

Subarachnoid Hemorrhage: Causes and Cures

Bryce Weir

CONTEMPORARY NEUROLOGY SERIES

SUBARACHNOID HEMORRHAGE:

Causes and Cures

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New York • OXFORD UNIVERSITY PRESS • Oxford

Oxford University Press

Oxford New York

Athens Auckland Bangkok Bogotá Buenos Aires Calcutta
Cape Town Chennai Dar es Salaam Delhi Florence Hong Kong Istanbul
Karachi Kuala Lumpur Madrid Melbourne Mexico City Mumbai
Nairobi Paris São Paulo Singapore Tokyo Toronto Warsaw

and associated companies in

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Published by Oxford University Press, Inc.
198 Madison Avenue, New York, New York 10016

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Library of Congress Cataloging-in-Publication Data

Weir, Bryce.

Subarachnoid hemorrhage : causes and cures / Bryce Weir.

p. cm. — (Contemporary neurology series ; 52)

Includes bibliographical references and index.

ISBN 0-19-512875-3 (hardback)

I. Subarachnoid hemorrhage. I. Title. II. Series.

[DNLM: 1. Subarachnoid Hemorrhage—etiology. 2. Subarachnoid Hemorrhage—therapy. 3. Subarachnoid Hemorrhage—physiopathology. 4. Cerebral Aneurysm—complications. 5. Cerebral Aneurysm—therapy.

W1 CO796N v. 52 1998 / WL 200 W425s 1998]

RC 394. H37W45 1998

616.8'1—DC21

DNLM/DLC

for Library of Congress

98-15924

CIP

1 3 5 7 9 8 6 4 2

Printed in the United States of America

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PREFACE

The entity of subarachnoid hemorrhage has captured the attention of neurologists and neurosurgeons throughout most of this century because, for the first time, an accurate diagnosis could be made and effective therapies administered. We now know that ruptured saccular aneurysms account for most bleeding of this kind. With CT scans and digital subtraction angiography, definitive diagnoses are usually possible. Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) have recently added to our diagnostic capabilities. Lumbar puncture is now only rarely performed. With the advent of the operating microscope, microsurgical techniques, a plethora of aneurysm clips, and safer neuroanesthesia, early surgery for ruptured aneurysms can often be performed safely. The last few years have also witnessed the introduction of endovascular therapeutic techniques that hold great promise.

In view of all this progress, it was a privilege to be asked to write this book for the Contemporary Neurology Series. I hope this monograph will be considered timely given the pace of acquisition of new knowledge. The consideration of economic and medicolegal aspects might initially seem surprising in this clinical work, but it is becoming more difficult to separate these topics from our daily practices. Failure to diagnose a subarachnoid hemorrhage can have such catastrophic consequences that it is viewed as a fertile field by malpractice attorneys.

As much as or more than any other neurological condition, a subarachnoid hemorrhage mandates the rapid and seamless collaboration of emergency room physicians, neurologists, neurosurgeons, interventional and diagnostic neuroradiologists, neuroanesthesiologists, intensivists, and pathologists. I hope this work will be valuable to all those working in these fields and to their students and residents.

A decade ago, I wrote *Aneurysms Affecting the Nervous System*, and I have used that text as a point of departure for this monograph, which is selective in reviewing the more recent literature. I also examine in detail the nonaneurysmal causes of subarachnoid hemorrhage. Not infrequently, I think that if I had only known 30 years ago what is in this book, I might have saved some of my patients who died from these cruel and frightening conditions.

