The author replies:
To the Editor:
The comments of Dr. Coelho are pertinent and the references quoted in his letter are important. In our review, Dr. Rao and I did not intend to present an exhaustive review of the literature.

The concept of functional similarity between various anatomic malformations was expressed earlier by the undersigned. Among references to this concept are the following:

EDWARDS JE: Functional pathology of certain cardiovascular malformations which may be treated surgically. Arch Surg 61: 1103, 1950


Dr. Rao and I regret omission of the references cited by Dr. Coelho but this was without an attempt to disregard important work and concepts of others.

JESSE E. EDWARDS, M.D.
United Hospitals-Miller Division
125 W. College Avenue
St. Paul, Minnesota

Lung Scanning and Angiography in Pulmonary Embolism
To the Editor:
With reference to the article 'The complementary roles of chest radiography, lung scanning and angiography in the diagnosis of pulmonary embolism' (Circulation 49: 179, 1974) by Moses et al., we would like to submit some of our data based on a similar study and published by our group.1 Although not cited in their paper, our publication was the first to describe the specificity of segmental perfusion defects in pulmonary embolism. Our figure for the specificity of segmental perfusion defects to predict the presence of specific angiographic abnormalities was 75% which is identical to that of Moses et al. (76%). However, to date we have not observed any false negative lung scans.

It would be interesting to know exact location of the embolus detected by the angiogram in the one patient with the false negative lung scan reported by Moses et al. It is well known that a perfusion defect in the region of lingula is often missed or misinterpreted, especially in the presence of cardiomegaly.

K. P. POULOSE, M.D.
R. C. REBA, M.D.
Department of Nuclear Medicine
Washington Hospital Center
Washington, D.C.

Reference

The authors reply:
To the Editor:
With respect to the comments of Drs. Poulose and Reba, the one patient with a false negative lung scan had an embolus in a 2 mm branch of the anterior basal segment artery to the left lower lobe seen on subselective angiography only.

Concerning the specificity of segmental perfusion defects, we might point out that we did cite a later paper co-authored by Dr. Poulose1 instead of the one referred to in the letter.

DAVID C. MOSES, M.D.
TERRY M. SILVER, M.D.
JOSEPH J. BOOKSTEIN, M.D.
University of Michigan Medical Center
Ann Arbor, Michigan

Reference

Vasodilator Therapy in Acute MI
To the Editor:
In their study of experimental coronary occlusion Dr. Hirshfeld and associates have demonstrated that simultaneous administration of nitroglycerin and methoxamine (to prevent hypotension) resulted in less ST segment elevation and less CPK depletion than in a group of control dogs.1 A possible clinical extension of their findings is to advise that when nitrates are administered to patients with acute myocardial infarction (AMI) decreases in systemic blood pressure should be prevented by simultaneous administration of vasopressors.

While severe hypotension in AMI is harmful2 previous studies with nitrates or other vasodilators have indicated that moderate hypotension is well tolerated and may improve left ventricular function as well as coronary collateral flow and myocardial viability.3,4

Thus far investigators of AMI have compared con-
trols vs nitrate or nitrate plus vasopressor groups. The critical experiment, yet to be performed, involves a comparison of nitrate therapy in AMI versus nitrate plus vasopressor therapy to determine whether or not the beneficial effects of nitrates on coronary collaterals are complemented more by decreased oxygen demands with moderate hypotension or maintenance of systemic perfusion pressure by use of vasopressors.

Allen B. Weisse, M.D.
Cardiac Care Unit
College of Medicine and Dentistry of New Jersey
Newark, New Jersey

References


The authors reply:

To the Editor:

Dr. Weisse has emphasized a critically important question regarding the use of "vasodilator" therapy in acute myocardial infarction. That is, is the systemic hypotension associated with the administration of nitrates or other vasodilators contributing to its beneficial effects or working against such effects. However, the experiments Dr. Weisse suggests should be done, have already been performed. Thus nitroglycerin, administered alone to closed-chest sedated dogs in which ischemia was induced by inflating a balloon cuff previously implanted around the left anterior descending coronary artery, caused a statistically significant reduction in ischemic injury. The beneficial effect was associated with an average decrease in mean arterial pressure of 14 mm Hg and an increase in heart rate of 20 beats/min. To determine the relation between nitroglycerin induced hypotension and associated reflex tachycardia to the drug's net beneficial effect on ischemic injury, we compared the beneficial effects of nitroglycerin administered alone with those occurring when the nitroglycerin-induced arterial pressure and heart rate changes were prevented by simultaneous administration of methoxamine. We found that such an intervention led to a further reduction in ischemic injury. Another series of experiments in dogs with chronic multiple coronary occlusions (produced by ameroid constrictors) demonstrated that nitroglycerin, administered alone during acute occlusion of the left anterior descending coronary artery, led to an increase in ischemic injury in some dogs. However, a decrease in ischemic injury occurred in all dogs when nitroglycerin-induced hypotension and tachycardia were abolished by simultaneous administration of methoxamine. Improved benefit in ventricular electrical stability subsequent to reversal of the arterial pressure and heart rate effects of nitroglycerin (by simultaneous administration of phenylephrine) also have been demonstrated.

We therefore conclude that in the experimental animal the fall in arterial pressure and reflex increase in heart rate produced by nitroglycerin opposes the beneficial actions of the drug on ischemic injury. If fall in pressure and rise in heart rate is not excessive, the net effect is still beneficial; however, nitroglycerin-induced benefit is optimal and occurs consistently only when nitroglycerin's effects on blood pressure and heart rate are attenuated or eliminated.

We also would like to emphasize that improvement in hemodynamics, as has been shown in several clinical studies investigating the use of vasodilators in the treatment of acute myocardial infarction in man, is not necessarily indicative of reduction in ischemia; the peripheral reflex effects of a vasodilator could enhance the over-all pumping performance of the heart at a time when the degree of injury to ischemic myocardium is unchanged or even increased.

Thus, the critical question posed by Dr. Weisse has been answered in the experimental animal. The next step would be to determine whether similar considerations apply to the treatment of acute myocardial infarction in man.

John W. Hirshfeld, M.D.
Division of Cardiology
Duke University Medical Center
Durham, North Carolina

Robert E. Goldstein, M.D.
Jeffrey S. Borer, M.D.
Stephen E. Epstein, M.D.
Cardiology Branch
National Heart and Lung Institute
Bethesda, Maryland
Vasodilator Therapy in Acute MI
ALLEN B. WEISSE

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