
CURRENT THERAPY IN OBSTETRICS AND GYNECOLOGY

QUILLIGAN

一九八二年九月廿一日



CURRENT THERAPY IN OBSTETRICS AND GYNECOLOGY

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1980

W.B. SAUNDERS COMPANY
Philadelphia London Toronto



W. B. Saunders Company: West Washington Square
Philadelphia, PA 19105
1 St. Anne's Road
Eastbourne, East Sussex BN21 3UN, England
1 Goldthorne Avenue
Toronto, Ontario M8Z 5T9, Canada

Library of Congress Cataloging in Publication Data

Quilligan, Edward J

Current therapy in obstetrics and gynecology.

1. Gynecology. 2. Generative organs, Female.—Diseases.
3. Pregnancy, Complications of. 4. Therapeutics.
I. Title.

RG125.Q53 618 79-65461

ISBN 0-7216-7414-3

Current Therapy in Obstetrics and Gynecology

ISBN 0-7216-7414-3

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Last digit is the print number: 9 8 7 6 5 4 3 2 1

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Preface

Several years ago Dr. Howard Conn asked me to write one of the articles for *Current Therapy*. At the time I thought this was a unique idea, an opportunity for an individual in practice to have, in a relatively few minutes' reading time, the thinking of an expert in the field about the ideal therapy for a particular problem. Of course, brevity in these articles demands certain sacrifices; it precludes a discussion of the pathophysiologic basis of the problem under consideration and a description of the diagnostic steps that are necessary before a specific therapy is decided upon. The assumption is made that these are known; if they are not, then a proper basis for therapy is not present, and one must achieve this base by consultation with other excellent texts in the field. In this first edition of *Current Therapy in Obstetrics and Gynecology*, I have attempted to follow the basic precepts developed by Dr. Conn.

The articles in the book are organized by sections addressing the areas logically covered by our specialty and areas outside our specialty that impinge on it significantly. The divisions are obstetrics, general gynecology, endocrinology, oncology, neonatology, and general medicine.

Some topics may perhaps have been inadvertently omitted in this first edition, particularly in the area of general medicine. Those missing topics will be primary concerns for expansion in future editions, and I would welcome any suggestions you may have concerning needed material in a second edition. I do hope that this edition will give you reading enjoyment and will be of practical use in the treatment of your patients.

I would like to thank all the busy obstetricians and gynecologists, pediatricians, and internists who took their valuable time to contribute to this book.

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OBSTETRICS
Section I

ACUTE HYPERTENSION IN PREGNANCY

FREDERICK P. ZUSPAN, M.D.

Acute hypertension in pregnancy is a condition that is also known as pregnancy-induced hypertension, toxemia of pregnancy, and preeclampsia-eclampsia. All of these terms mean the same thing and are interchangeable. The most current and most commonly used term is "pregnancy-induced hypertension" (PIH).

Severe complications of pregnancy-induced hypertension are totally and completely preventable and should never occur. The most severe forms have a perinatal mortality that exceeds 30 per cent and a maternal mortality of 10 per cent. The following therapy regimen is based upon our past experience in treating severe pregnancy-induced hypertension and eclampsia. With this treatment protocol, our fetal salvage rate exceeds 90 per cent and the maternal mortality rate is zero. When this methodology was first published in 1964, it was identified as the best salvage rate for eclampsia in the world literature, and after 16 years it continues to be so. The common denominators of therapy are a pharmacological amount of magnesium sulfate and the relative absence of other medications, except for antihypertensive medications to prevent a stroke. Diuretics, narcotics, tranquilizers, and other sedatives are *not* used.

TREATMENT

The key to treatment is to identify the patients that are prone to develop pregnancy-induced hypertension and to see them at weekly intervals. These high-risk patients are often young and have the following characteristics: (1) primigravida; (2) low socioeconomic class; (3) low level of education; (4) poor nutrition; and (5) associated contributing factors, such as diabetes, renal disease, chronic hypertension, multiple pregnancy, or hydramnios.

Specific therapy is as follows: (1) Insist on a nutritious diet that is high in protein (1.5 to 2 gm. per kg. of body weight per day); (2) insist on the patient's resting in bed for at least 1 1/2 hours on her side at noon every day; and (3) perform the roll over test on each weekly visit from the twenty-second week of pregnancy.* If the roll over test is negative, reassurance

*The roll over test: The patient lies on her side until the blood pressure taken on her upper arm is stable. Then have the patient roll onto her back and take the blood pressure again and repeat in 5 minutes. If the diastolic pressure increases by more than 20 mm. Hg, the test is positive.

can be given that the patient will not develop preeclampsia; however, between 60 to 90 per cent of the patients who have a positive roll over test and are essentially asymptomatic will go on to develop overt pregnancy-induced hypertension at a later time.

MANAGEMENT DICTUMS

The following dictums should be considered in the management of patients with acute hypertension: (1) a hospitalized patient under therapy should never have a convulsion; (2) magnesium sulfate, given intravenously, prevents convulsions; (3) therapy should concern both the mother and the fetus, and neither should be harmed by overzealous treatment or poor obstetric judgment; and (4) eclampsia does not cause residual cardiovascular or renal damage in the mother.