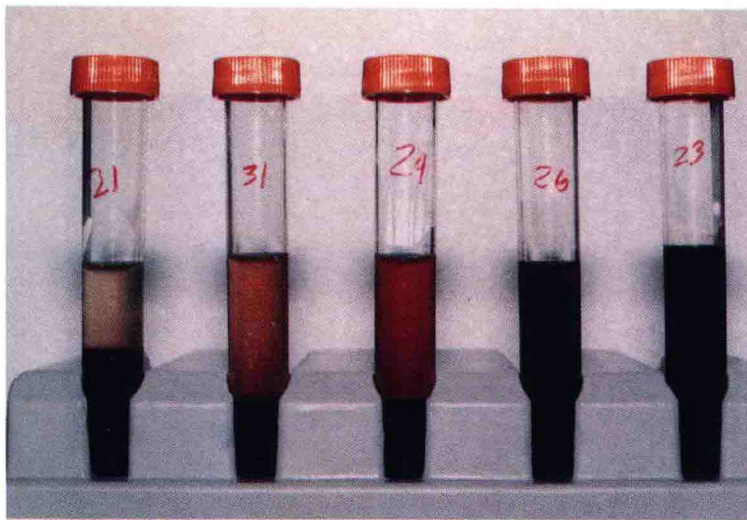
Many colleagues have aided me in this endeavor. Kenneth Petruk, Peter Allen, and Max Findlay were my partners in vascular neurosurgery at the University of Alberta. Lew Disney and Michael Grace collaborated with me on many aneurysm-related projects and papers. I am also deeply indebted to my colleagues at The University of Chicago, especially Loch Macdonald, who shares with me the care of the vascular cases. In addition, Ruth Ramsey, Saeid Mojtahedi, Jordan Rosenblum, Robert Wollmann, Chris Amidei, and Lydia Johns have been valued coworkers. I have also had the pleasure of collaborating over the years with Neal Kassell. Many friends in neurology and neurosurgery generously permitted the use of their material. My neurosurgical residents and nurses have always done the really hard work. My friends at the Brain Research Foundation of Chicago made possible the use of the colored illustrations. Sid Gilman, Editor-in-Chief of this series, and Bernice Wissler of the F. A. Davis Company made numerous and important suggestions and corrections. Marcus Stoodley, our vascular fellow, was a keen-eyed critic. Arlene

MacLean remained a typist without peer. Mary Lou Weir carried out the multiple reworkings of the text.

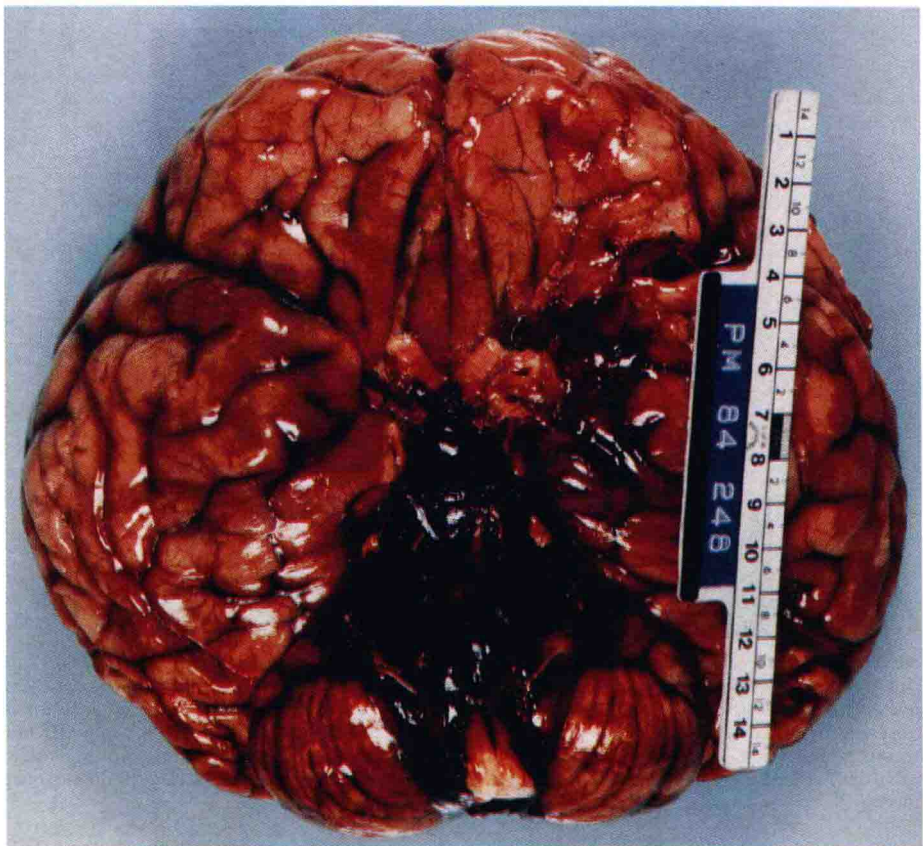
These people and others made this possible. Mary Lou, Pandi, Glynnie, and Brocke made it worthwhile.

