

HOSPITAL PHYSICIAN®

NEPHROLOGY BOARD REVIEW MANUAL

PUBLISHING STAFF

PRESIDENT, GROUP PUBLISHER

Bruce M. White

EDITORIAL DIRECTOR

Debra Dreger

SENIOR EDITOR

Bobbie Lewis

EDITOR

Robert Litchkofski

ASSISTANT EDITOR

Rita E. Gould

EDITORIAL ASSISTANT

Kara V. Warner

EXECUTIVE VICE PRESIDENT

Barbara T. White, MBA

EXECUTIVE DIRECTOR OF OPERATIONS

Jean M. Gaul

PRODUCTION DIRECTOR

Suzanne S. Banish

PRODUCTION ASSOCIATES

Tish Berchtold Klus

Mary Beth Cunney

PRODUCTION ASSISTANT

Stacey Caiazza

ADVERTISING/PROJECT MANAGER

Patricia Payne Castle

MARKETING MANAGER

Deborah D. Chavis

NOTE FROM THE PUBLISHER:

This publication has been developed without involvement of or review by the American Board of Internal Medicine.



The Association for Hospital Medical Education endorses HOSPITAL PHYSICIAN for the purpose of presenting the latest developments in medical education as they affect residency programs and clinical hospital practice.

Pregnancy and Renal Disease

Series Editor:

Stanley Goldfarb, MD, FACP

Professor of Medicine

Interim Chairman

Department of Medicine

University of Pennsylvania School of Medicine

Philadelphia, PA

Contributor:

Sylvia E. Rosas, MD

Assistant Professor of Medicine

Renal, Electrolyte, and Hypertension Division

University of Pennsylvania School of Medicine

Philadelphia, PA

Table of Contents

Introduction	2
Hypertension and Preeclampsia	2
Preexisting Renal Disease and Pregnancy	5
Hemodialysis	8
Urinary Tract Infection	9
References	10

Cover Illustration by Scott Holladay

Copyright 2002, Turner White Communications, Inc., 125 Strafford Avenue, Suite 220, Wayne, PA 19087-3391, www.turner-white.com. All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, mechanical, electronic, photocopying, recording, or otherwise, without the prior written permission of Turner White Communications, Inc. The editors are solely responsible for selecting content. Although the editors take great care to ensure accuracy, Turner White Communications, Inc., will not be liable for any errors of omission or inaccuracies in this publication. Opinions expressed are those of the authors and do not necessarily reflect those of Turner White Communications, Inc.

Pregnancy and Renal Disease

Sylvia E. Rosas, MD

INTRODUCTION

Pregnancy normally causes a variety of anatomic and physiologic changes in the kidney, including dilatation of the urinary system and an increased glomerular filtration rate (GFR). Moreover, pregnancy has a profound effect on renal function and may play a major role in the natural history of preexisting renal diseases. Preexisting renal disease may complicate the course of pregnancy and may significantly increase the risk for adverse maternal and fetal outcomes. Risk factors such as severe proteinuria, uncontrolled blood pressure, and severe renal impairment worsen fetal and maternal outcome. On the other hand, acute renal failure in several forms may complicate a pregnancy in a previously healthy woman. This manual reviews important steps in the diagnosis of preeclampsia and other forms of hypertension associated with pregnancy; the causes of acute renal failure in the pregnant woman; and issues regarding the management of preexisting renal disease and urinary tract infection in pregnant women.

HYPERTENSION AND PREECLAMPSIA

CASE PATIENT I

Presentation

A 19-year-old primiparous woman with an intrauterine pregnancy presents to her obstetrician for prenatal care during the first trimester. At this initial visit, her blood pressure is 160/90 mm Hg. At subsequent visits during her first trimester, she has blood pressure readings of 150/110 mm Hg and is placed on methyldopa 250 mg orally twice daily.

- What physiologic changes occur in pregnancy?
- What are the implications of these changes on blood pressure and renal function?

PHYSIOLOGIC CHANGES IN PREGNANCY

Beginning early in pregnancy there are dramatic increases in cardiac output and sodium and water reten-

tion, leading to blood volume expansion. At the same time, systemic vascular resistance and systemic blood pressure decrease substantially. These phenomena peak in the second trimester, and then plateau until pregnancy is concluded. The teleologic rationale for such an expansion of the blood volume is complex. Certainly the development of the fetus requires the accumulation of several liters of fluid and electrolytes to supply normal body function, and the blood volume that resides in the placenta also must be provided. In addition, some extra “reserves” are probably beneficial so that at the time of delivery, incremental blood and fluid losses do not lead to shock. The expansion of the plasma volume is accompanied by a lesser increase in red cell volume, leading to a small drop in the hematocrit.

These physiologic changes can produce substantial stress in patients with compromised cardiac or renal function, whose capacity to accommodate the additional 8 to 10 L of body water and 1 mEq of sodium may be quite limited. Despite the increase in cardiac output, blood pressure normally falls by 8 to 10 mm Hg during pregnancy due to the reduced systemic vascular resistance in the face of rising blood volume. The mean blood pressure in the second trimester is 105/60 mm Hg.¹ The fall in resistance and blood pressure may be due to increased nitric oxide synthesis² as well as to resistance to a variety of vasoactive substances, including catecholamines, endothelins, and angiotensin.³

Along with the changes in blood volume and blood pressure, there is a dramatic rise in the GFR and renal plasma flow during pregnancy. The increased GFR and renal blood flow also are a logical consequence of the rising blood volume and the need to filter an increased volume of water and solute content of the body fluids and to deal with the metabolic end products of the fetus. These effects begin early in pregnancy and may reach a value 50% higher than baseline by the third month of pregnancy.^{4,5}

In addition to the physiologic changes that occur in pregnancy, the clinical markers used to assess renal function are altered. The serum creatinine and blood urea nitrogen (BUN) levels tend to be 20% to 30% lower than normal during pregnancy; the clinician must keep in mind that higher values could indicate