The author replies:

To the Editor:

Dr. Hellstrom’s theories about vasospasm, although unproven, are interesting and provocative. I would certainly agree that further investigation of the role of such a mechanism in the pathogenesis of acute myocardial infarction is warranted. However, I would like to make it clear that our experimental model was not devised to determine the precise mechanisms of action of nitroglycerin in acute myocardial infarction and that the salutary effects of nitroglycerin we observed (with arterial pressure held constant by simultaneous administration of methoxamine) can be explained by “peripheral” actions, by “central” actions (not necessarily relief of vasospasm), or, more likely, by a combination of the two.

For example, we found in the experimental animal that nitroglycerin administered alone consistently diminishes left ventricular filling pressure, an effect only partially reversed when methoxamine is administered simultaneously at a rate to prevent nitroglycerin-induced hypotension (unpublished observations). These findings suggest that nitroglycerin-induced venodilatation is not fully reversed by doses of methoxamine that completely reverse the effects of nitroglycerin on the arterial system. Thus, if the decrease in filling pressure reflects a decrease in left ventricular volume, myocardial wall tension (and thereby MVO₂) would diminish even if arterial pressure were maintained constant by methoxamine infusion. Such a change would lead to a diminishment of ischemia caused by acute coronary occlusion. Apart from any influence on MVO₂, it also is possible that nitroglycerin diminishes ischemia in our preparation by increasing flow to ischemic myocardium — not by dilating coronary arteries in spasm (an unlikely consequence of mechanical occlusion of the proximal segment of the artery) — but by reducing resistance to coronary collateral flow. This could be accomplished by two actions of nitroglycerin. Collateral flow or flow within the ischemic bed during diastole could be increased indirectly by nitroglycerin-induced diminution in ventricular diastolic volume and pressure; the consequent decrease in wall tension would diminish resistance to flow through collateral channels and ischemic capillary beds. In addition, a direct vasodilator action of nitroglycerin on human coronary collateral channels was demonstrated recently by intraoperative studies of patients at the time of coronary artery bypass.

Finally, although Dr. Hellstrom has cited his own studies as substantiating a vasospasm mechanism during acute myocardial infarction, his data also can be explained by well-known autoregulatory mechanisms, rather than vasospasm.

In summary, I do not contend that vasospasm is not involved in the complex sequence of events occurring during acute myocardial infarction. Indeed, vasospasm may be an important mechanism in the rare patient with “variant” angina who goes on to infarct and is found to have normal coronary arteries. However, the purpose of my reply to Dr. Hellstrom’s letter is to point out that 1) our results with nitroglycerin can be entirely explained without invoking relief of vasospasm, 2) no experimental data yet published proves the existence of vasospasm during myocardial infarction, and 3) with the above rare exception, no clinical data can be pointed to which suggest that vasospasm is an important cause of acute myocardial infarction in man.

STEPHEN E. EPSTEIN, M.D.
National Heart and Lung Institute
Bethesda, Maryland

Mean Normalized Systolic Ejection Rates

To the Editor:

The recently published study by Peterson et al. is an important contribution to the analysis of left ventricular function in patients with and without depressed cardiac performance. The authors state that “the normal range for the mean normalized systolic ejection rate (MNSER) has not been previously reported.” Mean normalized systolic ejection rate (3.24 ± 0.47 SEM), however, has been previously reported and indeed the values reported previously have very close agreement with those reported by Peterson et al. (3.32 ± 0.18 SEM). It is interesting to note that MNSER in patients with abnormal cardiac function reported by Peterson et al. (1.04 ± 0.11 SEM) is also very similar to that reported previously (1.04 ± 0.47 SEM) in patients with cardiomyopathy.

KANU CHATTERJEE, M.B.
Cedars-Sinai Medical Center
Los Angeles, California 90029

References


Circulation, Volume 50, December 1974
Mean Normalized Systolic Ejection Rates
KANU CHATTERJEE

Circulation. 1974;50:1282
doi: 10.1161/01.CIR.50.6.1282-a

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 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/50/6/1282.2.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/