of failures*		*Per cent of failures*	
Attempted induction	Nulliparas	34	Nulliparas	14	Nulliparas	41
	Multiparas	126	Multiparas	37	Multiparas	29
		160		51		32
Attempted augmentation						
	Nulliparas	53	Nulliparas	16	Nulliparas	30
	Multiparas	76	Multiparas	6	Multiparas	8
		129		22		17
Combined inductions						
	Nulliparas	7	Nulliparas	0	Nulliparas	0
	Multiparas	26	Multiparas	0	Multiparas	0
		33		0		0
	Nulliparas	94	Nulliparas	30	Nulliparas	32%
	Multiparas	228	Multiparas	43	Multiparas	19%
Total		322		73		22

Table IV. Sparteine sulfate related to rate of administration

Use of sparteine sulfate	*No. of patients*	*Average rate of sparteine sulfate administration (mg./hr.)*		
		All patients	*Nulliparas*	*Multiparas*
Successful				
Augmentations	107	170	160	197
Inductions	109	120	91	123
Combined inductions	33	175	190	177
Unsuccessful				
Augmentations	22	83	86	79
Inductions	51	66	76	63

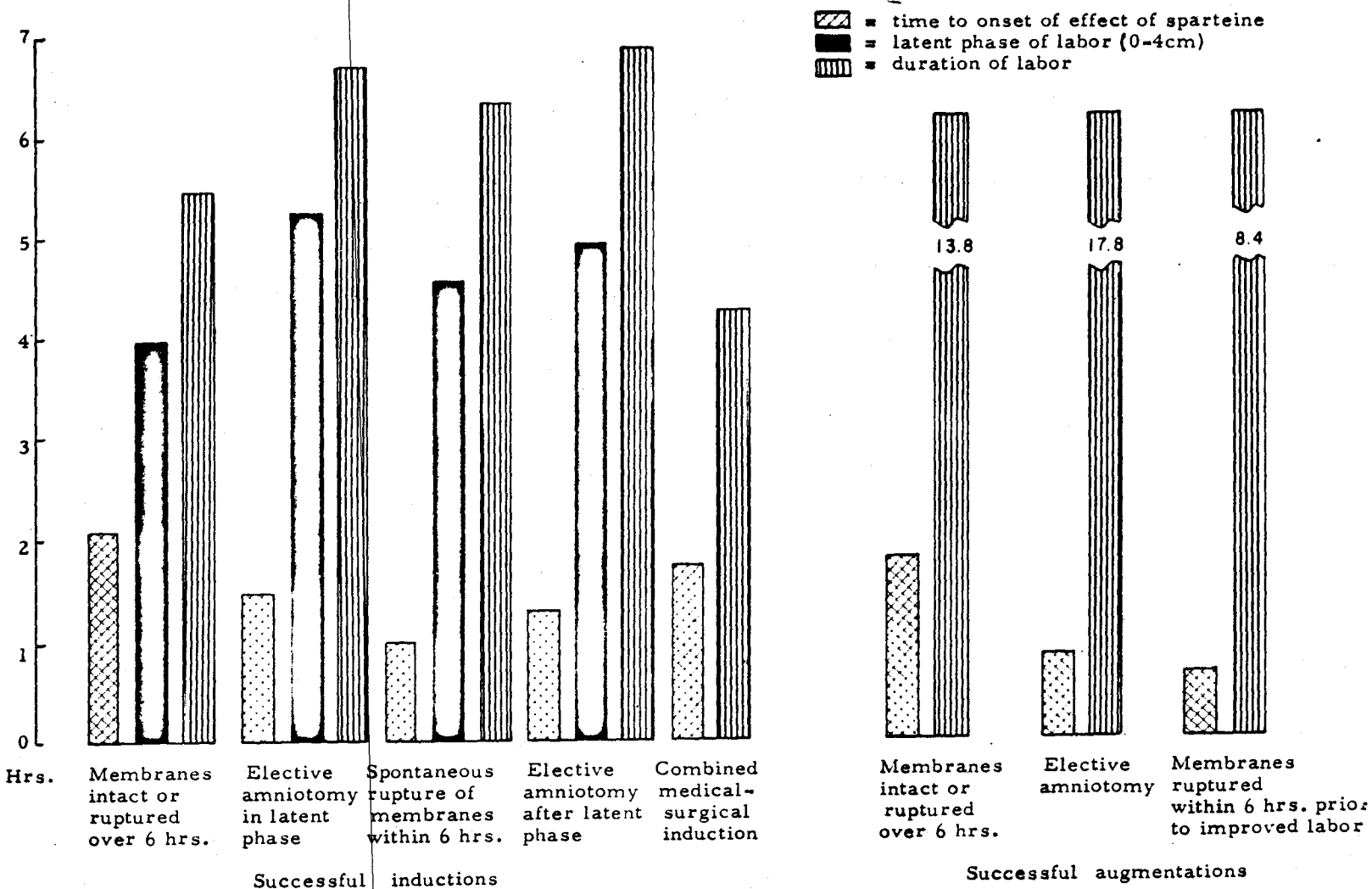

Fig. 1. Response to sparteine sulfate in successful cases.

tomy; when amniotomy was accomplished during the latent phase of labor, the dosage rate was 144 mg. per hour.

The average sparteine sulfate response time, latent phase of labor, and duration of labor in all cases treated successfully are presented graphically in Fig. 1. The time from administration of sparteine sulfate to its oxytocic effect varied within each group from several minutes to several hours. The shortest average latent phase and the shortest average labor occurred in the combined induction group (amniotomy and sparteine sulfate injection before labor).

Clinical experience indicates that intramuscular sparteine sulfate produces many tumultuous labors. Analysis of the labor records in this series, using the criteria of Friedman,[10, 11] disclosed that 104 patients had abnormally rapid labors; 77 were multiparas, 27 nulliparas. Perineal and cervical lacerations were not more frequent in these rapid labors. However, there were 12 episodes of fetal distress in this series (in addition to those reported above); in over half of these the only explanation was the tumultuous labor.

Comment

Dangers of sparteine sulfate. The reports in the American literature of adverse events following sparteine sulfate administration are summarized in Table V. These complications ranged in seriousness from tumultuous labor to uterine rupture and fetal death. If we add the unfavorable results of this study—4 placental abruptions, 4 episodes of uterine tetany, and 2 fetal deaths—the tally is as shown in Table VI:

These data are inadequate for conclusions regarding the incidence of fetal distress, tumultuous labor, or cervical lacerations. However, Cromer, Reeves, and Danforth[5] found a marked increase in tumultuous labor and cervical lacerations after sparteine sulfate treatment. In our series, the incidence of abnormally rapid labor was over 30 per cent.

Tumultuous labor may lead to placental abruption, uterine tetany, fetal death, and maternal lacerations. Even greater condemnation of tumultuous labor is found in the report of Barno,[1] who found, in a review of 15 maternal deaths due to amniotic fluid embolism, that 14 occurred following a violent labor.

Comparison of intramuscular sparteine sulfate and intravenous oxytocin in regard to incidence of adverse effects is pertinent. Two large series of patients to whom oxytocin was carefully administered intravenously were reported by Bishop[3] and Fields.[8] The complications in their patients which could be ascribed to the treatment are shown in Table VII.

Apparently, intravenous oxytocin results in a considerably higher incidence of placental abruptions and uterine tetany than does intramuscular sparteine sulfate. However, the most serious events, uterine rupture and fetal death, are just as frequent or are

Table V. Reports of undesirable effects attributable to sparteine sulfate

Authors	*No. of patients*	*Episodes of uterine tetany*	*Tumultuous labors and precipitate deliveries*	*Placental abruptions*	*Lacerations*		*Fetal death*
					Uterine	*Cervical*	
Plentl, Friedman, and Gray[19]	1,208	None	No data	2	None	No data	3
Savel and co-workers[21]	43	1	No data	1	None	No data	None
Stubblefield, Barloon, and Keltner[23]	100	1	More than 5	None	None	No data	None
Bedrosian and Gamble[2]	1	1	None	1	No	No	No
Boysen[4]	1	No	1	No	1	No	No
Plentl and Friedman[20]	407	None	7	None	None	No data	None
Marchick[17]	1	1	No	No data	No	No	1
Lahaye and Burkhart[15]	112	None	No data	1	None	No data	1
Filler, Filler, and Zinberg[9]	20	5	No data	None	None	No data	None
Cromer, Reeves, and Danforth[5]	872	None	175	None	None	140	None
Total	**2,765**	**9**	**Over 188**	**5**	**1**	**140**	**5**

Table VI. Obstetrical patients treated with intramuscular sparteine sulfate*

	No. of patients
Tetanic contractions	13
Placental abruptions	9
Uterine lacerations	1
Fetal deaths	7
Total	30

*From a series of 3,087 patients.

Table VII. No. of patients treated with intravenous oxytocin

	Bishop[3]	*Fields*[8]
Tetanic contractions	None	110
Placental abruptions	7	30
Uterine lacerations	None	None
Fetal deaths	1	None
Total	1,000	3,324

Table VIII. Patients given intramuscular oxytocin

	No. of patients
Tetanic contractions	2
Placental abruptions	None reported
Uterine lacerations	None
Fetal deaths	2
Total	714

even more frequent in patients treated with sparteine sulfate.

Another interesting comparison is that between intramuscular sparteine sulfate and intramuscular oxytocin. The latter method of inducing or augmenting labor has fallen into general disrepute in this country, and has been soundly condemned in Britain.[24] Daro,[6] Dieckmann,[7] and Miles[18] reported a total of 714 patients to whom oxytocin was carefully administered intramuscularly for induction or augmentation of labor. Their complications are reported in Table VIII.

Thus, the complications enumerated following sparteine sulfate administration are comparable in frequency to those following the much-maligned use of intramuscular oxytocin.

Unpredictability of sparteine sulfate. The extreme variability in effect of sparteine sulfate injections among different patients and even in the same patient was one of the most remarkable findings in this study. A marked range in time to onset or augmentation of labor was recorded. Moreover, it was impossible to predict the outcome of labor by the patient's initial response to sparteine sulfate.

Even patients highly responsive to sparteine sulfate differed markedly in the interval between the injection and the onset of action. Five patients in this series were so sensitive to sparteine sulfate that labor was induced and completed with only one injection. Yet, the period between the injection and onset of labor varied from 20 to 90 minutes in these 5 patients.

Although 3 episodes of uterine tetany occurred within 20 minutes of injection, in one patient (63-617, Table I) the tetanic contraction did not develop until 4 hours later. Poor absorption may be a factor in this case, and this may apply also to Patient 63-221 (Table II).

Stander, Thompson, and Stanley[22] recently published studies which showed that the reaction of the pregnant uterus to intramuscular injections of sparteine sulfate is quite variable, with a reaction time ranging from 4 to 20 minutes. Goodno and coworkers[12] in their series of in vitro and in vivo studies with sparteine sulfate also found a wide variation in the response to this drug. Landesman's[16] in vitro studies with uterine muscle strips demonstrated that tetany may occur at any time up to 40 minutes after administration. Finally, Filler, Filler, and Zinberg[9] in their series of 20 patients, found that even the same patient's response to the drug was highly variable. They described 5 tetanic contractions after 28 injections—a rate of nearly one in every 6 injections.

The foregoing inescapably leads to the conclusion that the intramuscular administration of sparteine is a dangerous, unpredictable method of stimulating or inducing labor. The in vitro studies referred to suggest that the drug is capricious by nature. However, review of the history of intra-

muscular and intravenous oxytocin hints that the intramuscular route of administration may be partly to blame. The chance of accidental intravenous administration, and the uncertainty of rate of absorption from fatty and edematous tissues of pregnant women, make the results of any potent drug unpredictable, and, therefore, dangerous, when that drug is given intramuscularly.

Summary and conclusions

Sparteine sulfate was administered intramuscularly for induction or stimulation of labor to 322 obstetric patients at Kapiolani Maternity and Gynecological Hospital in Honolulu, Hawaii, from Oct. 1, 1961, to August 1, 1963. The labor records of these patients were reviewed relative to significant complications of sparteine sulfate therapy. These difficulties may be divided into two groups:

A. Dangerous effects of sparteine sulfate.

1. Four tetanic uterine contractions developed, with one fetal death as a result.

2. Four placental abruptions occurred, causing one fetal death.

3. Abnormally rapid labor ensued in over one third of the patients and was the cause of more than half of the episodes of fetal distress in the series.

B. Unpredictability of sparteine sulfate.

1. The time to onset of effect ranged from minutes to hours.

2. The ultimate success or failure of treatment could not be predicted by the patient's response to the first injection.

3. Initial response was delayed as long as 1½ hours, even in patients highly sensitive to sparteine sulfate.

A review of the literature reveals that sparteine sulfate given as an oxytocic has resulted in a comparable percentage of fetal deaths and uterine ruptures as in a large series treated with intravenous oxytocin, and results in a rate of complications equivalent even to that of intramuscular oxytocin. Moreover, in vivo and in vitro studies have conclusively demonstrated the unpredictable behavior of sparteine sulfate.

Because of its extreme potency and capricious nature, sparteine sulfate can no longer be regarded as a "safe" oxytocic when administered intramuscularly. The obstetrician must give this drug with the same caution and careful attendance which is required in the use of intravenous oxytocin.

Appreciation is extended to Mrs. Mabel Okazaki, Records Librarian at Kapiolani Maternity and Gynecological Hospital, Honolulu, Hawaii, for her help in preparing this study.

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FOLLOWING PATIENTS IN LABOR

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The following practices should be routinely carried out on every laboring patient:[1]

1. The maternal temperature, pulse, and respiratory rate should be taken and recorded at least every 4 hours during labor.

2. The fetal heart rate should be recorded at least every 30 minutes during the first stage and every 5 minutes during the second stage of labor. If complications are present, oxytocin being administered for induction of labor, or if irregularities of rate or rhythm are occurring, recording should be done more frequently.

3. The maternal blood pressure should be taken and recorded every hour during labor.

4. Any variation from normal levels must be reported to the physician at once.

The management of the first stage of labor is in many instances the responsibility of the nurses working in the maternity unit. Nurses, therefore, should be well versed in the patterns of normal labor, and in normal progression of labor, so that the abnormal will be easily and readily identified. The prompt recognition of a dysfunctional labor pattern and early reporting of it to the physician is of great benefit to both the mother and the fetus.[2]

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1. Standards for Obstetric-Gynecologic Services: The American College of Obstetricians and Gynecologists 1969 Page 40

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The functional divisions of labor

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A schema is offered which divides clinical labor into three functional divisions—preparatory, dilatational, and pelvic. They are defined on the basis of cervical dilatation–time and descent-time patterns. Distinctive characteristics of each are discussed in terms of functional objectives, techniques of assessment, effects of adverse factors, manifestations of pathology, and therapy.

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IT IS A SPECIAL TRIBUTE to the wisdom, courage, and foresight of Howard C. Taylor, Jr., to whom this Festschrift is dedicated, that the well-worn and thoroughly studied area of clinical labor was deemed by him sufficiently worthy of fresh investigation to be enthusiastically supported in his department. By virtue of his personal encouragement, work on the application of graphic analytical techniques to the labor phenomenon was undertaken in 1951 and has been pursued essentially without interruption to date.

Through the intermediary of a simple device which allowed us to relate progressive changes in cervical dilatation and in station of the fetal presenting part to elapsed time in labor, we were privileged to enter upon an entirely new discipline of obstetrics. The technique of assessing progress in labor had earlier been rather nebulous. It was based on vague limits of expected change or on arbitrary maximum durations of total labor or of first or second stage. We now possessed a dependable and reliably accurate tool to help accomplish critical appraisal of labors in progress. A major part of the subjective art of obstetric evaluation and management was giving way to the objectivity of science.[5]

The characteristic patterns of dilatation and descent functions were uncovered, the dissected portions of the patterns were studied in all types of labor variants, the distribution of these derived data and their statistical limits were determined, and abnormalities were clearly defined. The catch-basket of uterine inertia was replaced by very specific, distinctively different, and diagnostically homogeneous disorders of aberrant progress. Having defined the several major disorders in this way, we proceeded to study each of them separately in turn to determine causative or contributory factors, effectiveness of treatment, and prognosis in terms of fetal and delivery outcome. Simultaneously, we were able to examine the effects of various influences acting to alter the course of labor, including both those with which the patient and her pregnancy are intrinsically endowed and those to which she is subject because of our ministrations as obstetricians.

As these studies evolved over the past two decades, an overview began to take form. With the completion of each succeeding investigation, it became more and more transparent that the labor phenomenon, its complexities and myriad variations notwithstanding, lent itself to being divided, like ancient Gaul, into three parts, each different, unique, and functionally special. Each of these separate functional parts could be easily recognized; each was affected by different factors; each responded in a singular manner to various influences; and each was subject to its own set of aberrations with distinctive etiology, therapy, and expected prog-

nosis. Recognition and understanding of these differences must perforce be considered essential for the practitioner in this field. For the sake of convenience, we shall refer to these functional divisions of labor merely in terms of their ostensible physiologic objectives, viz., preparatory, dilatational, and pelvic divisions, respectively. They are illustrated in Fig. 1, which presents a composite of average cervical dilatation–time and descent-time curves subdivided into component functional parts. Their clinical characteristics are separately listed in Table I for quick reference. For clarity, our terminology will distinguish the classical stages of labor, the phases of labor based on dilatation and descent patterns, and the functional divisions.

Preparatory division of labor

The arbitrary division of labor, in use universally heretofore, into first and second stages is based on the convention which recognizes a period of dilatation followed by another of descent. Textbooks succinctly define these intervals from onset of labor to full cervical dilatation and from full dilatation to birth of the baby, respectively. The so-called mechanisms of labor, referring to the accommodations of the fetal presenting part to the pelvis during its descent through the birth canal, are merely superimposed on this schema without specifically designating

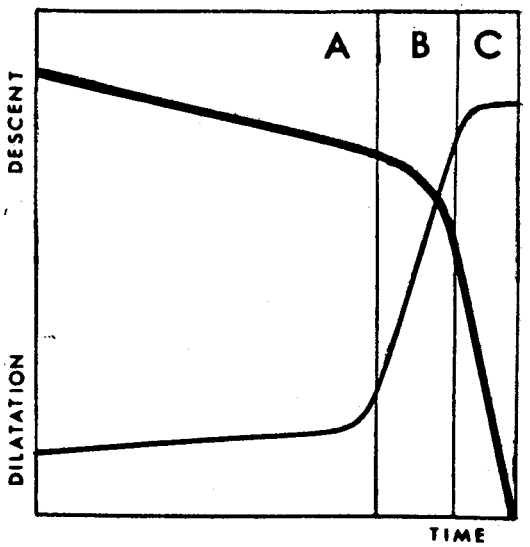


Fig. 1. Composite of cervical dilatation time (thin line) and descent time (heavy curve) as they interrelate. Total labor is divided into its functionally distinct parts by vertical lines. The functional divisions are *(A)* preparatory, *(B)* dilatational, and *(C)* pelvic.

Table I. Principle clinical features of the functional divisions of labor

Characteristics	*Preparatory division*	*Dilatational division*	*Pelvic division*
Functions fulfilled	Contractions coordinated cervix prepared	Cervix actively dilated	Pelvis negotiated by mechanisms of labor, descent and delivery
Interval included	From onset of contractions to end of acceleration phase	Phase of maximum slope	From onset of deceleration phase to delivery of infant
Measured by	Duration in time elapsed (hr.)	Rate of dilatation, linear (cm./hr.)	Rate of descent, linear (cm./hr.)
Adversely affected by	Excessive sedation, unprepared cervix	Myometrial dysfunction, disproportion	Disproportion
Secondary adverse factors	Myometrial dysfunction, false labor, anesthesia	Excessive sedation, anesthesia*	Malposition, anesthesia*
Pathology manifest by	Prolonged latent phase, prolonged acceleration phase (?)	Protracted active phase Secondary arrest of descent	Prolonged deceleration phase, protracted descent
Aberrations benefited by	Therapeutic rest	Support or stimulation†	Stimulation†

*Continuous caudal or epidural anesthesia when improperly administered, i.e., too early, too high level, or with coexisting adverse factor.

†Oxytocin stimulation indicated only in absence of cephalopelvic disproportion for arrested progress.

where they might fit temporally. Study of clinical labor phenomena provides some insight, particularly pointing out the inherent shortcomings of our current teachings. Only a very small portion of the first stage, for example, is actually occupied with cervical dilatation, and descent is not exclusively limited to the second stage.

Let us first examine that portion preceding dilatation. The onset of regular uterine contractions perceived by the patient is generally, but not universally, accepted as the onset of labor. There are many who require that the cervix be dilating before they will consider labor to have begun, essentially ignoring the sometimes quite long and potentially important interval prior to active dilatation. The debate is of more than academic interest. Physiologists stand firm because they cannot objectively distinguish contractility patterns before labor or even in false labor from those occurring after labor begins (as determined by the patient's subjective perception). Nevertheless, the interval from the onset thus defined and the beginning of active dilatation, which we have termed latent phase, has clinical significance. Aside from the sociologic aura surrounding childbirth that pertains here, there are important physiologic considerations.