Bryce Weir
Chicago, Illinois

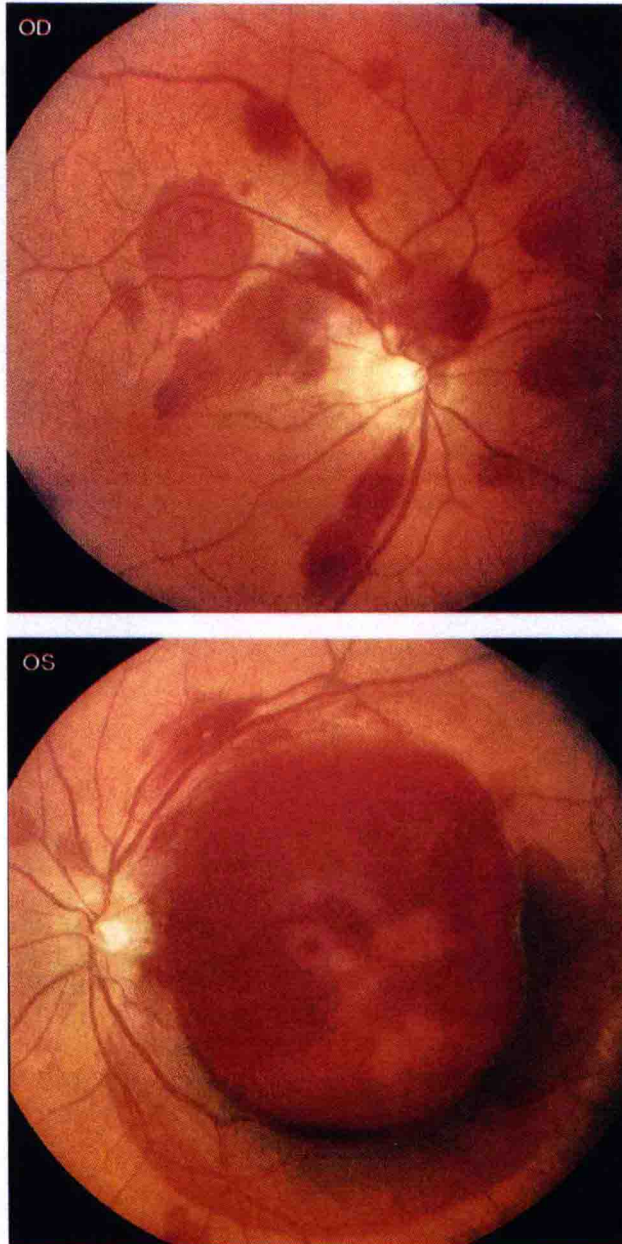
Color Plates



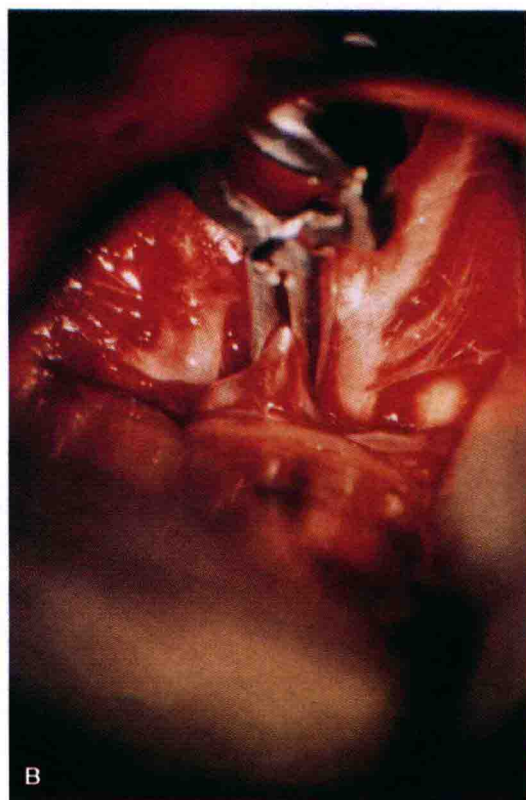
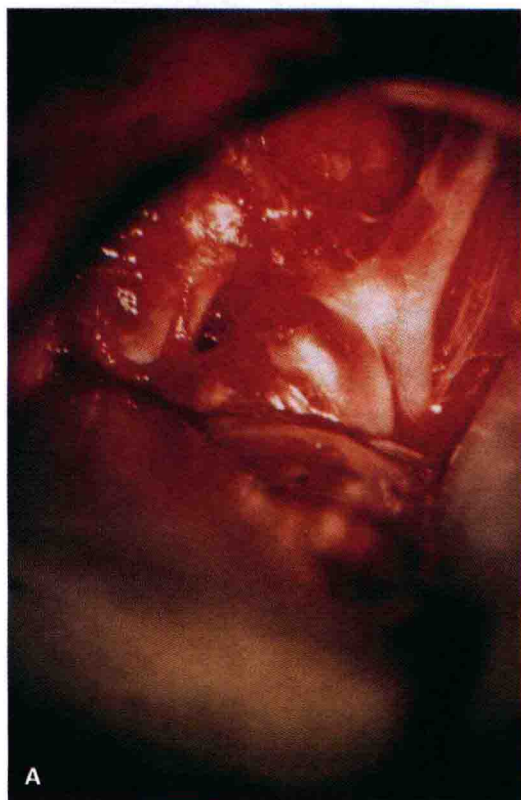
Color Plate A. Blood and artificial CSF were incubated at body temperature in the dark for (left to right) 0, 1, 2, 3, 4, and 5 days. With progressive lysis of erythrocytes there is a greater release of intracellular contents into the supernatant. This is a presumed explanation for the delayed and gradual onset of vasospasm.