MILD PREECLAMPSIA

Once a diagnosis is made, admit the patient to the hospital, thus removing her from the environment that made her ill. Place the patient at complete bed rest on her side with a nutritious diet. Administer a sedative (phenobarbital, 30 mg. three times daily) to make her appreciate bed rest more. Expect a diuresis within 36 to 48 hours after admittance to the hospital that will decrease the patient's weight by more than 2 kg. Signs and symptoms should abate in 3 to 5 days. Consider induction of labor if the patient is at or near term.

SEVERE PREECLAMPSIA

Assume that this individual is seriously ill and that there is a potential for death to both mother and fetus. Admit the patient to the hospital. Place her at bed rest with bathroom privileges only and a daily weight reading. Based on laboratory studies, put special emphasis on frequent blood pressure readings. Examine deep tendon reflexes at periodic intervals (every 2 hours), as these are usually hyperactive. The major therapy is administration of magnesium sulfate, which may be performed either intravenously or intramuscularly. We prefer the intravenous route, since it is more appropriate for the disturbed pathophysiology and less painful. Magnesium sulfate is administered by an infusion pump or some controlled administrative system. Four gm. of magnesium sulfate are given *slowly* over a period of 20 minutes as a loading dose, then 1 gm. per hour is given thereafter. The patient is monitored by exami-

nation of: (1) deep tendon reflexes, which should always be present but hypoactive; (2) urinary output, which should exceed 25 ml. per hour; and (3) respirations, which should not decrease below 10 per minute. The therapeutic blood level is between 6 to 8 meq. per liter. Consider magnesium sulfate a dangerous drug, since it can, in toxic doses, depress respiration as well as cause cardiac arrest. Magnesium sulfate is excreted principally by the kidneys, hence if urinary output is diminished, the dose of magnesium sulfate should be diminished. An overdose of magnesium sulfate can be counteracted by the administration of calcium chloride intravenously (1 gm.). I have always felt that the major protective mechanism of magnesium sulfate is to increase uterine blood flow and enhance fetal welfare. Magnesium sulfate is not a hypotensive agent; however, it will help level off fluctuations in blood pressure. The magnesium crosses the placenta easily, and the level in the mother is the same as that in the fetus. If the diastolic blood pressure is greater than 100 mm. Hg, hydralazine (Apresoline) should be given as outlined in the section on "Eclampsia."

DECISION FOR DELIVERY

Most ill patients with severe pregnancy-induced hypertension should be delivered of their offspring. Unless the fetus weighs less than 1500 gm., the vaginal route is preferable, and most patients are easily induced with small doses of oxytocin given by infusion pump. Electronic fetal heart rate surveillance is essential.

ECLAMPSIA

Assume that the patient with eclampsia has a 10 per cent chance of dying and the fetus a 35 per cent chance. An aggressive action-oriented program that is the same as the one just outlined for severe preeclampsia should be instituted. Once the patient is under good control, which should take no more than 2 hours, a decision should be made about delivery. Convulsions are controlled with magnesium sulfate and not with other drugs. Diuretics are not used, and antihypertensive therapy should be instituted to prevent a stroke. A diastolic pressure of greater than 100 mm. Hg is considered excessive. The antihypertensive of choice is hydralazine given first with a 5 mg. bolus injection and followed thereafter by administration using an infusion pump, with the hydralazine being put in a plastic bag and the blood pressure

being controlled by the level of the drug. The diastolic pressure should not go below 80 mm. Hg.

ANESTHESIA FOR DELIVERY

Major conduction anesthesia, i.e., spinal anesthesia, is contraindicated in all forms of pregnancy-induced hypertension, as this will further decrease uterine blood flow and may be harmful to the fetus. The usual form of anesthesia is a pudendal block of local infiltration supplemented with nitrous oxide. All of our patients, if they can swallow, receive a chilled antacid every 3 hours to assist in neutralization of gastric acidity and prevent aspiration pneumonia if aspiration does occur.

CHRONIC HYPERTENSION IN PREGNANCY

FREDERICK P. ZUSPAN, M.D.

Pregnancy tends to provoke the unmasking of chronic hypertensive disease, hence pregnancy is known to be hypertensogenic. Chronic hypertension is seen more commonly in the multigravid individual, but a primigravida cannot be totally excluded. Chronic hypertension usually antedates pregnancy or is seen before the 24th week of pregnancy. One of the most dreaded complications of chronic hypertension in pregnancy is the development of superimposed preeclampsia, since patients with this set of problems have the highest maternal and fetal morbidity and mortality.

Chronic hypertension during pregnancy presents a hazard for both mother and fetus. Fetal wastage in patients with mild hypertension is 16 per cent, whereas in those with severe hypertension (greater than 160/100), fetal wastage is 40 per cent. It has been shown that fetal morbidity and mortality are directly related to the severity of maternal hypertension and intercedent complications during pregnancy.

CLASSIFICATION

Chronic hypertension can be primary or secondary. Primary hypertension is not attributable to a specific disease and is often termed "essential vascular hypertension." Ninety-five per cent of the hypertensive patients seen have this type. The secondary type is hypertension that is secondary to a known disease process. It