We know essentially nothing about the mechanisms that trigger the onset of labor and only very little about the "warm-up" aspects of this preparatory division, but we are nevertheless aware that major changes take place. These may be expressed in subtle ways, as, for example, in the orientation, polarization, and coordination of myometrial contractility, or in obvious ways, such as in the softening and effacement of the cervix. They may also be expressed pathologically in the response of the fetus to periodic curtailment of his oxygen reserve by uterine contractions with production of hypoxic cardiotachometric patterns.[1, 10] A further abnormality will be manifest in the delayed evolution of effective contractility to overcome soft-part resistance resulting in aberrant prolongation of this phase. In order to stress the clinical significance of this aspect of labor, one need merely point out that cesarean section is not infrequently resorted to as a means for terminating pregnancy under the questionable assumption that a disordered state exists.[6]

The preparatory division may be considered to comprise that portion of labor from the onset of regular, perceived contractions to the end of the acceleration phase of dilatation. The latter point in time is marked by entry into that part of the active phase of dilatation which is characterized by linear maximum slope. Concurrently, the descent curve is flat in its own latent phase. We suggest that the latent and the acceleration phases of dilatation be looked at as if they were a single unit from the functional point of view. We do so because we recognize that both appear to be affected in the same direction and magnitude by the same beneficial or deleterious factors and both manifest related disorders which are correctable by similar management regimens.

In this regard we have learned that the preparatory division is particularly sensitive to sedation and anesthesia. The poorly polarized uterine contractions of this interval are easily disturbed. Myometrial function has not become sufficiently coordinated to withstand the inhibitory aspects of such exogenous influences. There is almost a proportionate dose-response relationship in this context, the greater the dosage of narcotic analgesic administered (and the greater the effect on the patient's sensorium), the longer the latent phase.[3] This ratio is distorted, of course, by other factors, most notably by the condition of the cervix itself at the onset of labor. In expected fashion, the more preparation needed to condition the cervix and other maternal soft tissues for later active dilatation, the longer the preparatory phase should be. This is precisely what has been encountered.[7] The changes that occur in the cervix during this interval are both overt and cryptic. The apparent transformation is seen with softening, effacement, and beginning dilatation; the subtle variations occur in the subcellular alterations of ground substance, collagen, reticulum, and other

fibrillar connective tissue components.[2] We have no ready explanation for the acute changes that take place in the functional capacity of the cervix at the end of the preparatory division and permit it to begin to respond by dilating to contractions that are essentially unchanged insofar as their effective centrifugal forces are concerned. An analogy might be drawn to the phenomenon of stone-cutting, in which many dozens of strokes with hammer against chisel may have no apparent effect, yet one more blow of the same intensity cleaves the stone sharply. The explanation must exist in as yet undetectable changes, perhaps in molecular orientation, that are produced by subliminal increments of force.

For want of more objective criteria of diagnosis, abnormalities of the preparatory division have been defined temporally on the basis of critical limits derived from distribution curves of collected relevant data. We have seen that the latent phase preceding active dilatation should not normally extend beyond 20 hr. in nulliparas or 14 hr. in multiparas. Comparable data are not available for the evanescent acceleration phase, which we have found difficult to measure accurately, but it is likely that abnormal prolongation of this phase exists as a pathologic state. Patients whose labors exceed latent phase limits have the disordered pattern called descriptively prolonged latent phase (Fig. 2). They have frequently been subjected to the deleterious effects of the several agents or influences one might expect to be at play, especially those we have already discussed, such as excessive sedation, anesthesia, and unfavorable cervical prelabor preparation. Some of these patients can be determined retrospectively to have been in false labor, others to be afflicted with some form of myometrial dysfunction later manifest as a recognizable aberrant entity of the active phase of dilatation or of descent. Unfortunately, the wisdom acquired through retrospection in these instances is not available to us in advance.

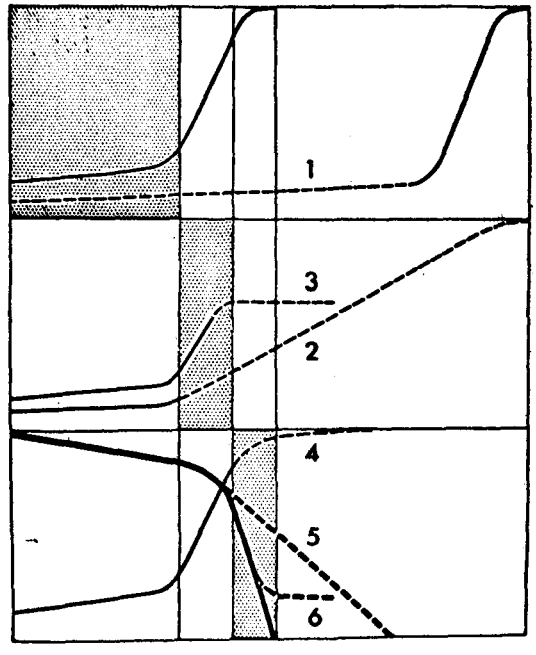


Fig. 2. Disorders of the functional divisions of labor presented graphically with cervical dilatation or fetal station as ordinate and time as abscissa. Top, the normal dilatation curve is compared with the preparatory division abnormality of *(1)* prolonged latent phase. Middle, dilatational division aberrant patterns include *(2)* protracted active-phase dilatation and *(3)* secondary arrest of dilatation. Bottom, pathologic states of the pelvic division encompass *(4)* prolonged deceleration phase, *(5)* protracted descent, and *(6)* arrest of descent. Unbroken portions of curves are normal aspects, broken lines, abnormal; thin lines are used to represent dilatation, thick, for descent. The relevant divisions in which the abnormalities become manifest are shaded.

Effective therapy of prolonged latent phase, the only objectively definable disorder of this preparatory division of labor, includes therapeutic rest or oxytocin infusion. Equivalent results can be achieved with either. However, rest with large doses of narcotic-analgesic agents is recommended for several reasons. Among these, considerations relevant to existence of unrecognized false labor, presence of emotional and physical exhaustion, and disordered electrolyte balance are foremost. The prognosis with this form of conservative management is quite good both with regard to delivery outcome and fetal morbidity and mortality rates. Most patients respond to effective uterine rest with normal active dilatation and descent, followed by vaginal delivery; those in

false labor can be recognized retrospectively by cessation of contractions; the small remaining number require oxytocin stimulation, unless this is contraindicated.

Dilatational division of labor

Although the first stage of labor is classically considered the stage during which dilatation occurs, it is well recognized that the cervix undergoes active dilatation only during a relatively small part of it. Aside from the dilatation that has occurred before labor begins and the negligible additional amount that takes place during the preparatory division (latent and acceleration phases), all dilatation except the terminal retraction aspects of the deceleration phase takes place during the relatively short interval of the phase of maximum slope. It is during this important span of time that the result of all the driving forces of uterine contraction acting on the uterine contents and against the resistance of the maternal soft parts becomes manifest.

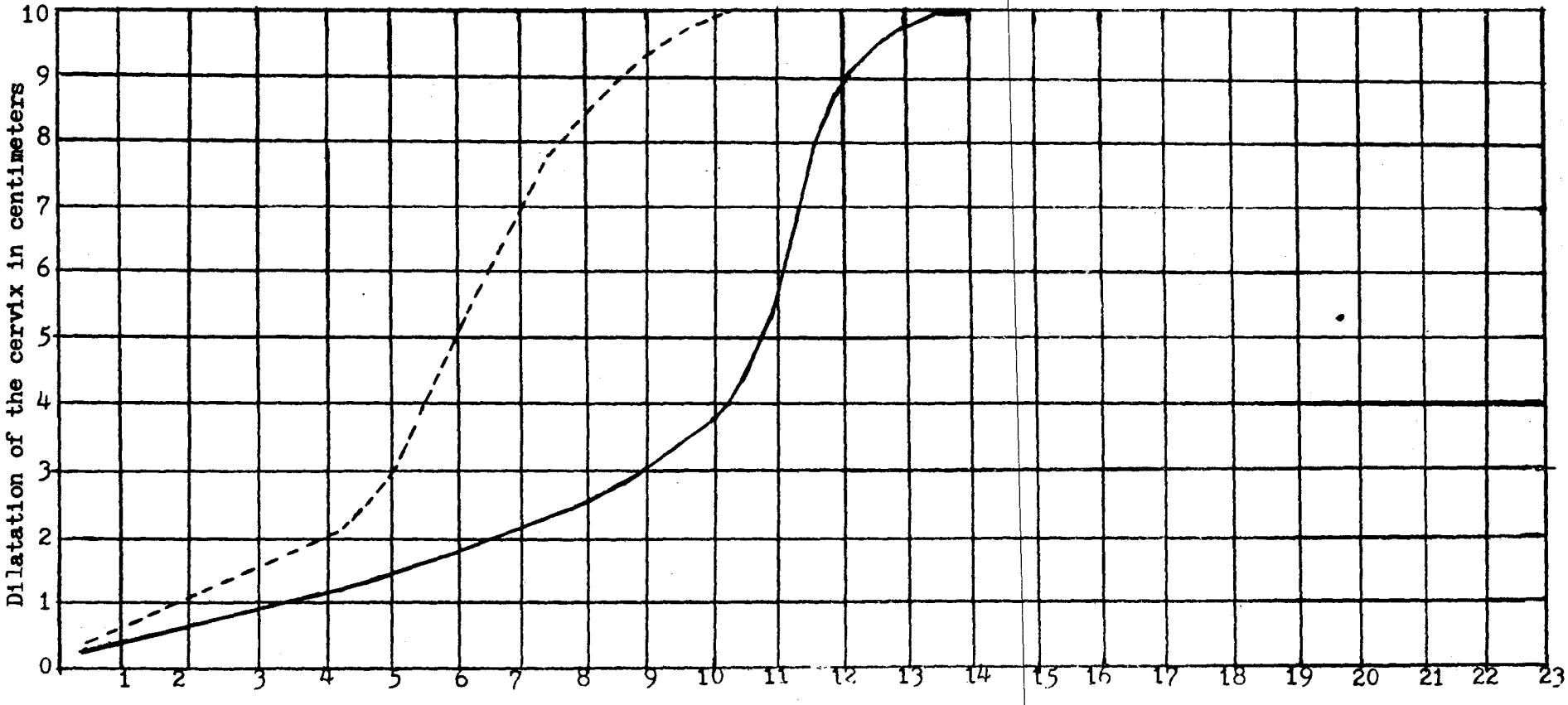
Dilatation of the cervix can be regarded in an engineering sense as the effective end result of these forces, very specifically reflecting the "efficiency" of the complex machine with which we are dealing. It is the linearity of the normal dilatation process, documented objectively,[4, 9] that permits us to determine the degree of effectiveness of a particular contractile pattern in overcoming the specific resistance present in a given patient in labor. Thus, a steeply inclined slope of dilatation is the result of a favorable combination of factors, regardless of the particular clinical picture of labor in terms of contractility pattern. By the same token, slow progress results from disadvantageous combinations. The term efficiency is used here in its mechanical sense, that is, the ratio of the useful energy delivered by a dynamic system (the work produced) to the energy supplied to it. This is an important concept that must be understood in order to make optimal use in practice of the measurements of dilatation and descent functions.

We have not included the deceleration phase of dilatation in this division because of its functional differences. Despite the fact that the retractive aspects of dilatation are continuing without interruption during this interval as the cervix "turns the corner" (see below), the descent phenomenon then in full progress takes on much greater significance. Since we are concerned with means for determining normal progression and, more pertinently, uncovering abnormalities, this distinction between dilatation and descent has practical significance. The dilatational division, therefore, for our purposes of functional consideration comprises only the phase of maximum slope.

In contrast to the preparatory division, the dilatational portion of labor is essentially unaffected by sedation or anesthesia administered for analgesic purposes. Myometrial function tends to be well established as illustrated by its ability to effect active dilatation. The inhibitory pharmacologic effects of exogenous agents, so readily demonstrable in the latent phase, are rarely seen here. It is only in the presence of a preexisting disordered state of myometrial function, difficult to define at best, or with some major intrinsic impedance, such as cephalopelvic disproportion, that any potentially deleterious influences of extraneous factors will become manifest. In brief, it is quite difficult to disturb the rapidly progressing dilatation curve. On the other hand, where the phase of maximum slope is progressing slowly or abnormally, the effects of negative factors may become apparent. Under such circumstances, excessive sedation or anesthesia can readily diminish or stop further progress.

The abnormalities of the phase of maximum slope are easily defined in terms of minimal critical limits of the rate of dilatation, which normally should not be less than 1.2 cm. per hour in nulliparas or 1.5 cm. per hour in multiparas. The cases of patients with the disorder of protracted active-phase dilatation (Fig. 2) are diagnosed by their abnormally slow dilatation rates. No form of active therapy for this disorder has been effective in speeding dilatation. However, progress is readily inhibited by seda-

FRIEDMAN LABOR GRAPH



----------: Multigravida
_____: Primagravida

FETAL NURSING DURING LABOR

Intensive nursing care for women in labor must include the guarding of the fetus. Therefore, the labor and delivery room nurse assumes the responsibility for two individuals, and the needs and problems of the fetus become especially demanding during labor and delivery. The obstetric nurse must be knowledgeable, alert, concerned, and conscientious in her care of her patients in the quest for a successful conclusion of the pregnancy.

The first objective of fetal nursing is to maintain the mother in the best possible state. Nursing care and knowledge that promotes proper physical and psychological conditions in the mother are applied here. If the mother becomes ill, of course therapeutic and restorative measures must be instituted.

It is important for the nurse to know the mother's past medical and obstetric history, as well as the prenatal history in the present pregnancy. In general, disease processes in the mother (diabetes, cardiac problems etc.) or disorders of the pregnancy itself (toxemia, bleeding, etc.) interfere with the growth and development of the fetus, thus approaches labor less able to stand the stress. Therefore, every hospital obstetrical unit should insist on a complete copy of the prenatal record being at the hospital before the patient is admitted in labor.

The nurse must also be well versed in the patterns of normal labor, in order to judge when an abnormal condition is developing and adding to the fetal endangerment.

The second objective of fetal nursing is to maintain the fetus in the best possible state. Let us consider the fetal situation during labor. Before birth the mother's body is her baby's total environment. Nursing care to promote fetal well-being must be directed at the mother, and since many dynamic processes are at work during labor and delivery, changes may take place from minute to minute. The nurse is limited by her inability to see the fetus. She cannot tell if he is peacefully curled up letting the maternal forces propel him smoothly through the birth process, or if he is flaccid and unresponsive, with his life in jeopardy from tight coils of cord around his neck or immersed in a sea of meconium. He cannot call "nurse" in his time of need or cannot even depend on his mother asking for help on his behalf.

How then, can the nurse achieve her objective? She must perform a critically important series of activities. She can observe maternal and fetal signs, record them, and report to the physician promptly any deviation from normal that she finds. It is, of course, the physician who must decide if obstetrical management is to be altered, and his judgment may be enhanced by the completeness and accuracy of the nursing observations. This is a heavy responsibility for the nurse, for she may be the only professional person present with the mother and fetus, particularly during the all important first stage of labor.

The nurse must be familiar with the special types of patients usually classified as "high Risk" Obstetrical patients, because their fetuses are less able to tolerate the stresses of even normal labor progression. There are generally nine or ten conditions that fall into the high-risk category. They are:

1. The teen-aged or older and highly parous mother
2. The patient with toxemia, chronic renal disease, or hypertension
3. The pregnant diabetic
4. The patient with third trimester bleeding
5. The pregnant cardiac
6. The Rh-sensitized patient
7. The patient with multiple fetuses
8. The patient with breech or other malpresentation
9. The patient in premature or post mature labor

With our present limited methods of fetal assessment it is not possible to anticipate fetal accidents - knotting of the cord, etc. These cannot be seen until after delivery. But since these are rare occurrences, clinical assessments of the fetus can contribute greatly to lowering mortality and morbidity rates.

There are generally three areas of assessment that are significant.

1. Marked change in fetal activity - usually hyperactivity
2. Passage of meconium stained amniotic fluid
3. Significant deviations in rate and rhythm of fetal heart tones
   a. Bradycardia - slow heart beat, often identified as below 100/min. ~~or less, but sometimes below 120/min.~~
   b. Tachycardia - rapid heart beat, above 160/min.
   c. Irregularity or fetal arrhythmia - described as a disturbance in beat to beat rhythm.

In recent years quantitative and qualitative evaluation of fetal heart beats have begun to increase the information about fetal heart activity during pregnancy and during normal and abnormal labors. Electronic monitoring of the fetal heart tones combined with intrauterine

pressure assessments have evolved into several definite types of fetal heart variances. Hon and his associates have defined three of these patterns as "early deceleration," "late deceleration," and "variable deceleration", or Type I, Type II, and Type III dips. By close observation of these patterns and careful Apgar scoring of the infant after delivery, several conclusions were reached. The "early deceleration" pattern presented as a slowing of the fetal heart at or near the beginning of a contraction, with fairly rapid recovery to the normal baseline occurring, and back to normal by the end of the contraction. Babies who exhibited this type of variance were scoring about 7 on Apgar at 1 minute and 9-10 at five minutes. Therefore, Hon feels that this pattern is due to the fetal head compression during a contraction and is probably innocuous. The second pattern or the late deceleration Type II dip presents as a slowing of the heart rate beginning late in the contraction and persisting for several seconds after the contraction ends, with a slower rate of recovery back to the baseline. These, Hon feels, are due to a disturbance of the uteroplacental blood supply, and he considers them a definite sign of impending trouble. The third (variable) pattern shows no definite coordination with the contraction patterns, and these he interprets as cord compression patterns - somewhere in utero the umbilical cord vessels are being compressed enough to cause variations in the rate.

Most Obstetrical texts vary greatly in recommendations as to the frequency for monitoring maternal vital signs and fetal heart tones. A standard for this practice is set down in the "Manual of Standards in Obstetric-Gynecologic Practice", and is an excellent routine to be followed for every laboring woman.

will usually appreciate an occasional comment such as "steady, clear, and strong" from the nurse.

The fetal heart should be auscultated with periodic frequency, and at many different points in the labor contraction pattern. Listen between contractions when the uterus is relaxed. Listen through the beginning of a contraction, and with the mother's cooperation, through the entire contraction. Pay close attention to drops in the rate, and correlate the time of their onset with the contraction pattern. Be particularly aware of the ominous Type II dip - one in which the rate begins to slow toward the end of the contraction and has a slow recovery rate so that it is not back to normal for about 30 seconds or more after the contraction ends. Dr. Hon and associates have several recommendations of measures to be instituted:*

1. Alter the position of the patient. Repositioning represents an attempt to redistribute the mechanical forces of a contraction in such a manner as to relieve cord compression if it is present. The majority of Type III (Variable) deceleration patterns can be alleviated by repositioning the patient.

2. Administer oxygen to the mother at 6 - 7 liters per minute with a tight face mask.

3. Correct factors that may lead to decreased intervillous space blood flow: lower the frequency and amplitude of contractions if oxytocics are being used for induction of labor; correct maternal hypotension by position change, elevation of patient's legs or administration of IV fluids.

4. Prepare for operative interference.

5. If ominous fetal heart rate patterns still persist for 30

minutes after above measures, labor should be terminated, operatively, if vaginal delivery is not immediately possible.

Although there is nothing absolute about the above set of rules for the management of fetal distress, Dr. Hon feels that their establishment in his hospital has lowered both the operative interference rate and the number of depressed babies in the Obstetrical service. At the University of Iowa we have not seen any significant drop in the number of Cesarean Sections, but we have felt that we have reduced our neonatal morbidity and mortality due to fetal distress by early recognition and by prompt delivery.

In conclusion, the Obstetrical nurse must accept the challenge that the care of mother and fetus present to her with alertness, thorough understanding, and prompt attention to unusual signs. It can be one of the highest rewarding experiences to know that her skill and knowledge helped to bring both of her patients through to a successful conclusion.

Norma Ferguson, R.N.

