Ischemic Preconditioning

Introduction

Antoine Hakim, MD, PhD; Roger Simon, MD

The quest for effective therapeutics for the treatment of stroke has resulted in more than a dozen clinical trials of putative neuroprotective drugs. None has proven successful. Although trial design may be a factor in these failures, the negative results of these attempts to find effective exogenous therapies for cerebral ischemia have stimulated research for endogenous modulators of neuroprotection. Such endogenous modulators produce the phenomenon of ischemic tolerance/preconditioning. Thus, stressors seem capable of producing a dose–response relationship in the brain where mild to severe preconditioning first produces the tolerance phenomenon and increasingly severe stress culminates in apoptosis and finally pan-

dose relationship in the brain where mild to severe preconditioning first produces the tolerance phenomenon and increasingly severe stress culminates in apoptosis and finally pan-

dose relationship in the brain where mild to severe preconditioning first produces the tolerance phenomenon and increasingly severe stress culminates in apoptosis and finally pan-

dose relationship in the brain where mild to severe preconditioning first produces the tolerance phenomenon and increasingly severe stress culminates in apoptosis and finally pan-

mammals, during hypoxic states. Examples include hibernation and hypoxia-tolerant states. Tolerance has been hypothesized to have a neuroprotective role in cerebral ischemia. More importantly, understanding tolerance may well lead to the development of small molecules or other methods of modulating the brain’s response to ischemia.

This session therefore addresses tolerance in the heart and the brain at the cellular level and at a genomic level. The clinical literature addressing the potential of transient ischemia to produce a preconditioning phenomenon is reviewed.

References

1. Dirnagl U, Simon RP, Hallenbeck JM. Ischemic tolerance and endoge-
6. Dahl NA, Balfour WM. Prolonged anoxic survival due to anoxia pre-


8. Janoff A. Alterations in lysosomes (intracellular enzymes) during shock; effects of preconditioning (tolerance) and protective drugs. Int Anes-


Received July 20, 2004; accepted August 5, 2004.
From the Canadian Stroke Network (A.H.), Ottawa, Canada; and the Dow Neurobiology Laboratory (R.S.), Portland Ore.
Correspondence to Dr Antoine M. Hakim, Canadian Stroke Network, Head, Neuroscience Research and University Chair, University of Ottawa, 451 Smyth Road, Ottawa, Ontario, Canada K1H 8M5. E-mail ahakim@ohri.ca

(Stroke. 2004;35[suppl I]:2675.)

© 2004 American Heart Association, Inc.

Stroke is available at http://www.strokeaha.org

DOI: 10.1161/01.STR.0000143238.24952.88

2675
Ischemic Preconditioning: Introduction
Antoine Hakim and Roger Simon

Stroke. 2004;35:2675; originally published online September 30, 2004;
doi: 10.1161/01.STR.0000143238.24952.88
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2004 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/35/11_suppl_1/2675

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/