and to a narrower lumen causing greater flow-induced shear stress, might itself actually cause the development of plaque rupture, platelet activation, and thrombus formation as a secondary phenomenon.

We certainly agree with Dr Golino that unstable angina is a complex syndrome with multiple precipitating mechanisms. However, Dr Golino has confused Dr Braunwald's different categories of unstable angina, which were proposed as a helpful clinical classification system that recognized the heterogeneity of the syndrome, with a true pathophysiological classification system. Clearly, Braunwald's classification has been helpful, but I am sure he would be the first to agree not that it will ultimately be replaced by one that is based on pathophysiological differences but that it is likely the clinical presentations will turn out to be epiphenomena that will probably only imprecisely reflect the differing etiologic mechanisms.

Our study was not designed to elucidate all of the mechanisms responsible for the precipitation of unstable angina or to correlate the clinical presentation with etiology. That will require a large multicenter effort. It was a very focused study; what it accomplished was to identify smooth muscle proliferation as an accompaniment and probable precipitating cause of unstable angina in a subgroup of such patients. We and other investigators are already attempting to determine the causes (undoubtedly multiple) of the SMC proliferation. We hope this effort, along with the efforts focusing on elucidating the mechanisms responsible for plaque rupture and the development of intracoronary thrombi, will ultimately lead to a comprehensive pathophysiological classification system that will have important therapeutic implications.

Moshe Flugelman, MD
Stephen E. Epstein, MD
National Institutes of Health
National Heart, Lung, and Blood Institute
Bethesda, Maryland

Interposed Abdominal Compression–CPR: Which Patients Are Benefited? Why?

Sack et al recently reported the results of a comparison of interposed abdominal compression–cardiopulmonary resuscitation (IAC–CPR) versus standard CPR (STD–CPR) on the return of spontaneous circulation and 24-hour survival for in-hospital episodes of cardiac arrest. The study was limited to patients in whom the initial rhythm was asystole or electromechanical dissociation: 143 patients were randomized, 67 received IAC–CPR, and 76 had STD–CPR.

Data analyses demonstrated statistically significant improvements in the frequency of return of spontaneous circulation (49% versus 28%) and 24-hour survival (33% versus 13%) with IAC–CPR versus STD–CPR. However, these overall group comparisons obscure important differences that are obvious when the outcome data for the diabetic patients are compared with those of the nondiabetic patients. In fact, the data (Fig 3, page 1697) demonstrate that only the diabetic patients were benefited by IAC–CPR, and the benefits were astounding: for return of spontaneous circulation, 78% with IAC–CPR and 14% with STD–CPR (P = .009) and for 24-hour survival, 56% with IAC–CPR and 1% with STD–CPR (P = .008). There were no statistically significant improvements in either outcome measure with IAC–CPR for nondiabetic patients. The authors make no mention of the fact that the benefits of IAC–CPR were limited to diabetic patients. On the contrary, they state that the presence of diabetes had no effect on either outcome measure; this deduction must have come from group comparisons of all diabetic patients (combining both types of CPR) with all nondiabetics. It would seem that the questions asked in the data analyses completely overlooked the diabetic subgroup results. (The editorial that accompanied the article also made no comment about the exclusive response to IAC–CPR in diabetics.)

One may only speculate as to why diabetic patients were dramatically benefited by IAC–CPR. There may have existed, perhaps, a decreased coronary vasoconstrictor tone in these patients due to a diabetic sympathetic neuropathy, thereby improving the effects of interposed compression on coronary flow. If this were the explanation, it would be important to try to transfer, through pharmacological intervention, the benefits of IAC–CPR in the diabetic patient to nondiabetics. One of the major benefits in the use of epinephrine in the CPR protocol is considered to be the improvement in coronary perfusion pressure that develops from thoracic compression in the presence of increased systemic vascular resistance (produced by epinephrine); β2-adrenergic-mediated coronary vasodilatation by epinephrine may also aid coronary blood flow. The question, then, is how coronary flow may be still further enhanced in the nondiabetic case.

In view of the established role of epinephrine in cardiac resuscitation, it would seem most reasonable to seek possible adjuncts. Use of an agent that would improve coronary vasodilatation and/or flow without markedly affecting systemic vascular resistance appears to be the objective. Dipyridamole will cause coronary vasodilatation at doses that spare arteriolar tone; the negative effects of this drug on coronary collateral flow in ischemic regions may be of relatively minor consideration at a time of asystole or electromechanical dissociation. An organic nitrate such as nitroglycerin might also be beneficial. The study of Paradis et al in human resuscitation efforts demonstrated that the pressure gradient between the aorta and the right atrium during the compression phase of CPR was strongly predictive of the return of spontaneous circulation; this gradient averaged 12.5 versus 2.5 mm Hg in those with and without return of spontaneous circulation, respectively. Nitroglycerin might be helpful in increasing this gradient because of its relatively greater venodilator potency compared with arteriolar dilation.

Other candidate drugs may be available that would help to mimic the vascular state of the diabetic patient, since that factor may be responsible for the marked advantage in the response to IAC–CPR in those patients.

Mario A. Inchiosa, Jr, PhD
Elizabeth A.M. Frost, MD
Departments of Pharmacology and Anesthesiology
New York Medical College
Valhalla, New York

References
Interposed abdominal compression-CPR: which patients are benefited? Why?
M A Inchiosa, Jr and E A Frost

Circulation. 1994;90:1113-1114
doi: 10.1161/01.CIR.90.2.1113

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1994 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/90/2/1113.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation 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 Circulation is online at:
http://circ.ahajournals.org/subscriptions/