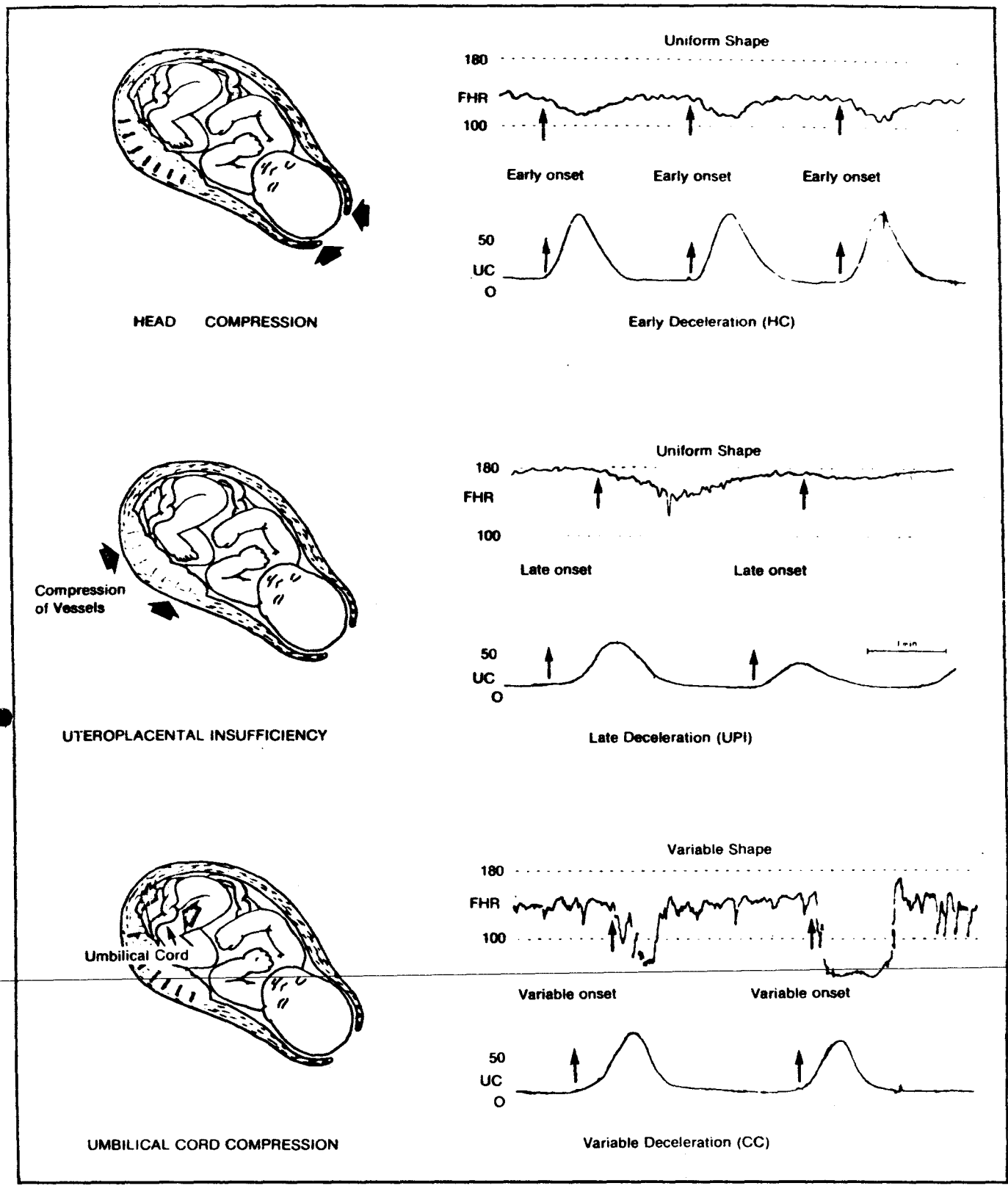


Fig 3.—Diagrammatic representation of proposed pathogenetic mechanism of FHR deceleration pattern. In head compression pattern (HC), onset of deceleration (arrows) *coincides with rise in intrauterine pressure* (arrows). *Uniform shape of deceleration reflects shape of associated uterine pressure curve. Uteroplacental insufficiency pattern (UPI) is characterized by uniform shape and onset late in contraction. Umbilical cord compression pattern (CC) is of variable shape and does not reflect shape of associated intrauterine pressure curve; its onset is variable in relationship to onset of contraction (from Hon[5]).*

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ANESTHESIA FOR DELIVERIES

ANESTHESIA FOR DELIVERIES

Few obstetrical units have the luxury of trained anesthesia personnel being present for normal deliveries. Therefore, it is most important that the delivery be accomplished using anesthesia that the attending physician and nurses are adept in administering and monitoring. Pudendal block or local perineal infiltration are usually excellent choices.[1] If the patient receives a regional block (saddle, caudal, etc.) it is most important that maternal vital signs be monitored closely during and following the procedure.[2]

The use of Trilene or Penthrane inhalations as analgesia for the mother during delivery is a common practice in many obstetric units. Two safeguards are recommended: the inhalor should never be set above the 4th mark on the dial, and it should be controlled by the patient herself.[3]

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ACOG TECHNICAL BULLETIN

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OBSTETRIC ANALGESIA AND ANESTHESIA

Pain relief during labor and delivery is an important aspect of modern obstetrics. It consists of more than providing personal comfort to the mother; it is a necessary part of good obstetrical practice. Thoughtfully chosen analgesia can improve labor and proper anesthesia permits difficult deliveries to be accomplished with safety. Poorly chosen analgesia may compromise labor and depress the fetus while improperly chosen and/or administered anesthesia may cause maternal or fetal morbidity and mortality.

Obstetric anesthesia must be considered as emergency anesthesia demanding a competence of personnel and availability of equipment similar to or greater than that required for elective procedures.[1]

It is recognized that the choice of analgesic and anesthetic techniques often is influenced by the experience and training of the anesthetist, by the personal preferences of obstetricians and patients, and by the circumstances of labor and delivery. Safety of pain relief depends on the careful choice of the analgesic agent, and the proper selection and skillful administration of the anesthetic. The obstetrician in consultation with the anesthetist should plan the type of anesthesia and agent for delivery during the course of labor. Both must be aware that there is a preferred method of anesthesia depending on the circumstances in each case and be prepared to accommodate to the situation at the time of delivery if it calls for a different type of anesthesia.

The obstetric patient should be anesthetized only when a qualified physician is immediately available to supervise the delivery and to deal with any complication that might arise. Under no circumstances should attempts be made to delay the imminent birth of a child by anesthesia, or by any other means, simply because a particular physician is not attendance. If the patient's physician is not there, a qualified professional alternate should be called upon to conduct the delivery.

A patient should not be left unattended while she is on the delivery table, either before, during or after the delivery. Similarly, she must be observed in an appropriate recovery area until she has reacted from the anesthetic and her vital signs are stable. The administration of general analgesia or anesthesia implies frequent monitoring of the patient's vital signs. Similar observation during regional block anesthesia is equally implicit so that adverse responses may be recognized promptly. Each patient should have vital signs monitored by one appropriately trained person who at that time has no other responsibility.

Fundamental tenets of safe analgesic and anesthetic practices are outlined below. These must be kept in mind by all who are responsible for their use in labor and delivery.

Pain Relief During Labor

There is no adequate, safe, routine analgesic agent universally applicable to all parturients. Patients differ in their response to labor and to medication. The need for sedation and analgesia must be evaluated and the selection of drugs individualized. Routine orders for medication are to be condemned. No analgesic or amnesic drugs should be ordered unless the progress of labor has been carefully evaluated by the responsible physician or qualified professional alternate.

All drugs used for pain relief in labor cross the placenta and most of them cause some degree of depression of the fetus. The degree of depression is directly related to the dose of the drug, the route and time of its administration before delivery, and the status of the fetus. Analgesic and amnesic drugs also may have adverse effects on the mother. All patients under analgesia must have careful supervision. Contractions should be evaluated and frequent determinations made of blood pressure, pulse, and fetal heart rate.

Barbiturates allay apprehension but do not relieve pain, except in overdosage. Their use should be limited to mothers with undue apprehension and only given early in labor. There is no known effective antidote to barbiturate depression in the infant except artificial respiration. Barbiturates should not be substituted for true analgesic drugs when these are required. The pain threshold is variable and each patient should be evaluated individually. Morphine and synthetic narcotics such as meperidine (Demerol) and alphaprodine (Nisentil) are examples of analgesic drugs useful in the first stage of labor. It is preferable to use smaller doses intravenously and to repeat them if necessary.

Tranquilizers have been found to be effective during the first stage of labor. They may be used alone or in combination with reduced doses of narcotic agents. Some tranquilizers potentiate the hypotensive effect of conduction block anesthesia and this must be considered carefully in their use.

Scopolamine and atropine are not analgesics but are sometimes used to inhibit secretions. Small doses have no known effect on the infant. Scopolamine, but not atropine, is a cerebral depressant and amnesic agent and, in some patients, causes excitement and hyperkinesis. Patients who react in this way should be carefully protected from injuring themselves or attendant personnel.

Certain anesthetic gases are useful for analgesia; that is, inhalation of low concentrations of these anesthetic gases is sufficient to provide significant pain relief while maintaining the patient in a conscious state (general analgesia). Since the concentration of anesthetic gases necessary to maintain analgesic levels varies among patients, parturients must be carefully observed to avoid levels of anesthesia which may cause excitability or obtund pharyngeal and laryngeal reflexes. Therefore, the technique should be used only with appropriate apparatus and under surveillance by trained personnel.

Paracervical block anesthesia can effectively reduce the pain of uterine contractions. Personnel using this technique should be familiar with the anatomy and the safeguards which need to be observed. The use of a needle guide is strongly urged in order to limit needle penetration which should not exceed 6 mm. Fetal bradycardia occurs in a significant proportion of cases and appears to be dose related. The maximum dose of lidocaine or mepivicaine is 200 mg, that is, 10 ml of a 1% solution injected bilaterally over a 90 minute period. However, lower doses such as 5 ml of a 0.5% solution injected bilaterally are usually adequate. The recommended dose of bupivicaine is 5 to 10 ml of a 0.25% solution injected bilaterally over a 90 minute period.

Analgesic and sedative drugs should be avoided or kept to a minimum during premature labor because of their depressant effects on the baby. Similarly, paracervical block should be used with caution and in reduced doses since the frequency of fetal bradycardia increases significantly. Pain can be reduced by the use of caudal or lumbar peridural anesthesia if facilities and experienced personnel are available for their administration. The use of oxygen by mask may be useful during the latter part of labor, or in the event of changes in the fetal heart rate.

Pain Relief During Delivery

Anesthetic methods, aside from hypnoanesthesia, fall into two groups: those which produce anesthesia locally or regionally, and those which relieve pain by general depression of the nervous system. In the first group are local infiltration of the perineum and block of the pudendal nerves. Other regional techniques, often called conduction block, include low spinal (saddle block), caudal and lumbar epidural block.

General anesthetic techniques include inhalation and intravenous anesthesia. The most frequent and the most dangerous maternal complication of general anesthesia is from aspiration of vomitus which may be food or acid gastric juice. No obstetric patient's stomach can be considered to be empty. *If the patient has eaten solid foods or milk-containing fluids within six hours prior to the onset of regular uterine contractions, or if labor has been prolonged, elective general anesthesia should not be administered except by endotracheal intubation.* Local, regional or conduction block anesthesia is recommended under these circumstances.

In conjunction with local or pudendal block anesthesia, subanesthetic inhalation analgesia, given so as not to obtund pharyngeal and laryngeal reflexes, affords satisfactory pain relief for most uncomplicated vaginal deliveries. This technique is becoming increasingly popular. If, however, general anesthesia is medically indicated for delivery, a cuffed endotracheal tube should be inserted. Such anesthesia should be administered only by those skilled and experienced with the technique. When maternal protective reflexes are obtunded, pressure upon the uterine fundus should be avoided. This maneuver frequently evacuates liquid gastric contents into the oropharynx. *Because of the hazard of aspiration of gastric contents with general anes-*

thesia, every obstetric patient should be warned during the last trimester of pregnancy that when she suspects labor may be starting, not to eat or drink prior to evaluation by her physician.

A hemoglobin or hematocrit determination and a urinalysis are essential for each patient admitted in labor. Except in unusual circumstances, the hemoglobin or hematocrit and urine sugar and protein should be known prior to administration of anestheia for delivery.

The choice between general anesthesia and local, regional or conduction block depends upon many factors, such as the condition of the patient and/or fetus, the length of time since the patient has eaten, the duration of labor, the presence of obstetrical or other complications, and the availability of personnel to administer proficiently general or conduction block anesthesia. If conditions are not favorable for general or major conduction block anesthesia, a more limited nerve block or local anesthesia should be used. Regardless of the type of anesthesia given for delivery, the patient should be carefully monitored by personnel trained in the prompt recognition of complications and skilled in their management.

Personnel for Obstetric Anesthesia

Safety of obstetric anesthesia depends principally upon the skill of the anesthetist. *The same level of competence of anesthesia personnel should be required for obstetric procedures as for surgical procedures.*

Responsibility for obstetric anesthesia may rest with the Chairman of the Department of Anesthesia or he may share it jointly with the Chairman of the Department of Obstetrics and Gynecology. If the hospital has no Department of Anesthesia, the responsibility may be given to the Chairman of the Department of Obstetrics and Gynecology or the Chief of Staff. In any event, obstetric anesthesia should be directed by a responsible member of the medical staff of the hospital and proper guidelines established for approving those who are to administer anesthesia for delivery.

Obstetricians, house officers, nurse-anesthetists, and others, excepting experienced anesthesiologists, seldom are fully competent in all forms of inhalation, intravenous, local, regional and conduction block anesthesia. Those approved for the administration of obstetric anesthesia should be approved only for those agents and techniques in which they have demonstrated competence.

The Delivery Room

Basic requirements include protection against anesthetic explosions, adequate lighting with an auxiliary electrical system, air conditioning, and all the factors necessary to promote cleanliness and asepsis. At least one delivery room should be equipped with an operating table and instruments for the performance of cesarean sections unless a nearby operating room can always be made immediately available.

Prior to each delivery, a specifically designated person should inspect the delivery room to assure that all necessary supplies, anesthesia and resuscitation equipment, cribs and incubators, and emergency facilities are present and in working condition. The maintenance and storage of supplies and equipment also should be the responsibility of a specifically designated person.

The following equipment and supplies should be available in each delivery room:

1. A delivery table which can be easily and rapidly adjusted from the head of the table to place the patient in the Trendelenburg position.

2. An anesthesia machine equipped with safety mechanisms to prevent the delivery of inadequate amounts of oxygen. Every anesthesia machine should be equipped with special yokes and pins so that bottles of a specific gas will fit only that particular yoke.

3. Explosive agents stored and administered in accordance with the requirements of the fire underwriters. A copy of these regulations should be available in the delivery section of every hospital.

4. A table of sufficient size for receiving the infant and for resuscitation. It is quite important to provide an overhead source of radiant heat in order to minimize infant heat loss by evaporation. The surface of the table should be covered with a blanket to eliminate heat loss by conduction.

5. A preheated crib or incubator which provides easy access to the infant and minimizes the loss of body heat. An open table, properly warmed, is preferable for this purpose. Small babies, and those who are distressed, should be transferred to the nursery in a transport incubator with its own battery operated heat source and portable oxygen supply. If an infant must unavoidably remain in the delivery room for any length of time, and assuming there is no distress, the heated incubator is acceptable.

6. Suction devices for both mother and infant.

7. Oxygen supplies for both mother and infant. Each should have a flowmeter and a pressure control. For both term and premature infants there should be face masks which can be attached to an adequate reservoir bag and an appropriate escape valve.

8. Resuscitation equipment should include oropharyngeal airways, endotracheal tubes, aspiration catheters, and suitable laryngoscopes for the use of those responsible for maternal and infant resuscitation.

9. Prepackaged disposable equipment, which offers good assurance of preventing cross infection, is strongly urged whenever local or regional anesthesia is to be administered.

10. Delivery packs, emergency instruments, sterile solutions, and medications as may be required.

11. A warm blanket and sterile pack for the infant, as well as the necessary material according to local requirements for the care of the infant's eyes and umbilical cord, and the approved means of infant identification.

General Anesthesia

While general anesthesia used for vaginal delivery and for cesarean section differs importantly from that used for surgical procedures on the nonpregnant patient, specific details regarding agents, doses, concentrations, flow rates, etc., are intentionally omitted. The anesthetist should be familiar with all variations of general anesthesia needed for both vaginal and abdominal delivery. *General anesthesia should not be administered by personnel who have not had formal training in its use.*

Halothane and ether produce outstanding uterine relaxation for intrauterine manipulations. With both agents, the relaxation outlasts the anesthesia; hence one should be prepared for possible postpartum hemorrhage.

Local Anesthesia

Perineal infiltration and pudendal block are the usual forms of nerve block anesthesia used for vaginal delivery. All personnel using these should be familiar with the anatomy involved and the safeguards which need to be observed. The dose of lidocaine or mepivicaine should not exceed 7 mg/kg and that of bupivicaine, 3 mg/kg. Usually 20 ml of a 1% lidocaine or mepivicaine solution or 20 ml of a 0.25-0.5% bupivicaine solution are adequate.

Spinal Anesthesia (Saddle Block)

Because of several advantages, spinal anesthesia has become a major form of anesthesia for delivery in many regions of the country:

1. Relative ease of administration, often administered by the obstetrician.

2. A fully conscious patient.

3. Maintenance of uterine tone.

4. Minimal risk of vomiting and aspiration.

Because of continued uterine tone, saddle block anesthesia may not be the technique of choice for:

1. Complete breech extraction (it can be used to advantage for assisted breech delivery).

2. Version and extraction.

3. The delivery of twins when version on the second twin may be necessary.

On the other hand, saddle block anesthesia is valuable for delivery of a patient who has had prolonged desultory labor, because the maintenance of uterine tone decreases the tendency to postpartum hemorrhage. This technique should be used with caution in shock, severe hemorrhage, hypovolemia, or chronic backache, and rarely in any neurological disorder or any systemic disease that may give rise to neurological manifestations.

Caution: the increased lordosis, the decreased size of the extradural space, and the greater lability of the vasomotor system increase the potential risk of spinal anesthesia in obstetric patients.

For greater safety the following safeguards should be observed:

1. Give spinal anesthesia only in the delivery room and observe and monitor the patient constantly.

2. Before administering spinal anesthesia,

   a. Take and record blood pressure, pulse and respirations on the Obstetrical Anesthesia Record form.

   b. Start intravenous fluids using an 18 or 19 gauge indwelling plastic catheter.

3. Do not inject spinal anesthesia during a contraction.

4. Within 30 to 45 seconds after injecting the spinal anesthetic agent depending upon the level of anesthesia desired, place the patient in the supine position.

5. Record blood pressure, pulse and respirations at frequent intervals following the spinal anesthesia—at least every 2 minutes for the first 10 minutes, then at least every 5 minutes for the next 20 minutes.

6. Check sensory level frequently during the first 20 minutes after giving spinal anesthesia.

7. If the systolic blood pressure falls below 100 mm, intravenous fluids should be run in rapidly, and the uterus should be displaced to the left off the vena cava. The patient's legs should be elevated with the trunk and head kept horizontal.

8. Ephedrine hydrochloride (12.5 mg) should be readily available for intravenous administration, if the above methods fail to correct hypotension. Small doses intravenously, repeated as needed, are safer than large doses.

9. Always have a bag and mask and oxygen available and functioning so that oxygen can be given by intermittent positive pressure should respiratory difficulty develop. In case of "total spinal" anesthesia, facilities for endotracheal intubation and maintenance of adequate ventilation for a prolonged period must be available.

10. Observe careful aseptic technique in giving spinal anesthesia. *Be certain that the drugs used have been autoclaved, not cold sterilized. Use individual spinal ampules; do not use stock multidose solutions.*

11. Oxytocic drugs (Pitocin, Syntocinon, ergonovine, etc.) where indicated in the post-partum period should be administered either intramuscularly or added to the intravenous solution and given as a dilute drip to avoid the hypertensive or hypotensive effects they may have when given by intravenous *push*.

The dose of spinal drugs for the obstetric patient is smaller than for her surgical counterpart and it should be tailored to the height of the patient (the length of her spinal column). The following are conservative doses for the drugs commonly used for spinal anesthesia in obstetric patients:

1. For Vaginal Delivery:
   a. Procaine hydrochloride (Novocaine)—30 to 50 mg with or without dextrose.
   b. Tetracaine hydrochloride (Pontocaine)—3 to 5 mg with dextrose.
   c. Dibucaine hydrochloride (Nupercaine)—2.5 to 3.75 mg with dextrose.
   d. Lidocaine hydrochloride (Xylocaine)—35 to 50 mg with dextrose.
2. For Cesarean Section:
   a. Procaine hydrochloride (Novocaine)—50 to 75 mg with or without dextrose.
   b. Tetracaine hydrochloride (Pontocaine)—5 to 10 mg with dextrose.
   c. Dibucaine hydrochloride (Nupercaine) 3.75 to 5.0 mg with dextrose.
   d. Lidocaine hydrochloride (Xylocaine)—50 to 75 mg with dextrose.

Caudal and Lumbar Peridural Anesthesia

These techniques incorporate all of the advantages of spinal anesthesia with the additional advantages of continuous relief of pain during the first stage of labor (catheter technique) and a diminished frequency of postspinal headache. However, the risk of high or total blockade is not obviated and there is the possibility of an untoward reaction to the anesthetic drug. They require constant supervision by trained personnel following the establishment of anesthesia. All of the contraindications to spinal anesthesia as well as the safeguards described above apply to these techniques. Specific details of the techniques of administration, agents, doses, etc., are intentionally omitted.

Infant Resuscitation

Only comments regarding personnel will be discussed here. While the obstetrician is primarily responsible for the care of the parturient as well as the newborn during the immediate post-delivery period, it is often impossible for him to treat both patients simultaneously. Therefore, he frequently must delegate the responsibility for infant resuscitation to another person. That person should be the member of the delivery room team best qualified to perform this task. To insure continuous availability of such adequately trained and experienced personnel, each obstetric service should establish a formal ongoing program for the training of specifically assigned personnel in the theory and techniques of infant resuscitation.

Anesthetic Emergencies

Obstetric and anesthetic emergencies occur acutely, often without warning. An anesthetic emergency usually significantly influences the otherwise normal obstetrical status of the patient

and vice versa. Successful management of these complications depends upon an anticipation of their occurrence, prompt recognition and immediate therapy. Obstetric and anesthesia personnel must be aware of the predisposing causes, prevention, and specific therapy for the common anesthetic emergencies associated with obstetric anesthesia. These include:

1. Respiratory obstruction.
2. Aspiration of liquid or solid gastric contents.
3. Cardiac arrest.
4. Systemic hypotension from spinal, caudal or lumbar peridural anesthesia.
5. Respiratory paralysis from spinal anesthetics.
6. Reactions from overdosage of or acute sensitivity to local anesthetic drugs.

Common to all of these anesthetic emergencies as well as most obstetric emergencies is the need for intravenous therapy with drugs, fluids, or blood. Therefore, *following admission to the delivery suite, intravenous fluids should be started on all obstetric patients in whom anesthesia is anticipated, preferably through a large bore indwelling plastic catheter that can be firmly secured to minimize the possibility of infiltration.* This measure is also advantageous during prolonged labor when intravenous fluid therapy will minimize dehydration, acidosis and electrolyte imbalance.

Since acute emergencies usually require additional trained personnel and equipment, it is recommended that a method for summoning immediate assistance be established in every delivery suite. This might be similar to the "red alert" system used to summon teams and equipment for the management of cardiac arrest occurring in other areas of the hospital.

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This Technical Bulletin is prepared for the Committee on Technical Bulletins of The American College of Obstetricians and Gynecologists by the College's Committee on Obstetric Practice in cooperation with the American Society of Anesthesiologists and the American Association of Nurse Anesthetists. It describes methods and techniques of clinical practice that are currently acceptable and used by recognized authorities. However, it does not represent official policy or recommendations of The American College of Obstetricians and Gynecologists. Its publication should not be construed as excluding other acceptable methods of handling similar problems.