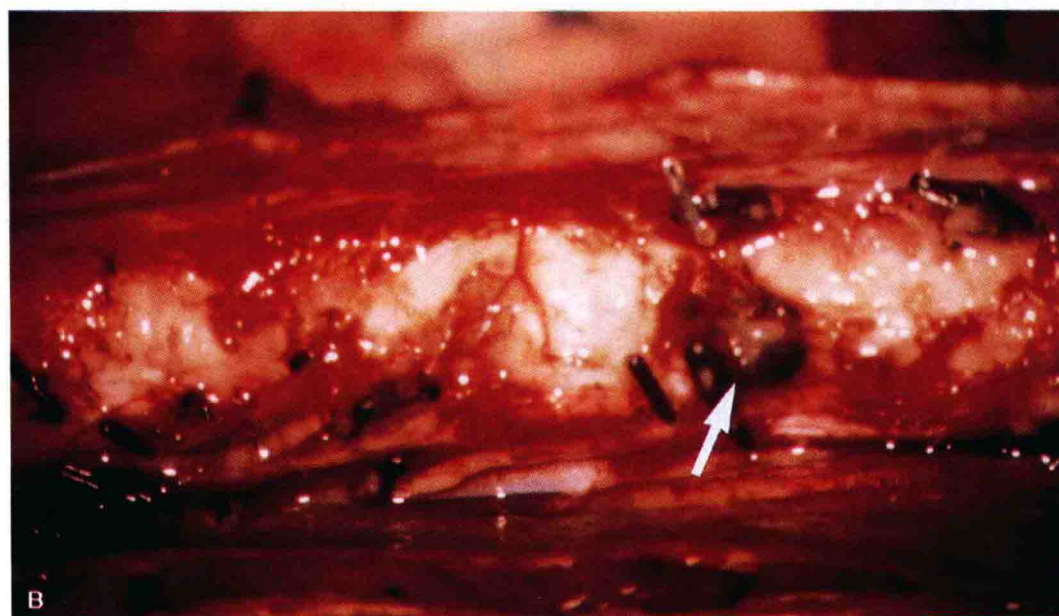
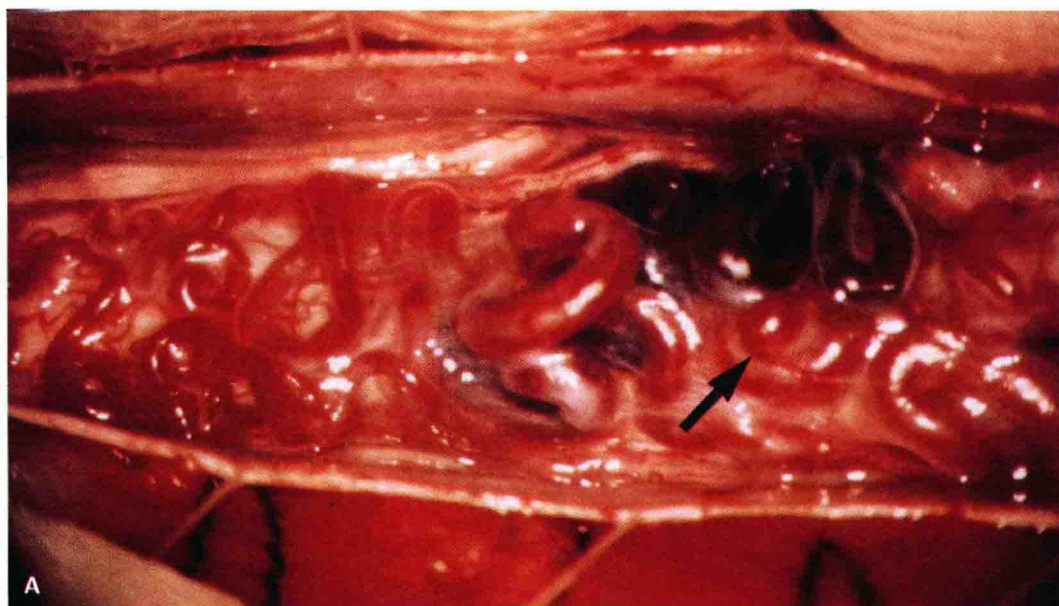
Color Plate B. Basal view of the brain following a massive subarachnoid hemorrhage from a basilar tip aneurysm. The thick clot formed in the basal cisterns is obvious, as is the diffuse cerebral swelling.



Color Plate C. **OD** (right eye): Flame-shaped and superficial retinal hemorrhages are scattered around the disc and posterior pole. **OS** (left eye): Extensive macular hematoma (preretinal or subhyaloid hemorrhage) is seen, as well as superficial retinal hemorrhages. Visual acuity was hand motion. The patient had a ruptured anterior communicating artery aneurysm. At 4 months follow-up, visual acuity 20/20 **OD**, 20/70 **OS**; fundusoscopic **OD** normal, **OS** severe vitreous hemorrhage. (From Toosi, SH and Malton, M: Terson's syndrome—significance of ocular findings. *Ann Ophthalmol* 19:7–12, 1987, p 8, by permission of the American Society of Contemporary Medicine, Surgery, and Ophthalmology.)



Color Plate D. (A) Anterior communicating aneurysm in a young boy, which was clipped the day of his hemorrhage. He was discharged home in normal condition within a week. His uncle had died in hospital of a rebleed while waiting for elective “delayed” surgery for his aneurysm. (B) Straight Yasargil aneurysm clip occludes the neck of the aneurysm flush with its origin from the anterior communicating artery.



Color Plate E. (A) A 37-year-old woman presented with sudden-onset leg weakness and numbness, back pain, and loss of bladder control. A spinal arteriovenous malformation (AVM) with subarachnoid hemorrhage was diagnosed. (B) After removal of the malformation, a thrombosed, associated aneurysm (*arrow*) was left embedded in the lower thoracic cord. The patient made a complete neurologic recovery.

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