THE AMERICAN COLLEGE OF
OBSTETRICIANS AND GYNECOLOGISTS
One East Wacker Drive Chicago, Illinois 60601

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MEMORANDUM

RE: Subgluteal and retropsoal infections in obstetric practice

The following information is not intended to diminish enthusiasm for, or utilization of, the highly desirable techniques of paracervical or pudendal block anesthesia for obstetrical patients. However, it is appropriate that physicians utilizing these techniques, and maternity unit personnel where obstetrical patients have these types of anesthesia be made aware of recent information.

As described on pages 137-150 of the January, 1972 issue of Obstetrics and Gynecology (volume 39, number 1) by Hibbard, Snyder, and McVann and following three recent cases at the University Hospital in Iowa, we should like to make known the following: Postpartum infections, presumably originating in paracervical or paravaginal tissues, have spread either laterally to the hip joint capsule or cephalad to the retropsoal space. The clinical picture of retropsoal or subgluteal infection is bizarre and the potential complications are profound. Generally, early signs of this infection include marked hip pain with inability to stand or walk. Physical examination usually demonstrates marked tenderness in the region of the hip joint with limitation of external rotation of that leg, pain on either straight leg raising or leg raising with knee flexion.

These infections are quite infrequent but there is a chance that bacteria might be introduced into paravaginal or paracervical tissues despite careful technique of local injection because of the impossibility to sterilize the vaginal canal completely.

We bring to your attention that retropsoal aud subgluteal abscesses can occur in the early puerperium, and that prompt diagnosis and proper antibiotic therapy are mandatory.

Prepared by:

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THE APGAR SCORE

THE APGAR SCORE

Evaluation of the newborn's condition in a thorough and consistant manner is accomplished by routine Apgar scoring of all infants at 1 minute and 5 minutes of life. The scores should always be recorded on the infant's chart.[1]

Conscientious assessment and totaling of the score rather than just "assigning a number" will give valuable information to all personnel. A low Apgar score will immediately point out the infant who will need closer or constant observation in the nursery, especially during the first few hours of life.[2,3] The Apgar scoring is a function that can be assumed by the nurse in the delivery room, and probably should be delegated to someone other than the physician who delivers the infant. At the critical times for the Apgar score to be determined the physician is usually primarily concerned with the third stage of labor in the mother.[2]

Some type of timing device should be utilized to mark the 1 and 5 minute intervals, such as a clock that rings a bell or buzzer at the proper time.

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1. Apgar, Virginia: Evaluation of the Newborn Infant,2nd report JAMA 168: 1985-88 Dec. 13, 1958

2. Apgar, Virginia: The Newborn (Apgar) Scoring System--Reflections and Advice Ped. Clinics of North America Vol 13 #3 Aug. 1966 645-650

3. Scanlon, John W.: How is the Baby? The Apgar Score Revisited Clin. Pediatrics, Vol.12 No. 2 Feb. 1973 61-63

APGAR SCORING SHEET

EVALUATION OF RESPIRATORY RESPONSE

Sixty seconds after the complete birth of the infant (disregarding the cord and placenta) the following five objective signs are evaluated and given a score of 0, 1, or 2. A score of 10 indicates an infant in the best possible condition. Infants with scores of 5 to 10 usually need little treatment. A score of 4 or below indicates the need for prompt treatment and diagnosis. Approximately 90% of normal infants score 7 or more one minute after birth. Another evaluation should be done at 5 minutes after the delivery.

SIGN	0	1	2	Score
Heart Rate	absent	slow (below 100)	over 100	
Respiratory effort	absent	slow irregular	good crying	
Muscle Tone	limp	some flexion of extremities	active motion	
Reflex irritability (response to flicking sole of foot)	no response	grimace	cry, startle	
Color	blue, pale	body pink, extremities blue	completely pink	

Totals: 1 minute_____ 5 minutes_____

Patient_____Date_____

Physician_____Nurse_____

Hospital #_____

The Newborn (Apgar) Scoring System

Reflections and Advice

VIRGINIA APGAR, M.D.

(from Pediatric Clinics of North America
13: 645, 1966)

Almost two decades ago the need was felt for a way to judge the condition of a newborn baby quickly and accurately shortly after birth. In 1949 the criteria in use were "breathing time" and "crying time." As a result of the frequent use of heavy medication during labor and of general anesthesia for delivery, an initial gasp, a period of apnea and finally established respiration were common. Should "breathing time" be recorded as the first gasp, or respiration which followed the period of apnea? Also, infants who had undergone more than usual birth asphyxia, or who had anomalies of the central nervous system, often did not cry at all. What, then, was the crying time?

Between 1949 and 1952 several signs easily observed in the newborn were considered. Five were chosen which could be evaluated without special equipment and could be taught to the delivery room personnel without difficulty.

The original intention of establishing a scoring system was to predict survival, to compare several methods of resuscitation which were in use at the time, and through the infant's responsiveness after delivery, to compare perinatal experience in different hospitals. The influence of various obstetrical practices such as induction of labor, elective cesarean section and maternal anesthesia and analgesia might well be reflected in the score. It was furthermore hoped that the scoring system would ensure closer observation of the infant during the first minute of life. The value of the system for neonatal research and for predicting neuromuscular deficit in early childhood was demonstrated later.

The system is working well in identifying infants who have severe metabolic imbalance. It is not working well as a baseline for future follow-up studies. There are two reasons for this inadequacy.

1. After receiving reports from many hospitals and visiting many personally, experience has demonstrated that the person delivering the infant should *not* be the one to assign the score. He or she is invariably emotionally involved with the outcome of the delivery and

Table 1. *The Collaborative Study on Cerebral Palsy, Mental Retardation and Other Neurologic and Sensory Disorders of Infancy and Childhood*

ONE-MINUTE APGAR SCORES
27,176 SINGLE BIRTHS

SCORE	TOTAL	1	2	3	4	5	6	7	8	9	10	11	12
0	0.1	0.1	0.2	0.3	0.1	0.1	0.1	0.1	0.1	0.4	0.1	0.0	0.0
1	1.8	2.3	0.9	0.8	2.2	2.1	1.6	0.9	1.2	1.8	1.8	4.6	0.6
2	2.0	2.2	1.7	2.4	3.2	1.8	2.0	1.6	1.4	2.1	2.1	2.1	1.0
3	2.2	2.8	2.5	1.9	2.4	1.8	1.6	1.0	2.3	2.1	2.3	2.3	1.6
4	2.8	3.2	3.4	1.8	3.6	2.1	2.8	1.9	2.7	3.3	2.7	3.5	1.8
5	4.5	5.0	4.3	3.9	7.4	4.6	5.1	4.5	3.7	5.4	3.6	7.0	2.3
6	6.5	7.1	6.3	8.3	8.9	6.2	5.6	6.7	6.1	8.3	5.3	7.0	4.2
7	8.7	8.6	15.8	5.1	16.7	8.1	5.5	10.9	7.1	12.3	8.9	7.4	4.6
8	20.8	27.2	34.9	6.8	33.9	16.9	10.4	27.3	14.8	20.8	21.3	12.8	11.2
9	40.5	40.0	28.6	46.2	18.5	34.9	45.8	40.9	41.5	35.1	47.8	46.9	41.6
10	8.2	1.3	1.1	21.8	2.9	4.0	19.4	0.6	19.2	3.8	4.1	6.5	31.1

FIVE-MINUTE APGAR SCORES
27,176 SINGLE BIRTHS

0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.1	0.0	0.0	0.0
1	0.6	0.5	0.5	0.2	0.0	0.9	0.6	0.4	0.5	1.4	0.7	1.7	0.4
2	0.5	0.4	0.2	0.9	0.1	0.3	0.6	0.5	0.2	1.0	0.7	1.2	0.2
3	0.6	0.6	0.5	0.6	0.5	0.9	0.5	0.2	0.2	0.5	0.7	1.0	0.4
4	0.6	0.5	0.2	0.7	0.5	1.0	0.8	0.3	0.8	0.5	0.8	0.7	0.5
5	0.9	0.7	0.6	0.9	2.2	1.5	0.5	0.4	0.8	1.4	0.9	2.5	0.8
6	1.7	1.3	1.4	2.6	2.7	2.4	1.1	0.9	1.6	2.8	1.8	3.0	1.4
7	2.5	2.8	2.8	2.0	3.6	3.2	1.4	3.0	1.7	4.2	2.5	3.2	0.9
8	8.3	10.1	11.2	2.3	20.7	9.7	2.2	8.7	3.8	8.5	9.7	2.7	2.8
9	54.3	76.8	68.6	41.4	39.7	61.9	17.5	74.4	13.1	61.0	51.5	56.6	34.2
10	20.7	6.3	14.1	48.4	29.6	17.7	74.7	10.5	77.2	18.1	30.8	27.3	58.4

with the family, and cannot or unconsciously does not make an accurate decision as to the total score. I know of no reliable study which compared scores given by various delivery room personnel, but my impression is strong that obstetricians give higher scores than anesthesiologists, nurse anesthetists, pediatricians or delivery room nurses. A number have claimed "never to have had a baby with a score less than 5." The data from Finland[1] in Figure 2 show an incidence of score 10 in 83 per cent of births! These scores were assigned by the midwife delivering the baby.

In 27,176 infants who are part of the Collaborative Study for Neuromuscular Deficit being conducted by the National Institute of Neurological Diseases and Blindness of the National Institutes of Health,[7] score 10 ranged from 0.6 to 31.1 per cent in 13 hospitals. In almost all cases specially trained personnel, not the obstetrician, decided on the score (Table 1).

It is ideal to have a specially trained observer, whether physician or nurse, but this happy situation is seldom practical. Until such time when a pediatrically oriented person is routinely present for all deliveries, the anesthesiologist or anesthetist is in a good position to assign the score, especially if the infant is placed in a bassinet near the head of the delivery table. With regional anesthesia he is entirely free to make the necessary observations, and with general anesthesia the mother should be nearly awake or at least light enough so that protective reflexes have returned.

More and more the ability and accuracy of observations of the circulating nurse are coming to be appreciated. She usually has considerably more experience with delivery room crises and discipline than an intern or resident and is often the only other person present in addition to the obstetrician. Her services should be encouraged. The only drawback is her multiplicity of duties at the time of delivery. This leads to the second point which needs clarification.

2. The time to assign the score was and still is 60 seconds after birth. This interval was chosen in 1952 after observing several hundred infants for the time of maximum clinical depression. Thirty-, 60-, 90- and 120-second observations were made with two observers, who agreed that 60 seconds after both the top of the head and bottoms of the feet were visible was the time to apply the score.[2] Serial measurements of the infant's arterial acid-base state during the first minutes of life show that maximal acidosis is present several minutes later than 60 seconds.[5] The importance of the one-minute score cannot be overestimated, however, from the point of view of assisted ventilation.

There should be some automatic way to announce the passage of 60 seconds. Only clinicians in anesthesia have learned to live by the second hand of a watch. To others, a minute is an unbelievably short interval. On some occasions, such as cardiac arrest, it is a very long interval. A simple automatic timer should be firmly fixed on the wall, set for 60 seconds. Fifty-five seconds would be preferable, for observation of the five signs should take no longer than five seconds. A one-minute timer is less expensive, however, and more available (e.g. from General Electric Company).

When the head and feet of the infant are both visible, the timer is started and can be forgotten until the alarm sounds, whereupon the score is assigned. The circulating nurse can add this brief chore to her list of duties without difficulty. This method has worked well in several hospitals.

One misconception has arisen which is easy to correct. In some clinics no active resuscitation is done until after the score has been assigned at one minute. This is, of course, wrong. It has been demonstrated that pH can drop from 7.4 to 6.9 in less than five minutes.[5] Time is of great importance in preventing or reversing such a sharp change. With infants known to be suffering from unusual asphyxia due to such complications as prolapse of the cord, impacted shoulders or a large breech presentation, resuscitation should begin just as soon as a free airway has been assured. Saling's[8] intrapartum method of sampling scalp blood should aid greatly in identifying such babies. The first score is decided at 60 seconds, even though resuscitation has been in progress.

The score at one minute does not reflect oxygenation itself.[6, 9] It is rather a reflection of acid-base status. Obviously, samples should be drawn from the umbilical artery rather than the vein, to judge the con-

dition of the infant. Umbilical venous blood reflects the state of placental exchange, not the infant's arterial blood.

Recently it has been demonstrated that the five-minute score is more predictive of survival than the one-minute score.[4] This is not surprising, for it is to be expected that the longer asphyxia exists, the more likely that death or permanent damage will occur. A comparison of infants with one- and five-minute readings with respect to their neurologic abnormalities at one year of age also shows significantly more predictive value of the five-minute score.[7] Nevertheless, if the first observation is made as late as five minutes, an appreciable number of infants will have their asphyxia untreated because of lack of an earlier observation, with subsequent higher mortality and morbidity. A study of those infants with low five-minute scores in relation to what treatment, if any, was administered would be of interest.

The distribution of scores from 12 institutions in the Collaborative Project at five minutes (Table 1) again indicates extensive differences between the various centers. These might be a reflection of differences in obstetric practice or in maternal analgesia or anesthesia among institutions; it could also be due to variations in the application of the scoring system. In comparing the details of score distribution and neonatal deaths in three institutions, Sloane Hospital for Women, University Hospital at Puerto Rico and the University Hospital at Helsinki, considerable differences are again demonstrated (Figs. 1, 2). Even within one institution there have been changes over several years (Fig. 3). Yet when the score distributions are grouped 0 to 3, 4 to 6, 7 to 10, the differences become negligible (Fig. 3, *lower*).

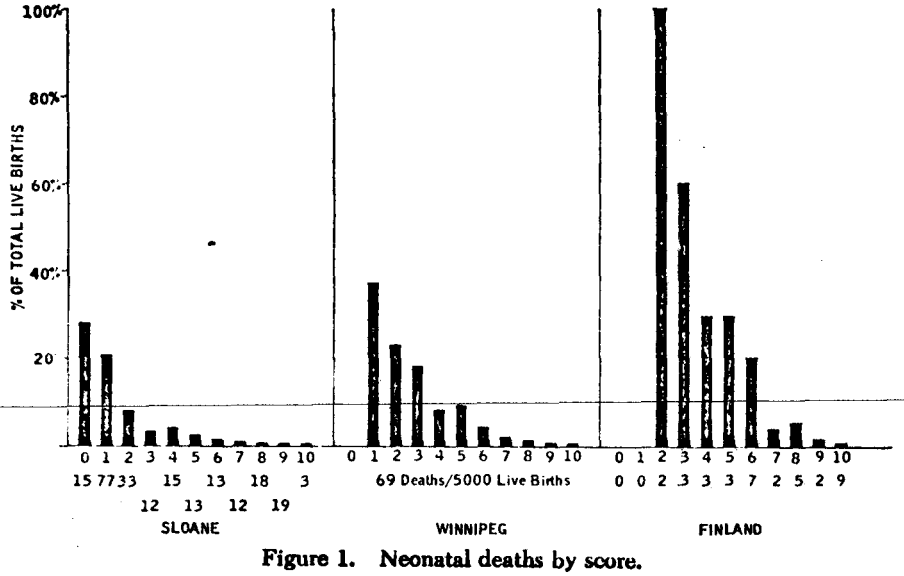

Figure 1. Neonatal deaths by score.

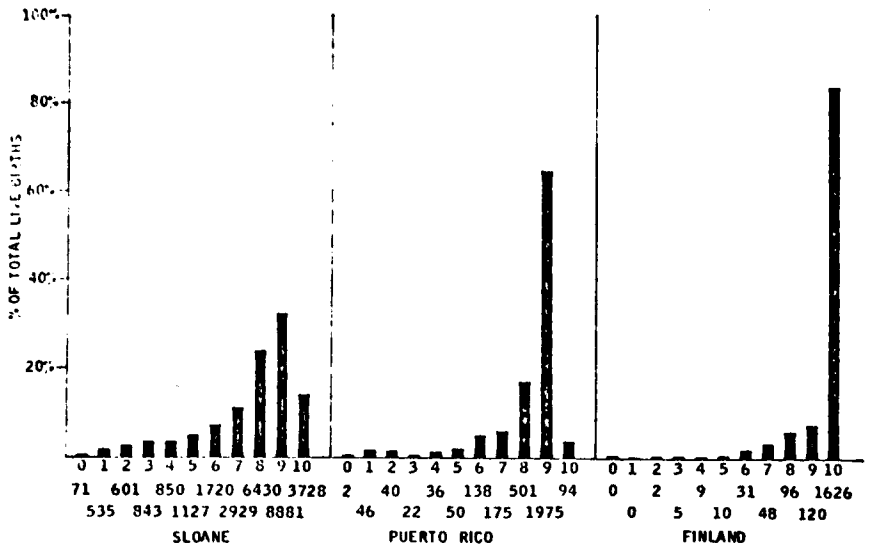

Figure 2. Distribution of scores, all births.

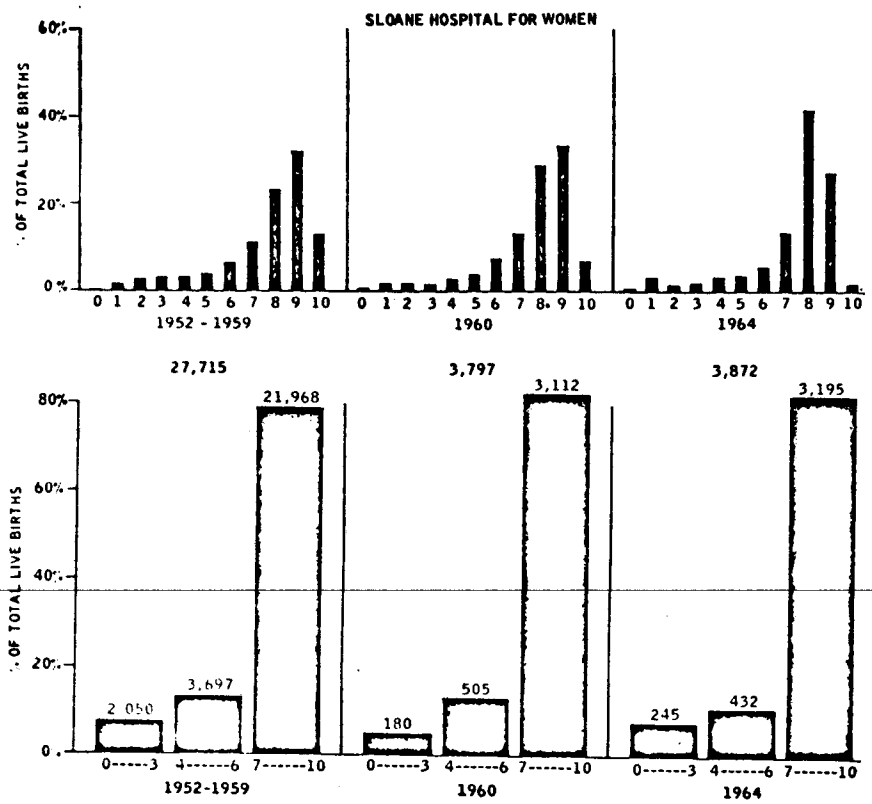

Figure 3. Variation in scores in the same hospital.

These differences might indeed be real. Nevertheless, unless interpretation of the clinical signs and the time at which the score is given are standard for all institutions, conclusions will not be valid. This becomes even more important when the scoring system is being used for correlations with subsequent neurologic development.

SUMMARY

It is strongly advised that an observer, other than the person who delivers the infant, be the one to assign the score.

An automatic method of announcing the passing of 60 seconds is recommended.

Although mortality and the presence of significant neurologic damage correlate better with the five-minute than the one-minute score, the one-minute score should nevertheless be retained. It is essential to observe the infant from the moment of birth in order that prompt treatment can be given if necessary. Nine months' observation of the mother surely warrants one-minute observation of the baby.

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How Is the Baby?: The Apgar Score Revisited

JOHN W. SCANLON, M.D.*

"Learn of the Muse, and may thy Pains succeed,
Don't, 'till 'tis born, defer thy Pious care;
Begin betime, and for its Birth prepare."
Paedotrophia, Scevole De St. Marthe, 1579

THIS year, 1972, marks the 20th anniversary of Virginia Apgar's presentation to the 27th Annual Congress of Anesthetists of a new method for newborn evaluation. Before then, the usual approaches to evaluating an infant's condition at birth were the observations of a competent pediatrician, and noting the time interval from birth to the first breath or to the first cry.

Historically, descriptions of newborns dealt mostly with the presence of severe malformation or with serious morbid events, as indicated by the following quotation attributed to William Heberden in 1807: "It sometimes happens, that immediately after birth the face and neck put on a black or livid appearance, the lips become purple, and the breathing short. These symptoms, if they do not soon go off, usually terminate in a speedy death." Care in the delivery area was given over to cultural or religious dicta extolling the benefits of swaddling, bathing, cord care and hygiene, as in the following quotation, again from St. Marthe's *Paedotrophia*:

"Then the kind nurse, with tender fingers clears
His mouth from filth and even his eyes and ears."

Virginia Apgar's scoring system was promptly accepted and has become the most universally used system for describing a newborn's condition at birth and for measuring the impact of perinatal events on that newborn. This system not only compel's physicians to quantitate those neonatal functions necessary to sustain life, but by so doing makes them direct much more of their attention to the newborn. Another important consequence of the routine use of Apgar scores has been to provide a framework of familiarity upon which to establish the need for, and to evaluate the results of, resuscitative measures.

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Dr. Apgar's purpose in designing her score was to assess the status and follow the progress of the acutely asphyxiated and severely depressed newborn. Time has brought consistent confirmatory documentation, clinically and physiologically, of the accuracy of her design.[2, 3] As a consequence, therefore, the Apgar score has been and is still being widely applied as the sole yardstick for measuring a great number of perinatal factors which might affect the newborn, such as maternally administered drugs, obstetric maneuvers, and abnormal conditions in the mother.

The five variables scored (heart rate, color, tone, respiratory effort, irritability) are essentially vital signs. Yet few pediatricians would be content to judge an infant's status *only* from this basic information. The Apgar will miss subtle changes from intrauterine events, since an effect must be considerable to significantly lower the number. Furthermore, limiting the scoring period to the first 10 or 15 minutes after birth neglects a most critical time of life. Perinatal mortality figures indicate that it is the first 24 postnatal hours which are the most hazardous for any human being. In addition, adaptation to extrauterine existence must be established during this time. As Dr. Apgar herself said, "The Apgar score is no substitute for a careful physical examination and careful observations over the first few hours of life, nor will it predict neonatal death or survival in individual infants."[2] With infants of drug-addicted mothers, experience has demonstrated that life-threatening perinatal influences may not become manifest for hours or days after birth.[4] The Apgar score misses all delayed effects from intrauterine events.

The Apgar score has also been utilized as a predictive index for future neurologic normality. It has real importance in this regard as an initial, crude screening tool. However, when data from the Collaborative Study[5, 6] are critically evaluated, a limited predictive value for the Apgar score is apparent. Infants of low birth weight, or otherwise at risk, who have a low (0–5) five-minute score, do have a significantly decreased chance for neurologic normality at one year of age. Birth asphyxia and other neonatal morbidity are syn-

ergistic, each contributing to an unfortunate outcome. Similarly, the incidence of neurologic deficits is fourfold greater in full-term normal birth weight infants with low five-minute Apgars than in similar full-term "normal Apgar" infants weighing 2.5 kg or more. This last observation has been the basis for general pessimism regarding the outcome of all infants with low Apgar scores. From actual data, however, only 4.3 per cent of these term, low five-minute Apgar score babies will be abnormal at one year of age; *95.7 per cent will not.*

The prediction of more subtle effects on outcome in the higher range of the Apgar score is suggested by Lewis *et al.*[7] These investigators studied visual attentiveness in 40 babies at three, nine, and 12 months of age. Twenty of these infants had five-minute Apgars of 10 and the other 20 had scores ranging from 7 to 9. The capacity of the "perfect 10" Apgar baby's response to complex visual stimuli was consistently and significantly greater than this same ability in infants who had scored between 7 and 9. These data are not to be interpreted that the Apgar score is a sensitive or precise measure of behavioral capacity. Borgstedt and Rosen[8] were not able to demonstrate a correlation between Apgar scores and electroencephalographic or behavioral changes caused by maternal obstetric sedation. Rather, Lewis's data support the concept that an Apgar score is exceedingly subjective. Most physicians will agree on a score of 10, that is, a perfectly pink, crying, active baby. But when a baby scores less than 10, unrecognized biases of an "interested" observer might raise some marginal score up to the 7–9 range, adding minimally depressed babies to Lewis's lower Apgar group.

This subjectivity of the Apgar score is at once an advantage and a handicap. Certainly one reason why the Apgar has become the standard for newborn evaluation is that it is simple, rapid, and requires no special equipment or extensive training to implement. Yet Dr. Apgar herself initially, and repeatedly, warned that to be valid, her score must *not* be performed by an observer who is involved in the care of either mother or infant.[1,9] Comparative data, collected by Dr. Apgar, document this scoring variability. During ten years of study, the median score from Sloane Hospital in New York City was 8, from University Hospital in Puerto Rico it was 9, and from University Hospital in Helsinki, it was 10.[9] We have witnessed, all to often, the awarding of a casual, "eyeball" Apgar after only a cursory glance at the newborn. This misinformed score does more harm than good.

As previously mentioned, the Apgar score has been extensively used in clinical perinatal pharmacology.[10] The pediatrician must indeed be wary of statements that a maternally administered drug is "safe" or "shows no untoward effects" for the newborn from data which are based on Apgar scores alone. First of all, there is no historical precedent for using Apgar scores to study drug effects in the newborn. The infants in Dr. Apgar's original report were almost all delivered following some form of anesthesia or analgesia.[1] This lack of nonmedicated control is, in Dr. Apgar's words, regrettable, because it provides no master control group for any subsequent study. Considerable data now indicate that many obstetric drugs, previously thought to have no activity in the newborn, actually do have considerable effects when evaluated by other standards.

In perinatal pharmacology, a number of more recent studies have documented the effects of prenatal sedation[11,12,13] or regional or general anesthesia[14] upon the newborn in dosages once regarded as "safe." These studies rely to a great extent on newly developed behavioral tests. In none of these was the degree of newborn behavioral impairment reflected by unusually lowered Apgar scores.

The use of behavioral technics for assessing the effects of intrauterine events on the newborn is not really new. In 1957, Graham *et al.*[15] reported significant behavioral differences within a group of 60 newborns whose obstetric histories suggested anoxia, when compared with the behavior of 62 historically nonasphyxiated controls. More recently, investigators have attempted to combine behavioral testing with neurologic and electrophysiologic standards in order to increase diagnostic and prognostic sensitivity in the newborn period. These testing procedures, while shedding much light on the complexities of neonatal neurologic adaptation, are themselves complicated and frequently require expensive and/or cumbersome equipment. These restrictions limit their general clinical applicability.

What can the clinician use with his patient to complement the observations begun with the Apgar score? First of all, the initial physical examination of the newborn provides irreplaceable information about transition from intrauterine to extrauterine life. The physical,[16] neurologic,[17] and ponderal[18] criteria of maturity correlated with the estimated gestational age can be intelligently interpreted in light of any given obstetric history. Since the neurologic responses indicative of major and minor residual CNS damage are those related to tone, muscular excitability, or the absence of reflexes,[19] attention to recording specific abnormalities is mandatory. Repeated observations at regular intervals will give even more substantive information. One hallmark of a normal newborn is his ability to vary many responses to environmental stimuli. Brain damaged newborns will often have disorganized, depressed, obligatory, or stereotyped activity. An infant's ability to change his level of arousal (state) when stimulated,[20] his

ability to modify his response to repetitive stimulation (response decrement),[21,22] and the rapidity with which he recovers from the initial "shock of birth"[23,24] are all indicators of normality.

Finally, newer observational technics have begun to quantitate the early mother-infant interaction.[25] The traditional view of this interaction has been that the mother is the sole activist. This may not be true. Because of his small size, apparent helplessness, and distressing cry, the newborn can trigger mothering activities.[26] Thus, anything which changes the newborn's behavior (such as minimal asphyxia, subtle drug effects, etc.) might alter his ability to elicit an appropriate maternal reaction. The pediatrician has an invaluable opportunity to observe this pair at first hand. As Brazelton[21] points out, regular and repeated observations of interactional situations in the newborn provide irreplaceable predictive information for further behavioral assessment.

What we are suggesting, really, is the use of the pediatrician's stock in trade: careful, close, intelligent observation of the patient. These observations, and their interpretation, will serve to complement the more acute but less sensitive measurements of the Apgar score.

In summary, the Apgar score has stood the test of time as an index of acute, severe neonatal impairment at birth. More sensitive clinical measures, plus an understanding of the Apgar's limitations, will enable the clinician to better answer the question posed by our title.

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COUNTING OF UMBILICAL CORD VESSELS

COUNTING OF UMBILICAL CORD VESSELS

We are all aware of the fact that normally there are three vessels in the umbilical cord. However, about 1% of infants can exhibit the congenital anomaly of the absence of one umbilical artery.[1-5] The fact that this one anomaly exists will alert physicians and nurses to the need for closer examination and observation of this baby to rule out other anomalies. Statistically, the three internal systems that may show hidden anomalies are the cardio-vascular, the G. I., and the G. U. systems. We recommend the routine examination of the umbilical cord as it is cut, and the recording of the number of vessels found on the infant's chart.

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The Single Umbilical Artery and What It Means

A Message Straight from the Navel

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The infant born with an SUA is a high-risk infant and should be treated accordingly immediately after delivery. The variety and multiplicity of congenital abnormalities associated with this finding call for a very thorough initial examination, continued close scrutiny throughout infancy and childhood, and prompt investigation of any developing symptoms or abnormalities. It is likely that the high death rate associated with the SUA could be lowered if steps are taken to uncover and correct such subtle abnormalities before they stir up difficulties.

THE Latin term, umbilicus, is the diminutive of the word, "umbo," or the protuberant part of a shield. Depending upon which side of the shield you view, this implies either a protrusion or a concavity. To avoid semantic and perceptual confusion, the term 'navel' is preferred for this anatomic area. The Anglo-Saxon "nafela" or "nafe," from which the term derived, originally meant the center or hub of a wheel to which the spokes were attached (Fig. 1).[1] The navel, though seemingly quite useless after birth, still has one function—as a messenger. Absence of one umbilical artery, like a spoke missing from a wheel, may suggest a defective product.

In 1870, Hyrtl,[2] when describing the blood vessels of the placenta in "normal and abnormal circumstances," noted the association of a missing umbilical artery with congenital abnormalities including "monster fetuses." The first American report of the association of a single umbilical artery (SUA) with congenital abnormalities appeared 85 years later.[3] Of the 55 newborns with SUA studied by Bourne and Benvischke, only 13 survived and appeared to be normally formed, though long-term follow-up of these 13 was not available. Of the 42 infants who were stillborn or did not survive, 64 per cent had congenital abnormalities; these abnormalities were usually multiple and involved many systems. Mothers of these infants tended to be older, and had a higher incidence of toxemia and hydramnios. In the series were five sets of twins in which one twin had an SUA.

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SUA and Congenital Abnormalities: A Review of Several Studies

Attention was drawn to the importance of the SUA by Gellis in the 1965–66 *Yearbook of Pediatrics*.[4] The lead article was on an analysis of 60 cases from 1,554 autopsies and 3,353 placental specimens which included first and early second-trimester aborted fetuses.[5] Of the 33 liveborn infants, 15 had died during the neonatal period and three others died within 21 weeks of birth. Over half (55%) were found to have congenital abnormalities which tended to be severe, multiple, and often of a lethal type. Almost all systems were involved, though cardiovascular malformations were the most frequent (Table 1). In each system, the abnormalities were diverse rather than specific. Multiple organ systems tended to be involved in each baby. The SUA was associated with a known trisomic syndrome in two

FIG. 1. The Navel—Center of the Body. The symbol of the American Academy of Pediatrics emphasizes the importance of the navel. Extensions of the lines or folds radiating out from the infant intersect at the navel—like the spokes of a wheel. The presence of a single umbilical artery in the navel may portend serious consequences for the infant. Examination of this central anatomic area should be routine.

patients and a suspect trisomic syndrome in nine patients. SUA was relatively high in monozygous twins.

Dr. Feingold,[4] at the end of the review of this article, commented that, because the patients had either died or had a suspected abnormality leading to placental examination, the 2.9 per cent incidence of SUA reported was selectively higher than the "acceptable figure of 1 per cent." The possibility of hidden defects in the 13 children who were normal at birth, but lost to follow-up, was raised. Dr. Feingold recommended routine examination of the cord in newborns and suggested that the pediatrician usurp this responsibility because, in his experience, the obstetrician was incorrect in reporting the number of vessels 75 per cent of the time!

The importance of looking for "hidden" urologic defects in association with SUA was substantiated by a study of 32 newborn infants with SUA who had no renal symptoms or findings on physical examination.[5] Intravenous pyelograms were performed on 24 of 29 survivors. In all but three, this procedure was performed during the first four months of life. Eight of the 24 (33%), had abnormal pyelograms; their genitourinary abnormalities were not uniform. A review of four other prospective studies indicated that the incidence of SUA may vary from 0.2 to 1 per cent and that congenital abnormalities may be associated in 20 to 40 per cent.

Cairns and McKee,[7] in another prospective study of 2,000 deliveries, found a 1 per cent incidence of SUA. Of the SUA babies, two (10%) had congenital malformations, whereas of the 1,980 babies with two umbilical arteries, 31 (1.5%) had congenital abnormalities.

TABLE 1. *Observed Congenital Defects in 33 Cases of SUA*[6]

Cardiovascular system	26
Genitourinary system	16
Gastrointestinal system	15
Neurological defects	9
Musculoskeletal system	21
Respiratory system	14
Others (facial, ear, eye anomalies)	15

The Wide Variation in the Incidence of SUA: Why?

How can one account for the wide variation in the incidence of congenital abnormalities in association with SUA? One explanation is that populations, observers, and observations differ. Feingold suggests that not all congenital abnormalities are detectable at birth. Thus, the total incidence of lethal and nonlethal defects noted in stillborn and liveborn infants beyond the 20th week of gestation varies from 1.4 to 2.9 per cent, but subsequent examinations during the first year of life raise the incidence to 7.5 per cent.[8] Indeed, in the most thorough studies, fewer than half of the malformations found among liveborn infants were suspected or noted at birth.[9] The way in which one *defines* a congenital abnormality may affect the incidence —what is "significant" to one observer may not be to another. Race seems to affect the occurrence of SUA, since, in the largest series reported,[10] its incidence was 1.2 per cent among 11,371 white infants but only 0.44 per cent among 13,058 Negro infants. Yet, although fewer Negro babies had an SUA, the incidence of associated congenital abnormalities was higher in these Negroes (42.1%) than in the whites (23%). In this study, congenital anomalies were classified according to degree —major or fatal, nonfatal and borderline. All organ systems except the endocrine system appeared to be involved (Table 2). The incidence of SUA was higher in infants of diabetic mothers and in infants whose birth weight was less than 2,500 g.

The need for a prolonged, planned intensive study was filled in a recent prospective study of 2,572 singletons. The investigators discovered 29 infants with an SUA.[11] Those

infants who survived the neonatal period were followed from five months to three and one-half years in a program which included intravenous pyelography, chromosomal analyses, and a study of dermatoglyphics. The perinatal mortality rate was 21 per cent. Of the four stillborns, two had congenital abnormalities. "Disabling" malformations were found in only two of 22 survivors; four had superficial strawberry nevi. Psychomotor development was normal at follow-up. Only one of the 19 children who had an intravenous pyelogram was found to have a urinary abnormality. All of the 19 karyotypes were normal. Dermatoglyphic investigation revealed an increased number of radial loops and lowered ridge count in boys. Palm creases were normal.

Horse Versus Cart: Why SUA and Congenital Abnormalities?

It is possible that the infant with an SUA has been "blighted" during gestation by one of the many factors known to cause congenital abnormalities and that the SUA is but one manifestation of this blight. Bourne and Benvischke[3] theorized that the absence of an umbilical artery may be associated with resultant resistance to blood flow in the one remaining, and that possible decreased oxygen supply to the fetus impeded development. In their series, the twin fetus with an SUA tended to be the smaller or the one dying *in utero*. The wide spectrum of abnormalities and the fact that the normal umbilical arteries are ordinarily well formed by the third to fourth embryonic week suggests an early yet prolonged period of teratogenic influence.

Vlietinck *et al.*[11] state that the association of SUA with thalidomide embryopathy and induced ovulation, the lack of history of consanguinity and only two instances of familial SUA, support a "teratogenic etiology."

Implications for the Practitioner: What Should One Do with an SUA Baby?

The SUA is a significant portent of the possibility of other congenital abnormalities. When a single palmar crease, another topical anatomic harbinger of malformation, is present, this is also associated with premature birth, stillbirths, and neonatal deaths; 15 per cent of survivors will have congenital abnormalities.[12]

The finding of an SUA is much more ominous. A survey of the literature indicates that of 100 SUA fetuses reaching term, 11 will be stillborn, and 17 will die by one year of age.

TABLE 2. *Details of Congenital Malformations Associated with SUA in 203 Infants*[10]

Malformation	Number
Skeletal System	27
Polydactyly	
Vertebral anomalies	
Talipes (clubfoot)	
Multiple bone anomalies	
Syndactyly	
Absence of one finger	
Hallux and genu valgus	
Bilateral cervical ribs	
Micrognathia	
Pectus excavatum, mild	
Gastrointestinal System	24
Inguinal hernia	
Omphalocele	
Cleft palate	
Tracheoesophageal fistula	
Duodenal atresia	
Pyloric stenosis	
Meckel's diverticulum	
Agenesis of rectum	
Imperforate anus	
Agenesis of gallbladder	
Agenesis of spleen	
Diaphragmatic hernia	
Genitourinary System	13
Hypospadias	
Polycystic kidney	
Renal aplasia	
Agenesis of urinary bladder and urethra	
Ectopic kidney	
Double ureter	
Persistent mullerian duct in male	
Cardiovascular System	8
Multiple anomalies of heart and/or great vessels	
Congenital heart disease	
Respiratory System	12
Hypoplasia of lung	
Choanal atresia	
Central Nervous System	6
Anencephaly	
Microcephaly	
Hydrocephaly	
Hypoplasia of optic and olfactory nerves	
Integumentary System	16
Nevus	
Cafe au lait spots	
Hemangioma	
Skin tabs of finger	
Pilonidal sinus	
Rudimentary nipples	

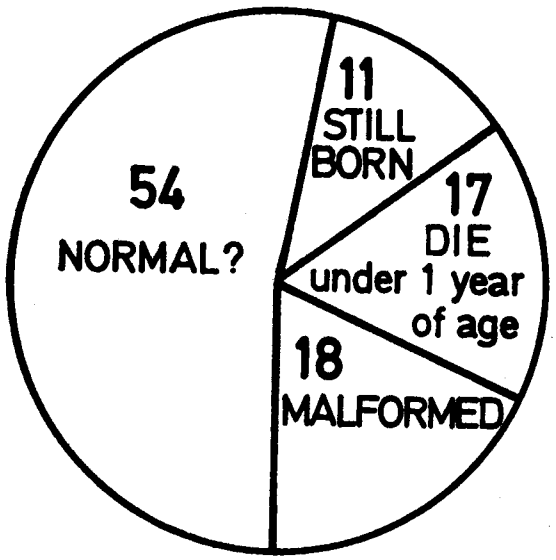

FIG. 2. Fate of 100 SUA babies who reach term. A child with an SUA at birth has a 1 in 2 chance of surviving and being normal.[11]

Of the 72 survivors, one-fourth will be malformed. Expressed differently, the child with an SUA at birth has a 1 in 2 chance of surviving and of being "normal" [11] (Fig. 2). It is not impossible that follow-up of SUA children through school age will uncover functional problems such as intellectual deficits as have been described in children with palmar crease abnormalities,[13] or even more subtle perceptual difficulties as have been found in treated phenylketonuria children with average intelligence.[14]

Because clamping of the cord and drying of the stump make counting the number of vessels difficult for the pediatrician, the obstetrician should routinely count and report on the number of cord vessels as part of his delivery room examination. Every delivery record should have a standard place to record this observation.

Steps To Be Taken When a Child with an SUA Is Discovered:

1. Because some of the abnormalities associated with an SUA can be life threatening immediately after delivery, *the pediatrician should be notified promptly* and the baby should be transferred as quickly as possible to the newborn ward, preferably to the observation or suspect nursery where the baby can be scrutinized and followed closely with other high-risk infants.
2. An exceptionally thorough examination should be performed as soon as the baby enters the nursery. During the examination, particular search should be made for abnormalities of the face and hands where, because of complex anatomy, subtle abnormalities are most likely to be found. Although not emphasized in the literature on SUA, the association of ear abnormalities with renal malformations is well known.[15, 16]
3. Simple additional diagnostic procedures, that are not necessarily performed routinely in other newborns, should be part of the examination of the infant with an SUA. Passing a feeding tube to the stomach through a nostril quickly rules out choanal atresia and tracheoesophageal fistula. The same catheter can then be used "at the other end" to guarantee that the anus is patent.
4. Vital signs should be monitored carefully for the first 24 hours. Placing the child on the "Silverman Wheel" is an efficient way to graphically chart respiratory functioning and more acceptable than raising a red flag above the crib.
5. Because of the frequent association of a diaphragmatic hernia with SUA, and the seriousness of this abnormality, any sign of respiratory distress calls for an immediate chest x-ray. In any event, a chest x-ray should be routinely performed before discharge.
6. An EKG may be necessary because of cardiorespiratory symptoms. The EKG may reveal cardiac rhythm abnormalities that are not obvious on auscultation.
7. Observe carefully for passage of meconium, abdominal distention, and vomiting. A flat plate of the abdomen is indicated early if gastrointestinal symptoms arise; contrast studies should follow if necessary.

8. Consider an ophthalmologic examination before discharge or in the first three months of life.
9. Transilluminate the skull and follow the head circumference daily until discharge.
10. The first well-baby visit should be earlier than routine. The timing of this visit should be dictated by the finding of any congenital abnormalities during the newborn period that portend the possibility of further clinical problems.
11. An intravenous pyelogram should be performed within the first two months of life. Symptoms or signs referrable to the genitourinary system may suggest the need for this procedure before hospital discharge.
12. Follow speech and hearing and motor and adaptive developments carefully throughout infancy.
13. Alert the pedodontist to the possibility of subtle dental abnormalities at his routine three-year dental check.
14. Finally, to detect previously undiscovered congenital abnormalities and subtle functional problems, consider the SUA infant as being at high risk throughout school age.

Acknowledgment

Permission to use the symbol in Figure 1 was granted by the American Academy of Pediatrics.

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FOLLOW-UP OF INFANTS WITH SINGLE UMBILICAL ARTERY

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ABSTRACT. Of 39,773 white and black consecutive single births, 344 (0.9%) had single umbilical artery (SUA). The incidence was higher in whites (1.2%) than in blacks (0.5%). Despite high mortality in infants with SUA (14.0%) the incidence was still 0.7% among surviving infants. Associated malformations were present in 19 of 36 dead infants with SUA, or 52.8%, and in 11 of 266 SUA survivors, or 4.1%. Cardiovascular and genitourinary anomalies were not higher in dead infants with SUA compared to all dead malformed infants.

A follow-up study of infants up to 4 years of age was undertaken, comparing 266 SUA survivors with 798 matched controls. Among malformations found in survivors, only inguinal hernia was significantly higher in SUA children compared to controls. The incidences of other specific abnormal conditions were not significantly different in the two groups. The mean values of body weight, body length, and head circumference at 4 months, 1 year, and 4 years of age, were almost equal in the two groups, as were the mental and motor scores at 8 months and the I.Q. at 4 years of age. *Pediatrics*, 52:6, 1973. SINGLE UMBICAL ARTERY, CONGENITAL MALFORMATIONS, PERINATAL DEATHS, IQ, NEUROLOGICAL ABNORMALITIES.

SINGLE umbilical artery (SUA) is known to be one of the most common malformations in man and is frequently associated with other congenital malformations.[1-15] Other significant associations are with low birth weight (25%), a consistently higher frequency of SUA among whites than among blacks, and a high relationship with diabetes and velamentous insertion of umbilical cord.[1,6] These previous studies have all been on newborn infants and perinatal deaths.

This is a prospective study of single umbilical artery with specific emphasis on a careful follow-up of surviving infants. The objectives are to determine the somatic, mental, and motor development of surviving SUA infants, and to reevaluate the incidence of specific malformations such as genitourinary malformations in dead infants and survivors with SUA. No such follow-up studies have appeared in the literature, and since this is by far the largest prospective collection of these individuals, the findings would be of considerable prognostic value to clinicians.

MATERIAL AND METHODS

The Collaborative Research Study is a prospective study of the relationships between pregnancy and pregnancy outcome.

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The Collaborative Study of Cerebral Palsy, Mental Retardation, and Other Neurological and Sensory Disorders of Infancy and Childhood is supported by the National Institute of Neurological Diseases and Stroke. The following institutions participate: Boston Lying-in Hospital; Brown University; Charity Hospital, New Orleans; Children's Hospital of Buffalo; Children's Hospital of Philadelphia; Children's Medical Center, Boston; Columbia University; The Johns Hopkins Hospital; the University of Minnesota; the University of Oregon; the University of Tennessee; and the Perinatal Research Branch, National Institute of Neurological Diseases and Stroke.

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TABLE I
INCIDENCE AND MORTALITY OF SUA CASES BY RACE AND SEX

		White Male	White Female	White Total	Black Male	Black Female	Black Total	Grand Total
No. of Births		9,891	9,270	19,161	10,321	10,288	20,612	39,773
No. of SUA Cases	Total	110 (93)	122 (112)	232 (205)	50 (40)	62 (57)	112 (97)	344 (302)
	Survivors	95 (83)	108 (101)	203 (184)	41 (33)	52 (49)	93 (82)	296 (266)
	Deaths	15 (10)	14 (11)	29 (21)	9 (7)	10 (8)	19 (15)	48 (36)
Incidence* of SUA	Total	1.1	1.3	1.2	0.5	0.6	0.5	0.9
	Survivors	1.0	1.2	1.1	0.4	0.5	0.5	0.7
SUA Mortality Rate*		13.6	11.5	12.5	18.0	16.1	17.0	14.0
General Mortality Rate*		3.2	3.0	3.1	5.1	3.7	4.4	3.8

* Percent.

Numbers in parentheses are the number of evaluated cases; these were numbers used for calculations in Tables II and III.

Mortality rate: rate of stillbirths and neonatal and infant deaths.

The mothers registered in this study were chosen randomly from 12 participating institutions in the United States. The bulk of the population consists of approximately an equal number of whites and blacks with a small percentage of Puerto Ricans and other races. This report was based on 39,773 white and black single births, of whom 344 (0.9%) had SUA. Of the 344 infants with SUA, 48 (14.0%) were stillborn or died during the neonatal period or in infancy. The remaining 296 are presumed to be living and 266 of these had careful follow-up examinations to the fourth year of their lives.

In the follow-up studies three controls were selected for every case, matched according to institution, race, sex, birth weight, and socioeconomic index.[16]

All examinations were performed according to a structured protocol. Each child had physical examinations at the newborn nursery and then at 4 months of age; a psychological examination utilizing a modified Nancy Bayley scale[17] was performed at 8 months of age. Periodic interval histories were obtained until 4 years of age. At the end of the first year, a thorough physical examination with emphasis on the neurologic parameters was performed. At 4 years of age the child underwent a detailed examination by a psychologist, using the Stanford-Binet intelligence scales. The records of SUA and control children during this four-year period were reviewed, and mean values of various parameters were calculated.

With the exception of abortions, all placentas were carefully examined–and the presence of a single umbilical artery was consistently sought, verified by histologic examination. The definition and criteria for group A and group B malformations were described in detail in another paper.[18] Group A malformations included all uniformly reported macroscopic abnormalities of structure present at birth and resulting from faulty embryological development which normally would have been completed by the end of the ninth week. Group B included anomalies which were thought to be a secondary effect of group A malformations or were felt not to be universally accepted as malformations, or where uncertainty as to consistency of reporting existed in autopsied material.

TABLE II A

INCIDENCES OF SPECIFIC MALFORMATIONS AMONG DEATHS

	Dead SUA Infants		*All Dead Infants*		
	Number	*Percent*	*Number*	*Percent*	
No. of Cases	36		1,498		
Multiply Malformed Patients	19	52.8	75	5.0	$P < 0.001$
Group A					
Central Nervous System	5	13.8	38	2.4	$P < 0.001$
Anencephaly	3	8.3	18	1.1	$P < 0.001$
Arnold chiari	1	2.7	9	0.6	
Spina bifida, all types	2	5.5	7	0.4	$P < 0.001$
Other CNS malformations	1	2.7	14	0.9	
Cardiovascular System	8	22.2	97	6.4	$P < 0.001$
Ventricular septal defect	5	13.8	38	2.5	$P < 0.001$
Atresia or stenosis, L valves	2	5.5	13	0.8	
Atresia or stenosis, R valves			11	0.7	
Other valve anomalies	1	2.7	11	0.7	
Atrial septal defect			16	1.0	
Tetralogy of Fallot			3	0.2	
Atrioventricularis communis			11	0.7	
Preductal coarctation	1	2.7	17	1.1	
Truncus communis	1	2.7	6	0.4	$P < 0.05$
Transposition, great vessels	1	2.7	11	1.1	
Vascular ring	2	5.5	8	0.5	$P < 0.001$
Anomalous pulmonary venous drainage			6	0.4	
Patent ductus arteriosus			13	0.8	
Other CVS malformations	1	2.7	15	1.0	
Other Blood Vessels	36	100.0	38	2.5	$P < 0.001$
Arteriovenous aneurysm			2	0.1	
Single umbilical artery	36	100.0	36	2.4	$P < 0.001$
Respiratory System	1	2.7	4	0.2	$P < 0.01$
Genitourinary System	10	27.7	64	4.2	$P < 0.001$
Polycystic kidney	7	19.4	24	1.6	$P < 0.001$
Atresia, ureter or urethra	3	8.3	25	1.6	$P < 0.01$
Agenesis, kidney			7	0.4	
Horseshoe kidney			6	0.4	
Double ureter	1	2.7	6	0.4	$P < 0.05$
Agenesis, urinary bladder	1	2.7	1		
Persistent cloaca	1	2.7	2	0.1	$P < 0.001$
Agenesis, uterus or ovary	1	2.7	3	0.2	$P < 0.01$
Bicornuate uterus			4	0.2	
Pseudohermaphroditism			1		
Other GUS malformations	1	2.7	7	0.4	
Digestive System	8	22.2	50	3.3	$P < 0.001$
Cleft palate	1	2.7	6	0.4	$P < 0.05$
Cleft palate with cleft lip			5	0.3	
Cleft lip			2	0.1	
Atresia, esophagus	1	2.7	2	0.1	$P < 0.001$
Atresia, small bowel			4	0.2	
Meckel's diverticulum	1	2.7	15	1.0	
Hirschsprung's disease	1	2.7	7	0.4	
Imperforate anus	2	5.5	5	0.3	$P < 0.001$
Agenesis, gallbladder	2	5.5	4	0.2	$P < 0.001$
Atresia, bile duct	1	2.7	3	0.2	$P < 0.01$
Other DS malformations	3	8.3	6	0.4	$P < 0.001$

TABLE II A *(Continued)*
INCIDENCES OF SPECIFIC MALFORMATIONS AMONG DEATHS

	Dead SUA Infants		*All Dead Infants*		
	Number	*Percent*	*Number*	*Percent*	
Musculoskeletal System	5	13.8	41	2.7	$P < 0.001$
Polydactyly	1	2.7	11	0.7	
Syndactyly			2	0.1	
Rib or vertebral malformations	1	2.7	12	0.8	
Agenesis, finger or major bones	2	5.5	6	0.4	$P < 0.001$
Skull bone or suture defects			7	0.4	
Chondrodystrophy			1		
Agenesis, abdominal or major muscles	1	2.7	4	0.2	$P < 0.01$
Other MSS malformations			7	0.4	
Unspecified Systems	8	22.2	39	2.6	$P < 0.001$
Diaphragmatic hernia	3	8.3	13	0.8	$P < 0.001$
Omphalocele	4	11.1	15	1.0	$P < 0.001$
Agenesis, spleen	1	2.7	3	0.2	$P < 0.01$
Atresia, ext. auditory meatus	1	2.7	3	0.2	$P < 0.01$
Other UNS malformations			5	0.3	
Group B					
Hydrocephalus, NOS			9	0.6	
Microcephalus	1	2.7	2	0.1	$P < 0.001$
Hypoplasia, L heart	2	5.5	11	0.7	
Hypoplasia, R heart	1	2.7	4	0.2	$P < 0.01$
Primary endocardial fibroelastosis			7	0.4	
Anomalous venous drainage			3	0.2	
Hypoplasia, lungs	6	16.6	37	2.4	$P < 0.001$
Abnormal lobation, lung	2	5.5	26	1.7	$P < 0.001$
Malrotation, bowel			27	1.8	
Hypoplasia, kidney			6	0.4	
Hypospadias			5	0.3	
Talipes, all types	4	11.1	31	2.0	$P < 0.001$
Kyphosis or scoliosis	2	5.5	9	0.6	$P < 0.001$
Arthrogryposis			7	0.4	
Accessory spleen	4	11.1	67	4.4	$P < 0.001$
Mongolism			13	0.8	
Trisomy 13 through 18	1	2.7	3	0.2	$P < 0.01$
Coloboma			3	0.2	
Microphthalmia			2	0.1	
Abnormal position or shape, ear	1	2.7	12	0.8	
Webbed neck	1	2.7	5	0.3	$P < 0.001$

RESULTS

Table I shows that for either sex the overall incidence in whites (1.2%) was twice as high as that in blacks (0.5%). This white preponderance in incidence persisted among survivors. In both white and black the general incidence was higher in females than in males, and conversely, the mortality of SUA infants was higher in males than in females, but these sex differences were not statistically significant. The SUA mortality (14.0%) was about four times higher than that of the general population (3.8%). Numbers in parentheses (Table I) are the numbers of evaluated cases, which were used for the calculations in Tables II, III, and IV; the numbers above these constitute the total number of known SUA infants. Thus, the difference between the two, or the nonevaluated cases, consist of dead in-

TABLE II B

INCIDENCES OF SPECIFIC MALFORMATIONS AMONG SURVIVORS

	SUA Survivors		*Control Survivors*		
	Number	*Percent*	*Number*	*Percent*	
No. of Cases	266		753		
Multiply Malformed Patients	11	4.1	0	0.0	
Group A					
Spina bifida	1	0.3			
Single umbilical artery	266	100.0			
Cleft palate	1	0.3	1	0.1	
Atresia, esophagus	1	0.3			
Atresia, small bowel	1	0.3			
Imperforate anus	1	0.3			
Polydactyly	2	0.7			
Syndactyly	3	1.1	2	0.2	
Rib or vertebral malformations	3	1.1			
Group B					
Hydrocephalus, NOS	1	0.3			
Microcephalus	3	1.1			
Talipes, all types	6	2.2	18	2.3	
Abnormal position or shape, ear	1	0.3	3	0.3	
Inguinal hernia	14	5.2	9	1.1	$P < 0.001$

fants who were not autopsied, survivors without follow-up and survivors with less than three matched controls.

The incidences of group A and B malformations based on all infants autopsied are shown in Table IIA. Of 1,498 total infants autopsied, 15.7% or 235 had group A malformations. Among deaths the incidence of two or more group A malformations occurring in the same infant, was significantly higher in the SUA group (52.8%) than in the total group (5.0%). The rates of specific malformations in the SUA group were three to six times higher than in the total group. Evaluation for statistical significance was hampered by small numbers. Among 266 SUA survivors group A malformations were found in 11, or 4.1% (Table IIB). There was no case of two or more group A malformations occurring in the same infant among 753 control survivors. Lethal malformations such as duodenal atresia, imper-

TABLE III

FREQUENCY OF ORGAN SYSTEM INVOLVEMENT IN DEAD INFANTS WITH MALFORMATIONS

	SUA Infants		*All Malformed Infants*		
	Number	*Percent*	*Number*	*Percent*	
No. of Cases	36		235		
Central nervous system	5	13.8	38	16.1	
Cardiovascular system	8	22.2	97	41.2	$P < 0.05$
Respiratory system	1	2.7	4	1.7	
Genitourinary system	10	27.7	64	27.2	
Digestive system	8	22.2	50	21.2	
Musculoskeletal system	5	13.8	41	17.4	
Unspecified system	8	22.2	39	16.5	

TABLE IV

MEAN VALUES OF VARIOUS DEVELOPMENTAL SCALES AND MOTOR-MENTAL SCORES IN SURVIVORS

	SUA Survivors	*Control* Survivors*
No. of Cases	266	798
Body Weight		
At birth, gm	3092.4±542.9	3271.2±462.7
At 4 months, gm	6167.7±926.9	6381.6±872.0
At 1 year, kg	9.2± 3.1	9.4± 3.1
At 4 years, kg	16.8± 10.1	16.4± 6.5
Body Length		
At birth, cm	49.7± 2.7	50.3± 2.4
At 4 months, cm	62.2± 3.3	62.5± 3.1
At 1 year, cm	74.1± 4.0	74.5± 3.4
At 4 years, cm	100.3± 6.8	101.3± 8.1
Head Circumference		
At birth, cm	33.6± 1.6	33.9± 1.3
At 4 months, cm	40.5± 1.7	40.7± 1.5
At 1 year, cm	45.6± 1.7	45.8± 1.5
At 4 years, cm	49.9± 1.7	50.0± 1.8
Motor score at 8 months	32.7± 4.9	33.7± 4.4
Mental score at 8 months	79.6± 5.8	79.9± 5.2
IQ at 4 years	100.7± 16.9	99.5± 16.6

* Three controls per case were matched according to institution, race, sex, birthweight and socioeconomic index.

forate anus and tracheoesophageal fistula were found in SUA survivors and these were repaired surgically. Inguinal hernia was significantly higher in SUA survivors (5.5%) than in controls (1.1%).

Table III shows the frequency of organ system involvement in dead SUA infants compared with all dead malformed infants (malformed group). Again, evaluation was limited to group A malformations. Each malformed case was entered only once in the involved system, regardless of the number of malformations within the system. The cardiovascular system (heart and great vessels) was the most frequently involved in the malformed group, whereas in the SUA group the genitourinary system ranked first. However, the rate of genitourinary system involvement in the SUA group (27.7%) was similar to that of the malformed group (27.2%). It is of interest that the cardiovascular system was far less frequently involved in the SUA group (22.2%) as contrasted to the malformed group (41.2%).

In Table IV the mean values and standard deviations of body weight, body length, head circumference, motor score (8 months of age), mental score (8 months), and I.Q. (4 years) are listed for 266 SUA survivors and 798 surviving controls. Body weight, body length, and head circumference were recorded at four developmental stages: birth, 4 months, 1 year, and 4 years. Although all attempts were made to closely match for birth weight, the mean birth weight of the SUA group was slightly lower than that of controls (Table IV). Differences in body measurements at birth disappeared during the ensuing four years. At 4 years of age the mean values of body weight, body length, head circumference, and IQ were nearly equal between cases and controls.

A summary of the clinical diagnoses and major events during the first year of life were analyzed in 286 SUA cases and 798 controls. The results of the 4-month pediatric, 8-month psychological, and 1-year neurological examinations were included in

this summary. This consisted of 252 separate items involving 23 organ systems and organs, with special emphasis on congenital malformations and neurologic abnormalities. Only definite diagnoses were considered, and except for inguinal hernia (SUA 5.5%, controls 1.1%) the incidence of specific abnormal conditions was not significantly different between cases and controls.

DISCUSSION

Previous reports have emphasized the high association of SUA with other congenital malformations as one of the most significant findings. In this study this was true of autopsied infants with SUA, in which the malformation rate was 52.8%, compared to only 5.0% in all autopsied infants. However, surviving SUA infants were surprisingly normal in their somatic and mental development. The malformation rate was 4.1% in these SUA survivors. This suggests that SUA infants with severe abnormalities tend to die off during the early phase of life, particularly during the perinatal period. Those surviving the perinatal period appear to develop as normally as children of comparable birth weight and socioeconomic status. Genitourinary malformations, although mentioned by some as being significantly associated with SUA,[19] were not more frequent in dead SUA infants compared to the total group of dead malformed infants (Table III) and were not found in any of those surviving up to 4 years of age. Bilateral renal malformations were relatively frequent in all malformed deaths (Table III), and the majority of these deaths occurred in the early neonatal period; this type of occurrence is therefore not unique to SUA infants.

It was interesting to find that the cardiovascular system was only about half as often involved in dead SUA infants (22.2%) as in the malformed group of dead infants (41.2%). The reason for this is unknown and could perhaps merely reflect the fact that the cardiovascular system is that most frequently involved in fatal malformations and that often these heart and great-vessel malformations are isolated, unaccompanied by anomalies of other organs, especially in males.[18]

The pathogenesis of SUA has been the subject of speculation, some investigators believing that it results from aplasia or atrophy of the missing vessel. Monie,[20] in his study of human embryos, found that a single umbilical artery is normally present in the second phase of development, replacing the plexus of arteries around the allantois, and that in the third phase the single artery becomes shorter and the right and left umbilical arteries advance distally within the body stalk. He believes that SUA could be due to persistence of this normally transient single umbilical artery, associated with degeneration of either the right or left umbilical artery. According to Monie,[20] in Long-Evans rat embryos the umbilical arteries normally unite within the body stalk to form a single artery about the 11th day of pregnancy and extends almost the full length of the cord during the remaining 11 or 12 days of gestation. It is interesting to know that while SUA is one of the most common malformations in humans it is also normally present during the early development of human embryos.

It has been suggested that SUA in man leads to hypoxia, in turn causing embryonic abnormality. However, our study shows that there remain a considerable number of infants in whom SUA is the only anomaly, and these infants appear to develop normally. It is more likely that in multiply malformed cases, SUA is merely one of the complex of malformations due to some precipitating factor, rather than it being the cause of the other malformations.

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EDITOR'S NOTE: A Commentary on this article appears on page 116.

SCREENING FOR RHo (D) IMMUNE GLOBULIN (HUMAN)

SCREENING FOR RHo(D) IMMUNE GLOBULIN (HUMAN)

A speciman of cord blood should be collected at the time of delivery for direct Coombs test on all infants of Rh negative mothers. If the direct Coombs on the cord blood is negative, the administration of RHo (D) Immune Globulin to the mother is indicated.[1] The administration of the drug must be done within 72 hours after delivery.[2]

Attention should be also given to the Rh negative patient who aborts a pregnancy. In these cases there is little chance to obtain a fetal blood sample. However, if the patient is Rh negative and not already sensitized (negative indirect Coombs) she should receive the Immune Globulin just in case the fetus she aborted was Rh positive.[3]

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2. Ibid
3. White, Charles A.: Rho(D) Immune Globulin to Prevent Rh Hemolytic Disease Am. Family Physician Vol. 3 No. 2 Feb. 1971 85-89

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Rh_o(D) Immune Globulin to Prevent Rh Hemolytic Disease

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Preparation of concentrated Rh_o(D) immune globulin has permitted use of antibody-mediated immune suppression to prevent erythroblastosis in Rh-incompatible pregnancies. Since sensitization of the Rh-negative mother occurs during labor or delivery, administration of this material shortly after delivery suppresses the expected immune response and the mother, if previously unsensitized, does not form anti-Rh antibodies. Therefore, in subsequent pregnancies, the maternal system shows no prior sensitization and fetal hemolytic reactions are avoided. The safety and efficacy of this method have now been established.

Most cases of severe erythroblastosis in infants are due to isoimmunization of Rh-negative women from previous Rh-incompatible pregnancies. Sensitization follows the entrance of fetal Rh-positive erythrocytes into the maternal circulation during labor or delivery. Synonyms for the Rh factor are Rh_o and D, according to the Wiener and Fisher-Race classifications. The active Rh_o (D) antibody produced by the mother leads to fetal hemolytic disease in subsequent Rh-positive pregnancies.

A transplacental fetal-maternal hemorrhage which occurs during pregnancy but before labor rarely results in isoimmunization, either because the immune reaction is depressed during pregnancy or because the hemorrhage is too small to evoke an antibody response. During delivery, however, fetal blood can enter the maternal circulation in amounts of 0.1 to 0.2 ml., which are sufficient to cause sensitization.

Since the Rh antigen exists solely in the erythrocytes of humans and primates, there are only two other ways in which an Rh-negative woman can receive the necessary antigenic stimulus—from her mother's Rh-positive erythrocytes (during her own fetal life) and from previous transfusion or intramuscular injection of Rh-positive blood. Both these means of immunization are uncommon.

Immune Suppression

In 1909, Theobald Smith found that in the presence of sufficient passive antibody, the corresponding antigen would not immu-

nize. Subsequent studies have shown that passive antibody is equally immunosuppressive when given before or just after antigen injection or when mixed with the antigen. This *antibody-mediated immune suppression* appears to apply to most antigen-antibody systems studied, although it is generally more effective against weak antigens and often can be overcome by large doses of antigen.

In 1950, Pollack, Görman and Freda began a program to determine whether antibody-mediated immune suppression could prevent sensitization of Rh-negative mothers. To provide a convenient, potent and reproducible preparation suitable for intramuscular injection and known to be free from the risk of transmitting serum hepatitis, the investigators fractionated and obtained the gamma globulin from plasma containing anti-Rh antibody. Their original study in male volunteers clearly demonstrated that, with adequate dosage, this 7S anti-Rh globulin could completely suppress a primary immune response. In addition, no unwanted side effects were observed.

Clinical trials in mothers were begun in April 1964 and were extended to include 42 centers in the United States and abroad. This article describes one of these clinical studies, at the University of Iowa Hospitals, in which 100 women were admitted to a controlled, double-blind program which tested the efficacy of Rh_0(D) immune globulin (Rho-GAM®).

Material and Methods

The Rh_0(D) immune globulin used in the study was prepared at the Ortho Research Foundation. It was fractionated from the plasma of patients previously immunized to the Rh antigen by Rh-incompatible pregnancies. The final product was a sterile, concentrated preparation of immunoglobulin G containing anti-Rh antibodies.

The treated group received 1 ml. of Rh_0 (D) immune globulin, containing no less than 300 mcg. of anti-Rh antibody, within 72 hours postpartum. The control group received 1 ml. of homologous gamma globulin without anti-Rh antibody. Although much larger doses of homologous gamma globulin have been reported to inhibit some antibody formation, such an effect was not the intent in this study; the aim was merely to give the control and treated patients products that were as similar as possible, except for the presence of anti-Rh antibody.

CRITERIA

The following criteria were used for admission to the study: (1) The mother had to be Rh-negative and previously unimmunized to the Rh antigen, as determined by the absence of an anti-Rh antibody in her serum prior to and at the time of delivery. (2) The infant had to be Rh-positive, ABO-compatible (infant's erythrocytes with maternal serum) and the direct Coombs reaction on cord blood specimen had to be negative. Only ABO-compatible cases were included because ABO incompatibility between fetal erythrocytes and maternal serum may partially protect against Rh immunization.

Routine ABO and Rh typing, as well as antibody screening, had been performed on the blood of all candidates between the fourth and seventh months of pregnancy. Screening of the maternal sera for the presence of atypical antibodies was also performed at the time of delivery to ensure that none of these patients had become immunized. On delivery of the infants, ABO and Rh typing and direct Coombs tests were performed on cord blood specimens. Candidates whose babies had positive direct Coombs reactions (regardless of the specificities of the antibodies) were excluded. The patients admitted to the study were

randomly assigned to the control and treated groups, so that both groups would have essentially the same socioeconomic background, race, age and gravidity.

Prior to administration, a 1:1,000 saline solution of the $Rh_0(D)$ immune globulin to be injected was mixed with a 2 to 5 percent suspension of maternal erythrocytes. Routine compatibility testing was then performed in saline, high concentrations of protein and by the anti-human globulin technique. The compatibility test served to prevent accidental administration of the $Rh_0(D)$ immune globulin to Rh-positive women.

FOLLOW-UP

Follow-up serologic evaluation of the patients was done within 48 to 96 hours after injection and at six weeks, three months and six months postpartum. Antibody screening was performed with commercially available screening cells and a broad-spectrum anti-human globulin (Coombs) serum. In the majority of patients, studies for the presence of circulating fetal erythrocytes in the maternal circulation were done on venous samples obtained within 72 hours before treatment and also from 48 to 96 hours after treatment.

Results

In this series, 66 women were placed in the treated group that received $Rh_0(D)$ immune globulin. Fifty-eight of the 66 were followed for six months but only partial follow-up data are available for the other eight women, who were lost to the study between six weeks and six months after delivery. Of course, all the patients were unsensitized at the time of delivery and each of the 66 demonstrated a passive indirect Coombs anti-Rh titer when tested 48 to 96 hours after injection. However, by the sixth month after injection, each of the 58 women again had a negative indirect Coombs test.

Thus, none of the treated patients available for six-month follow-up became actively sensitized; they are considered to have received complete protection against primary immunization during the pregnancy under study. Of these 58 women, nine have since delivered another Rh-positive infant without any signs of Rh sensitization.

The 34 women in the control group received gamma globulin which did not contain anti-Rh antibody. Twenty-eight of the 34 were followed for six months; follow-up was incomplete for the six others. All 34 patients had negative indirect Coombs tests through the first six weeks postpartum. However, one control patient manifested evidence of Rh sensitization by conversion to a 1:8 indirect Coombs titer at both three months and six months; also, at 10 months postpartum, her anti-Rh Coombs test had a 1:2 titer. This patient has not had a subsequent pregnancy. One other control patient has delivered another Rh-positive infant without signs of Rh sensitization.

The relatively small number of patients in this series does not permit reliable statistical scrutiny. However, compilation of data reported to the Ortho Research Foundation from worldwide clinical studies has provided complete six-month postpartum information on 4,080 patients. These data (*Table 1*) show that of the 2,880 women who received

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Rh_0(D) immune globulin within 72 hours after delivery, only three (0.1 percent) demonstrated active anti-Rh antibodies. In contrast, 84 of 1,200 control patients (7 percent) became sensitized.

Of course, the more critical data involve the 377 patients who have subsequently delivered an Rh-positive infant (*Table 2*). Of the 229 women who had Rh_0(D) immune globulin prophylaxis at the time of the preceding pregnancy, only one (0.4 percent) had an anti-Rh titer. This is particularly encouraging when compared with 21 instances of Rh sensitization among 148 control patients, an incidence of 14.2 percent.

TABLE 1.

Data on Patients Followed Six Months Postpartum*

Patient group	*Total number of patients*	*Anti-Rh antibody*	*No anti-Rh antibody*
Treated	2,880	3 (0.1%)	2,877
Control	1,200	84 (7.0%)	1,116

**Compiled from worldwide clinical studies of women receiving Rh_0(D) immune globulin within 72 hours after delivery.*

TABLE 2.

Results of 377 Subsequent Rh_0(D) Deliveries

Patient group	*Total number of patients*	*Anti-Rh antibody*	*No anti-Rh antibody*
Treated	229	1 (0.4%)	228
Control	148	21 (14.2%)	127

Comments

The safety and efficacy of antibody-mediated immune suppression in the prophylaxis of Rh sensitization are well established. This report of 100 women who participated in a double-blind study has corroborated the results of earlier Rh_0(D) immune globulin studies. None of the 58 treated women and one of the 28 control patients who were followed for six months postpartum demonstrated anti-Rh antibody at the end of that period.

EFFECT ON FETAL ERYTHROCYTES

One aspect of these data contrasts with earlier reports. This concerns the number of fetal erythrocytes found in maternal venous blood 48 to 96 hours after injection of either the Rh_0(D) immune globulin or the placebo gamma globulin. Although Rh_0(D) immune globulin apparently does not act by hemolysis of Rh-positive fetal erythrocytes, it has been suggested that these fetal erythrocytes might be sequestered more promptly into the maternal reticuloendothelial system after combining with anti-Rh antibody. Thus, one would anticipate a reduction in the number of fetal erythrocytes in the venous blood of the treated group of women but not of the control group, if sufficient time has elapsed for such sequestration to take place. However, our data show no consistent difference between treated and control patients in clearance of fetal cells. This suggests that some mechanism other than simple rapid clearance is responsible for the effectiveness of this technique.

Recommendations for Use

Since the Rh_0(D) immune globulin was licensed for manufacture and commercial distribution in April 1968, it is appropriate to review current recommendations for its use in obstetric patients. It must be administered to the Rh_0(D)-negative, D^u-negative postpartum patient within 72 hours of delivery of an Rh_0(D)-positive or D^u-positive baby. The mother must not be

immunized to the $Rh_0(D)$ factor (negative indirect Coombs test) and the baby should have a negative direct Coombs test. Since ABO incompatibility between the Rh-negative mother and her Rh-positive fetus offers only incomplete protection against Rh isoimmunization, the use of immune prophylaxis in these cases is recommended.

It has been suggested that a miscarriage can cause primary immunization by the entry of even a small amount of Rh-positive fetal cells into the Rh-negative woman's blood. Therefore, if an abortion occurs in an Rh-negative woman whose husband is Rh-positive, she should be considered for protective therapy with $Rh_0(D)$ immune globulin, which should be given within 72 hours of the abortion.

Several contraindications should be noted. $Rh_0(D)$ immune globulin should not be administered to: (1) an Rh-positive or D^u-positive patient, because it might produce hemolysis; (2) an Rh-negative patient who has received an Rh-positive blood transfusion; (3) a patient who is already immunized to the Rh-positive blood factor, because it will be ineffective, and (4) a patient prior to delivery, even if there is evidence of a large transplacental hemorrhage; the immunoglobulin might cross the placenta and provoke hemolysis in the Rh-positive fetus.

Adverse reactions have been mild, infrequent and usually confined to the intramuscular injection site. A slight elevation of temperature has been noted in a few postpartum women. Systemic reactions have not been reported in this type of patient and sensitization due to repeated injections of immunoglobulin G is unusual and of doubtful clinical significance. Human immune serum globulin does not transmit serum hepatitis.

The Future

Prevention of Rh hemolytic disease by $Rh_0(D)$ immune globulin is an important therapeutic advance. Since previously sensitized Rh-negative women will continue to contribute cases of fetal hemolytic disease until they are beyond the childbearing years, the effect of $Rh_0(D)$ prophylaxis on perinatal mortality and morbidity will be gradual. However, if all physicians will use this means of improving obstetric care, the incidence of erythroblastosis should almost vanish within the next generation.

POSTPARTUM CARE

POSTPARTUM CARE

The immediate postpartum period is often referred to as the "fourth stage of labor." During this time the patient is at risk for post-delivery and post-anesthetic complications.[1] It is during this period that postpartum hemorrhage--the most serious complication of normal birth--is likely to occur. It is also the time that some of the most serious anesthesia complications occur--hypotension, hypertension, hyperventilation, and emesis with aspiration.[2]

Recovery observations on the postpartum patient must be frequent, specific, and thorough, according her the same degree of concern as is given to any new post-operative patient. The following should be noted and charted:[3]

1. Beginning immediately after removal of the patient from the delivery room: height and condition of the uterine fundus, amount of lochia, Maternal temperature, pulse, and respiratory rate, and maternal blood pressure.
2. Following this, every 15 minutes x 4, check and record: fundus, lochia, pulse, and blood pressure. Repeat this again every 30 minutes x 4, and again hourly until the patient is 8 hours post-delivery. Check for bladder distention, and encourage the patient to void within 6-8 hours.
3. The above observations should then be carried out every four hours for the first 24 hours, and then should be done once every day until discharge. Temperatures should be taken on the postpartum floor at least QID.

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1. Bonica, John: Obstetric Analgesia and Anesthesia Vol 1 & 2 F. A. Davis Company, Philadelphia 1972 Page 927
2. Ibid Page 927
3. Standards for Obstetric-Gynecologic Services: The American College of Obstetricians and Gynecologists (1969) page 42-44

Efficient Postpartum Checks

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SCHOOL OF NURSING

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IN CARRYING OUT POSTPARTUM checks, we know more than ever before that it is important to be efficient. A nurse researcher, Reva Rubin, has identified a postpartum stage of restoration that begins after the mother has a deep, continuous, spontaneous sleep. Without this restoration stage, she may exhibit signs of sleep hunger: degrees of anxiety, restlessness, moodiness, and others.

Proper rest can be facilitated if your checks give maximum care during brief contact with the mother at specific intervals. These checks should include:

- palpation of the fundus of the uterus
- slight massage of the fundus and observation for bladder distention
- expression of clots and free blood from the uterus
- measurement of the fundus in relation to the umbilicus
- inspection of the perineum for discoloration and swelling
- inspection and change of perineal pads
- recording of blood pressure and pulse
- offering food and fluids if allowed
- ensuing comfort and safety measures throughout all steps of the check

The American College of Obstetricians and Gynecologists recommends checking the newly delivered mother every 15 minutes during the first hour postpartum. During these checks, the nurse should look for uterine atony, hemorrhage, deviations in blood pressure and pulse, and other complications. Checks should be repeated every half hour during the second and third hours postpartum. Or, naturally, more frequently if you see any deviations in the mother's condition.

Ideally, the checks should be done at specific time intervals so you can compare the mother's condition sequentially. Procedures basic to nurses—but often neglected—include explaining the procedure to the patient and providing privacy for the mother before and during the check.

Here are some specific points to remember.

1. Instruct the mother to concentrate on relaxing during the check. Palpation, massage, and expression of the fundus of the uterus may be uncomfortable, so anticipate this with her. But tell her that intentional relaxation of the abdominal muscles will speed examination and lessen discomfort.

2. You need not always ask the father to leave the room during the checks. Consult the parents regarding their desires. If the father stays, be creative: use the bed linens, furniture, father's body position, and your body position to afford the mother privacy.

3. Do not assume that because sufficient doses of oxytocics were given, continual surveillance of the uterus' condition is not necessary. The tone of the uterus depends on many interrelated physical factors, only one of which is whether oxytocics were given.

4. Remember that the mother's uterus contracts when she sees or hears her newborn. This sympathetic nervous system response aids in preventing postpartum hemorrhage. The increased contractions may cause discomfort, and the mother will certainly need her perineal pads changed as soon as the baby leaves.

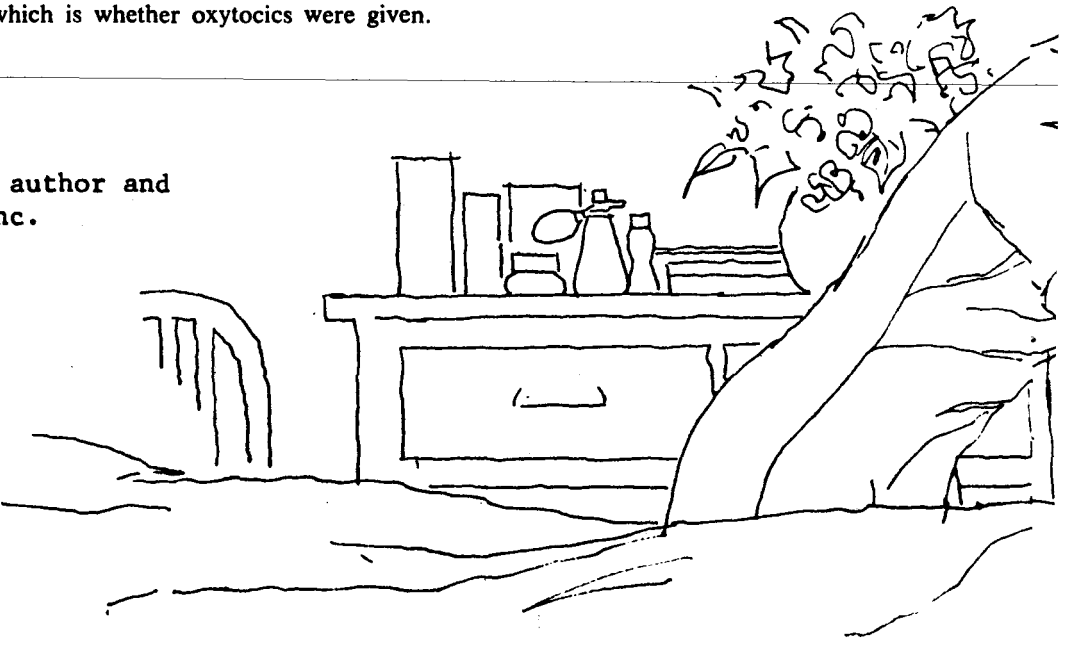
5. Palpate the uterus by placing the side of one hand on top of and slightly cupped under the fundus of the uterus, while placing the other suprapubically with *slight* exertion of pressure.

6. Remember that the fundus usually lies on the midplane of the pelvis, at or below the umbilicus.

7. Avoid unnecessary manual stimulation of the uterus in the absence of bleeding or increased size. It may cause overstimulation of the uterine muscles. This in turn can cause undue muscle fatigue, subsequent relaxation of the uterus, and possible hemorrhage.

8. Each time you palpate the uterus, observe the bladder area to see if there's a "bladder bulge." This is a spongy raised area over the symphysis pubis, and it indicates a filling or full bladder. The uterus must not be expressed if the bladder is full, but rather after it has been emptied.

9. Keep in mind that patients who had caudal or saddle block anesthesia do not usually experience urge to urinate.



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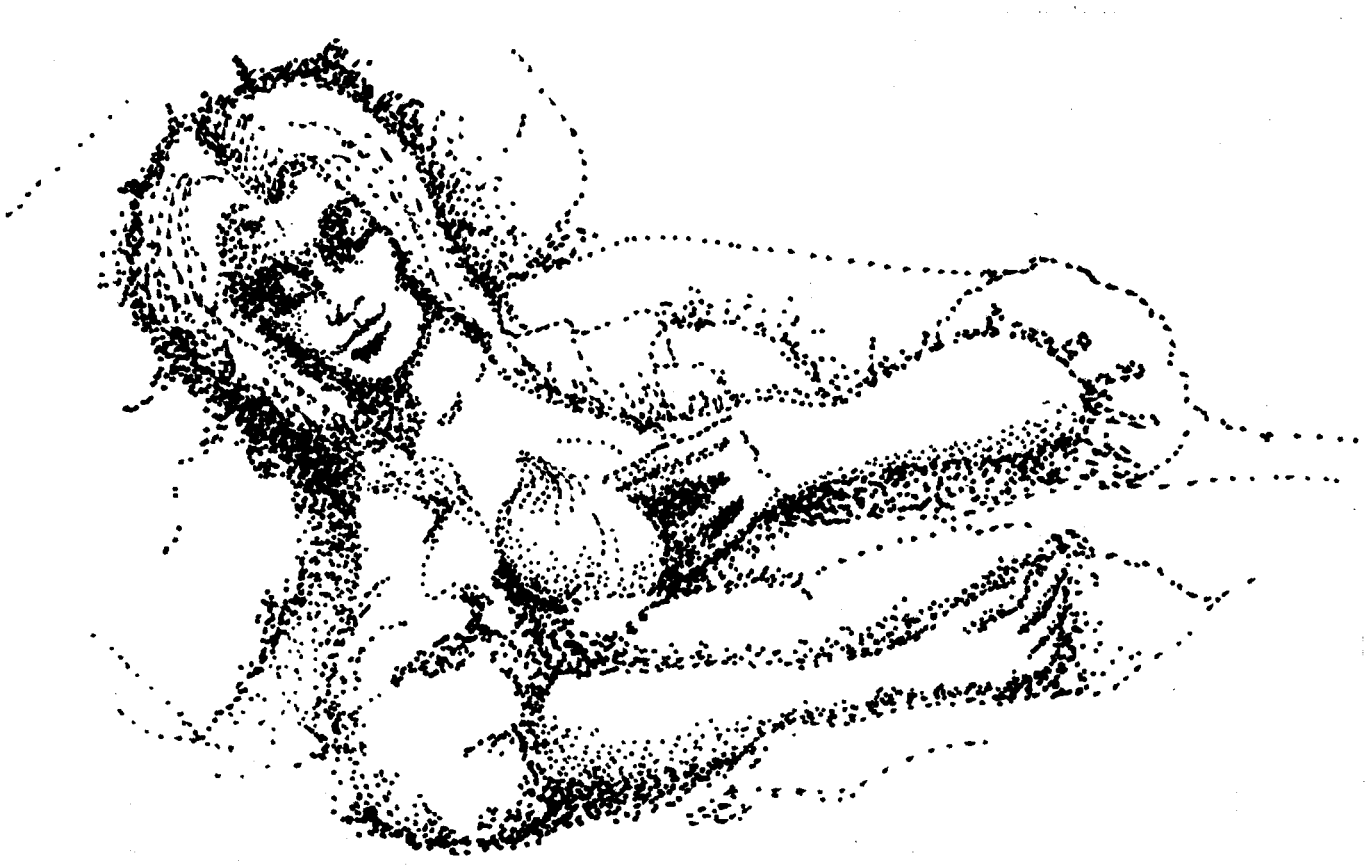

Breast Care in the Early Puerperium

BETTY ANN COUNTRYMAN, RN, AB, MN

The most frequent reasons given by mothers for early discontinuance of breastfeeding are breast and nipple problems accompanied by pain and discouragement. Most of these problems can be avoided by appropriate breast care in pregnancy, minimal medication in labor and delivery, attention of nursing personnel to the breasts and nipples in the early puerperium, and instruction of the mother in correct nursing techniques. In this article early and frequent, unscheduled nursing is advocated; management of engorgement is considered. Nurses' attitudes are discussed in terms of their effects on the mother's success or failure in beginning breastfeeding.

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Many infants who began to breastfeed at birth leave the hospital either partially or totally weaned, and many additional babies are weaned before 6–8 weeks of age. Reasons for this attrition are numerous. Some discontinuance of breastfeeding undoubtedly occurs because of unavoidable problems within the family, sometimes completely unrelated to the infant's feeding. But by far the most common reasons mothers give for early weaning are complaints of breast or nipple problems. Many of these difficulties arise from improper management of breastfeeding in the early puerperium. The nurse is in a position to help the new mother avoid or mitigate the problems in many cases.

Three primary factors influence the beginning of breast and nipple problems: 1) failure to prepare the breasts and nipples prenatally, 2) overmedication in labor and delivery, 3) insufficient attention by nursing personnel to early postpartal breast and nipple care, and failure to instruct the mother in breastfeeding techniques.

Appropriate breast care in the antepartum will prepare a mother for nursing,[1] and its importance should be emphasized to all expectant mothers. Such preparation may benefit the non-nursing mother, it prepares the undecided pregnant woman who may later elect to nurse, and it is especially valuable for the woman who definitely intends to breastfeed.

Little or no medication in labor and delivery contribute to a successful start in breastfeeding.[2,3] Following a highly medicated intrapartum and delivery, a mother may be too lethargic or nauseated to want to nurse her baby for 24 hours or more. The infant who has experienced such a delivery, too, is often over-medicated, sluggish and lacking in a strong sucking reflex. The baby's diminished interest in suckling may last up to three or four days after birth. During this time the mother's milk generally comes in and the baby's failure to nurse may lead to an aggravation of maternal engorgement accompanied by severe pain. When this occurs, many a mother gives up nursing and requests a lactation suppressant and formula for the baby. The problem could be prevented if the baby were not a victim of his mother's analgesia and/or anesthesia. In an uncomplicated labor and delivery, therefore, analgesics and anesthetics should be kept to a minimum in the best interests of successful initiation of lactation.

Breastfeeding Techniques

Following an uncomplicated delivery, care of the mother's breasts and instruction in breastfeeding techniques may begin almost immediately. During the first few hours after birth, the unmedicated infant has a heightened sucking reflex. In view of this, every effort should be made to give him the opportunity to suckle, preferably in the delivery room.[4] Certainly no more than an hour should elapse before the baby is put to breast if the delivery was not difficult or traumatic. At this early time there is rarely any difficulty in establishing the infant at breast and little need to teach him to suck.

At the first nursing the mother's nipples may be cleansed with clear water if cleansing is thought necessary. At no time should soap be used on the nipples of the nursing mother. Even the use of water is unnecessary under most circumstances since the nipple skin itself is cleansed by the natural antiseptic, lysozyme. No drying agent should be used on the mother's nipples since it tends to remove the secretions of sebum, a physiologic emollient. Under normal circumstances, the best nipple care is provided by the body itself, without outside interference.

Following the first nursing in delivery or recovery room, and within 1 or 2 hours, a second opportunity for nursing should be given, unless the infant is sleeping soundly. Thereafter, the baby should be permitted to nurse on demand—his OR his mother's demand.

When the mother leaves the recovery room, the ideal background for unscheduled frequent feedings is rooming-in, but it is entirely appropriate for the infant to be taken to his mother for demand feeding from a central nursery. If permitted to nurse at will, the pain and nipple damage which result from the over-vigorous sucking of the scheduled baby can be avoided.[5]

Whether in delivery, recovery or the mother's own room, a knowledgeable nurse should attend the first several nursing periods of the inexperienced nursing couple to give help and encouragement. The nurse should position mother and baby comfortably on their sides, facing each other. The mother's knees should be flexed. Supporting pillows should be placed at her back and under her head. The baby's mouth should be at the level of the mother's nipple. He should then be snugged up to the mother and permitted to root for the nipple. At no time should he be forced to the breast by pressure on his head or face. Instead, the mother may point up her nipple with thumb and forefinger so that the baby can grasp it more easily. Once the

baby has found the nipple, the mother may gently press back on her breast to maintain a free airway for the infant.

The first nursing may be very brief or may last 5–10 minutes. To prevent nipple trauma when removing the infant from the breast, if he does not spontaneously relinquish his hold, his suction should be broken by downward pressure on his chin or by insertion of a clean finger into the corner of his mouth.

During the first day or so, the baby should be permitted to nurse 5–7 minutes on each breast at every nursing period. This time limitation is a precautionary measure to prevent sore nipples and is particularly important for the thin-skinned mother or for one who has not prepared her nipples prenatally. After 24 hours or so, the time at each breast may be gradually increased, as tolerated. By the time mother and baby leave the hospital (about the fourth day), the baby should be nursing for 10–15 minutes on the first breast and for as long as he is productively sucking on the second.

The mother should be carefully instructed in nursing techniques. She should alternate the breast first used at successive feedings and understand the importance of breaking the baby's suction correctly before removing him from the nipple. She should be encouraged to wear a well-fitting, supportive nursing brassiere as soon as her milk begins to come in. She should become accustomed to lowering the flaps of the brassiere occasionally during the day for 15–30 minutes. The brassiere should have no plastic liner in the cups to occlude air from the nipples. If there is leaking of milk, the mother may use disposable breast pads but should change them whenever they become damp. Even slightly sore nipples should not be ignored. Brief periodic exposure to a sun (ultraviolet) lamp, placed at least 18 inches from the skin for 10–15 minutes, and increased exposure to the air will promote healing.

Engorgement

A critical time for close nursing supervision of the new mother and nursing infant presents itself if the mother's milk comes in abruptly. Engorgement may bring about hard, full breasts at which the baby finds it difficult or impossible to nurse. It is tempting to provide a nipple shield in the hope that the baby will be able to grasp the rubber projection. Although this may be attempted as a last resort, too often the baby refuses to take it. The frustrated, crying baby in such circumstances increases the mother's anxiety and inhibits her let-down. A better approach is to help the mother express milk manually until the area around the nipple softens sufficiently for the infant to nurse directly from the mother.

If severe engorgement prohibits the baby from grasping the nipple, use of a rubber nipple shield should certainly be attempted. When engorgement is relieved, the shield should be immediately abandoned since the baby may become accustomed to it and reject the mother's nipple. An effective shield can sometimes be made from a formula bottle nipple. To encourage an unimpeded flow of milk from the lacteal sinuses, the base of the rubber nipple should be large enough to cover the mother's areola. The Woolwich Shield has been reported to be effective when worn between feedings if engorgement flattens or depresses the nipples.* The shield is constructed of two plastic cups which fit together but can be separated for cleaning. The shields may be worn beneath the brassiere for as long as necessary to achieve nipple projection. The wearer must generally build up her tolerance by wearing the shields a few hours a day until she can wear them comfortably all day. An additional aid to the severely engorged mother is an oxytocin nasal spray, which may be prescribed by the physician to produce a let-down of milk and decrease tension in the breast.

For several nursings after a mother has been engorged, the nurse should examine the mother's breasts and nipples and report any signs of cracking, discomfort or renewed engorgement. The baby should be encouraged to nurse with increased frequency (at least every 2–3 hours) night and day to prevent over-strenuous sucking and to keep the breasts from becoming too full.

* Communication from E. Reese, Pacific Grove, California.

Prevention of engorgement is preferable to treatment and is generally possible with good management. Rooming-in is the best preventive measure. When mother and infant are together around the clock, engorgement rarely occurs since the baby can nurse in response to his mother's needs as well as his own. If rooming-in is not available, the infant should be taken to his mother as soon as her breasts begin to fill and as often thereafter as is necessary to maintain her comfort. The mother's request for the baby should be met with friendliness. Although the baby may not nurse if he is not hungry, the mother may be assured that he soon will be. Meanwhile, ice bags applied to the breasts, under doctor's order, will allay engorgement and provide temporary relief.

If nursing management of lactation has been meaningful, by the time mother and child are ready for dismissal from the hospital breast and nipple problems should be successfully solved if, indeed, any have occurred.

Attitude of the Nurse

No discussion of nursing care of the lactating mother is complete without some consideration of the nurse's attitude and feelings about the breast and the nursing act. One study[6] indicates that intervention by physician or hospital or both was the second leading cause reported by mothers for premature cessation of breastfeeding.

Although knowledge of techniques is important, perhaps the most valuable service the nurse can offer the nursing mother is an attitude of approval and confidence in the woman's ability to nourish her infant in her chosen way. A smile of approval, a relaxed and pleasant approach will often provide the necessary extra measure of self-confidence which a mother may need to initiate breastfeeding successfully.

I believe that reaction against Victorian prudery has brought about an espousal of the breast as a sexual symbol in today's culture. The success of this espousal during the past half-century has dealt a severe blow to understanding the primary function of the breast. The breast was, is, and will likely continue to be physiologically and anatomically designed as a feeding organ. Any other function, in fact, is only secondary.

Despite (or perhaps because of) this misconception, the nursing act has come to be looked on by many—even nurses and others in the health fields—as "animal-like," "embarrassing" or "dirty." Unfortunately, this attitude is not entirely incomprehensible in view of the repeated sexual innuendos which the breast and suckling evoke.

As a member of a health-oriented profession the nurse, however, is in a position to evaluate objectively and arrive at factual conclusions. It is appropriate and important that she learn the *facts* about breastfeeding. In doing so, it seems likely that she will recognize that breastfeeding is generally best for the infant and mother. The sequela to this, then, may be an increased desire to render effective service to the mother who elects to nurse.

Dyal and Kahrl[7] point out the nurse's aura of authority with the breastfeeding mother. This "aura" remains whether the nurse is knowledgeable or not; hence, incomplete and erroneous advice to an inexperienced nursing mother can be damaging not only to the nursing couple but to the nurse–patient relationship, as well.

The following steps would seem to be both logical and helpful for the nurse who works with the breastfeeding mother:

1. **Self-evaluation.** If her feelings about the nursing act are negative, an attempt at self-understanding should be made. Recognition of the reasons for her feelings should lead to a diminution of their intensity and an increased desire to fulfill her nursing role.
2. **Self-education.** By learning the advantages and techniques of breastfeeding, she can begin to prepare herself to render appropriate nursing care with an accepting attitude.
3. **Self-activation.** The nurse who avoids the breastfeeding situation often does so because of fear of her own incompetence. Repeated efforts to help the nursing mother, perhaps initially observing and assisting a more experienced nurse, may enable her to discover her own potential.

Such efforts will not only improve the quality of her care of the postpartal patient but will also

SPECIAL ARTICLE

MATERNAL ATTACHMENT

Importance of the First Post-Partum Days

MARSHALL H. KLAUS, M.D., RICHARD JERAULD, B.S., NANCY C. KREGER, B.S.,
WILLIE MCALPINE, B.S., MEREDITH STEFFA, B.S., AND JOHN H. KENNELL, M.D.

Abstract To determine whether present hospital practices may affect later maternal behavior, we placed 28 primiparous women in two study groups shortly after delivery of normal full-term infants. Fourteen mothers (control group) had the usual physical contact with their infants, and 14 mothers (extended contact) had 16 hours of additional contact. Mothers' backgrounds and infants' characteristics were similar in both groups. Maternal behavior was measured 28 to 32 days later during a standardized interview, an examination of the baby and a filmed bottle feeding. Extended-contact mothers were more reluctant to leave their infants with someone else, usually stood and watched during the examination, showed greater soothing behavior, and engaged in significantly more eye-to-eye contact and fondling. These studies suggest that simple modification of care shortly after delivery may alter subsequent maternal behavior.

IN certain animals such as the goat, cow and sheep, separation of the mother and infant immediately after birth for a period as short as one to four hours often results in distinctly aberrant mothering behavior, such as failure of the mother to care for the young, butting her own offspring away and feeding her own and other infants indiscriminately.[1,2] In contrast, if they are together for the first four days and are then separated on the fifth day for an equal period, the mother resumes the protective and mothering behavior characteristic for her species when the pair is reunited. Thus, there is a special period immediately after delivery in the adult animal. If the animal mother is separated from her young during this period, deviant behavior may result. An early short period of separation does not produce as severe a distortion of mothering behavior in all species.[3]

In recent years several investigators have studied whether a similar phenomenon occurs in mothers of premature infants.[4,5] Does the prolonged separation experienced by the mother of a premature infant affect the formation of her affectional bonds and change her mothering behavior months and years after the delivery? Early results from these studies suggest that the long period of physical separation common in most nurseries may adversely affect maternal performance of some women.

Studies of human mothers of premature infants necessarily differ in design from the classic studies of separation in the animal mother. The gestation of the mothers is severely shortened, the infant is small and appears fragile, the period of separation after birth is greatly extended, and it has not been possible to provide close physical contact immediately after birth similar to the natural human and animal situation.

In most nurseries in the United States, however, even full-term mothers are separated from their infants for a short, but possibly important time. Thus, it seemed essential to determine whether present hospital practices for the mother of a full-term infant influence later maternal behavior. This report tests the hypothesis that there is a period shortly after birth that is uniquely important for mother-to-infant attachment in the human being.

MATERIAL AND METHODS

We placed each of 28 primiparous mothers of normal full-term infants in one of two study groups, depending on the day of delivery. Neither group knew of this study in advance or to our knowledge was aware of the arrangements made for the other. (The mothers, however, were not questioned on this subject.) The 14 mothers in the control group had

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the traditional contact with their infants: a glimpse of the baby shortly after birth, brief contact and identification at six to 12 hours, and then visits for 20 to 30 minutes every four hours for bottle feedings. In addition to this routine contact, the 14 mothers in the extended-contact group were given their nude babies, with a heat panel overhead, for one hour within the first three hours after birth, and also five extra hours of contact each afternoon of the three days after delivery. (A heat panel was also placed over the control mothers' beds for one hour during the first three hours.)

To eliminate any influence from the enthusiasm or interest of the nurse that might obscure the results, the special nurses who cared for the mothers during the extended-contact period (five hours per day) spent an equal amount of time with the control mothers. After an initial standardized introductory statement they only answered questions, did not instruct any of the women in caretaking unless this was requested, and most of the time were available just outside the room.

The mean age, socioeconomic and marital status, color, premedication, sex of the infant and days hospitalized in both groups were nearly identical (Table 1). Only mothers who intended to keep their infants and to bottle-feed them were admitted to the study. The mean birth weights of the two groups of infants differed by 110 g.

Second question: "Have you been out since the baby was born, and who sat?" A score of 0 was given for "yes," and if the mother felt good and did not think about her infant while she was out. A score of 3 was given if she did not go out or leave the baby with anyone, or if she did go out but thought constantly about the baby.

The third question related to spoiling and could not be scored.

A second measure of maternal behavior was the observation of the mother during a standardized examination of her infant. A score of 3 was allotted if, during the examination of the infant, she was standing by the pediatrician and watching continuously; a score of 0 was given if she remained seated and looked elsewhere. We also noted whether or not the mother attempted to soothe the baby when it cried. If she did not interact with the baby, she was given a score of 0; if she was consistently soothing, she was given a score of 3. The scoring of the interview and observation of maternal performance was then determined by independent raters who did not know to which group the mothers belonged.

To study maternal behavior in another situation, we made time-lapse films of the mothers feeding their infants. They all knew they were being photographed and were told to spend as much time as they wished. Filming was done through a one-way mirror for 15 minutes at a speed of 60 frames per minute. Mothers' and babies' reactions could then

Table 1. Clinical Data for 14 Mothers in the Extended-Contact and 14 in the Control Group.

Group	Maternal Characteristics				Mean Score*			Nurses' Time	Hospital Stay	Mean Birth Weight	No. of Infants	
	Age	Married	N†	W‡	A	B	C				M§	F¶
	yr	*no. of mothers*						*min/day*	*days*	*g*		
Extended contact	18.2	4	13	1	6.7	6.7	4.9	13	3.8	3184	6	8
Control	18.6	5	13	1	6.5	6.9	4.9	14	3.7	3074	8	6

*In this (Hollingshead) scoring system, on a scale of 1 to 7, residence (A) of 7.0 = poorest housing, occupation (B) of 7.0 = unskilled workers, & education (C) of 5.0 = reaching 10th to 11th grade in high school.
†Negro. ‡White. §Male. ¶Female.

To determine if this short additional time with the infant early in life altered later behavior, we asked the mothers to return to the hospital a month after delivery for three separate observations. These observations were made between the 28th and 32d post-partum days and consisted of a standardized interview, an observation of maternal performance during a physical examination of the infant and a filmed study of the mother feeding her infant.

The first seven questions on the interview concerned the general health of the infant, such as the number of stools and the amount of milk taken. Three separate questions were related to caretaking and were scored 0, 1, 2, 3.

First: "When the baby cries and has been fed, and the diapers are dry, what do you do?" A score of 0 was given for letting the baby cry it out, and 3 for picking up the baby every time. An intermediate score was given for gradations of behavior.

be analyzed in detail at one-second intervals. Each frame of the first 600 was scored by analyzers who did not know which group the mothers were in. We analyzed each frame for 25 specific activities, ranging from caretaking skills (such as the position of the bottle) to measurements of maternal interest and affection such as "en face" (defined as when the mother aligned her face in the same vertical plane of rotation as the infant's[6]), whether the mother's body was touching the infant's trunk, and whether she fondled the infant. (We defined fondling as any active spontaneous interaction initiated by the mother not associated with feeding, such as stroking, kissing, bouncing or cuddling.) Inter-observer reliability coefficients were calculated for the individual behaviors. The average of the reliability coefficients was 0.83 for "en face" and 0.99 for fondling.

Results

Analysis of the interview data is shown in Figure 1. The extended-contact group (the solid black bars) had scores of 2 and greater, whereas the control mothers (cross-hatched bars) were at the lower end of the scale. The chance of this occurrence is less than 0.05, with the use of the Mann–Whitney U-test.[7]

The two groups scored differently on the results of the observations during the physical examination (Fig. 2). The extended-contact group did not score below 3, whereas the scores of the control mothers were distributed from 1 to 6 (p less than 0.02).

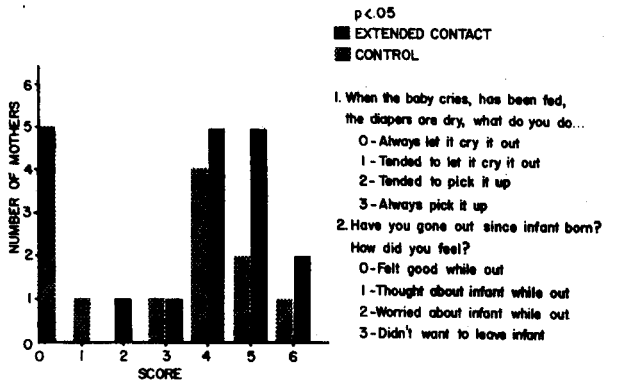

Figure 1. Maternal Scores from a Standardized Interview at One Month.

When the scores on the interview questions and the observations made during the examination are combined (Fig. 3), there is a separation of the scores of the two groups of mothers. The controls have scores of 2 to 10 spread out over the entire range, whereas mothers in the extended-contact group have scores ranging from 7 to 12 (p less than 0.002).

Figure 4 indicates the fondling and "en face" scores for both groups of mothers. Although the

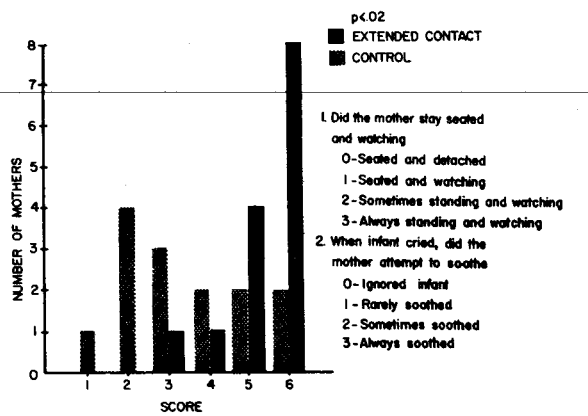

Figure 2. Scored Observations of the Mother Made during a Physical Examination of Her Infant at One Month.

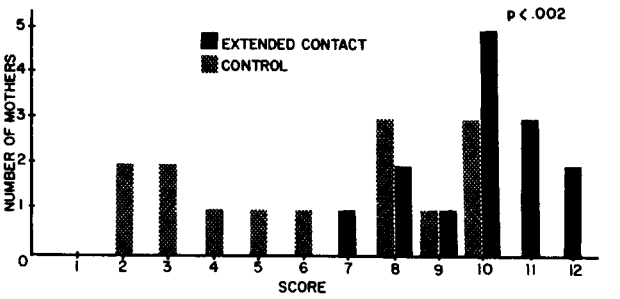

Figure 3. Summation of Scores of Performance from Both the Interview and the Observation of the Mother during an Office Visit at One Month Post Partum.

amount of time the mothers were looking at their babies was not significantly different in the two groups, the extended-contact mothers had significantly greater "en face" and fondling (11.6 per cent and 6.1 per cent of the total scored time, as compared to 3.5 per cent and 1.6 per cent in the control group). There were no significant differences in measures of caretaking, although the bottle was held away from the perpendicular more often in the control group. By all three measurements studied, differences between the two groups of mothers are apparent.

Discussion

It is surprising with the multitude of factors that influence maternal behavior[8] (such as the mother's genetic and cultural background, her relations with

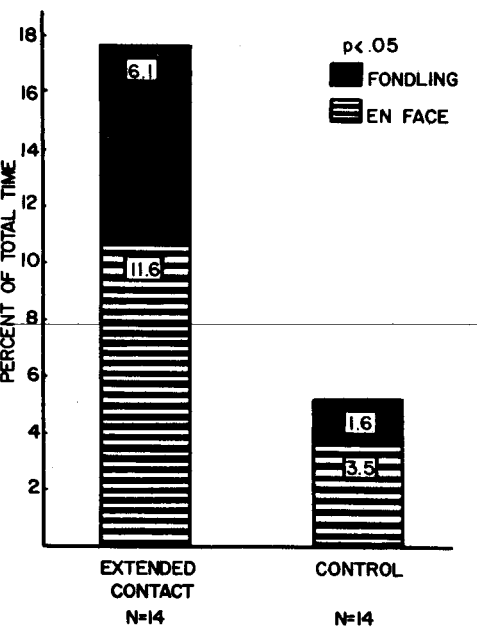

Figure 4. Filmed Feeding Analysis at One Month, Showing Percentage of "en Face" and Fondling Times in Mothers Given Extended Contact with Their Infants and in the Control Group.

GUIDELINES For MATERNAL CARE

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