losses as well as nitroglycerin and digitalis glycosides were applied with favorable effect to cardiovascular risk patients during the perioperative period.¹ ²

Frequency of myocardial infarctions and of heart failure during the early postoperative period suggest that in coronary artery disease adrenergic overactivity induces myocardial necrosis and augments the surrounding or preceding "twilight zone" of failing, but not yet irreversibly damaged myocardium.

Therefore in a pilot study practolol was applied before and after major surgical interventions to patients with high cardiovascular risk, characterized by hypertension ≥ 160/95 mm Hg and/or old myocardial infarction and/or combined coronary artery, cerebrovascular and arterial occlusive disease.⁵ Doses ranged between 100 and 300 mg orally and 10 to 15 mg intravenously per day. In the practolol treated group no myocardial infarction occurred during treatment. In one patient an infarction led to death six days after omitting the drug. Another patient, who was operated on for a second time without practolol treatment, died two days after operation from an acute myocardial infarction. Incidence of heart failure and mortality rate were lower and no adverse reactions were noted.

In a recently published study⁶ Dr. H. S. Müller and coworkers report similar results using propranolol after acute myocardial infarction.

It is concluded that β-receptor blockade probably protects high risk patients against hypoxemic heart failure, progression of necrosis and cardiogenic shock by reduction of an extremely high and long lasting elevation of oxygen demand following stressful operations.

ERICH VORMITTAG, M.D.
I. Chir. Universitätsklinik Vienna, Austria

References

2. BRAUNWALD E, MAROKO PR: The reduction of infarct size — an idea whose time (for testing) has come. Circulation 50: 206, 1974

Estimating Collateral Circulation

To the Editor:

The October 1974 issue of *Circulation* carried a paper of mine on the coronary collateral circulation¹ which concluded that well developed collaterals play a significant role in preserving regional myocardial contractility in patients with coronary artery disease. Also included in this issue was a paper by Carroll, Verani, and Falsetti² which addressed exactly the same problem, but arrived at exactly the opposite conclusion, namely that the development of collateral vessels is *not* associated with preservation of normal segmental wall motion. No doubt this will perplex many readers who have already been perplexed enough in past years by contradictory papers on this topic.

Both studies utilized a somewhat similar approach, in which regional or segmental wall motion in areas supplied by good collaterals was compared with that in areas supplied by poor collaterals. I believe Carroll, Verani and Falsetti have erred, however, in their criteria for differentiating "good" from "poor" collaterals. These authors state that "a thread-like, poorly opacified collateral was considered 'poor,' a large, brightly opacified collateral was considered 'good' and 'fair' collaterals were those in the middle of this spectrum. . . . All patients in the 'good' class had at least two good collaterals plus any number of fair or poor vessels. Patients in the 'fair' group had at least two fair collaterals, any number of poor collaterals, but no more than one good collateral."

Their classification implies that effective or "good" collateral circulation can be equated simply with the presence of at least two large vessels, and that conversely, thread-like collateral vessels are ineffective or "poor," even though they may exist in relatively large numbers. This assumption is not justified and, I believe, negates their conclusion. The excellent post-mortem injections studies of Baroldi and Scomazzoni³ have demonstrated a myriad of tiny interconnecting "homocoronary" and "intercoronary" anastomatic vessels in all normal hearts. These vessels serve as the precursors of the coronary collateral circulation. For any given obstruction, hundreds of potential collateral channels are thus available. Nobody knows why in some cases most of the collateral flow passes through one or two relatively large anastomotic vessels, while in others a much larger number of smaller vessels is utilized. Presumably this is related to the relative positions of the obstruction and the anastomatic channels or the speed with which the obstruction develops. Baroldi and Scomazzoni even postulate that the large anastomatic channels may result from progressive necrosis of myocardium with partial loss of
collaterals and consequent increase in caliber of the remaining channels. This would suggest that large-vessel collateral circulation is if anything less, rather than more, effective. In any event, there is simply no evidence to support the hypothesis that two large collateral vessels will necessarily carry more blood to ischemic areas than 10, 25, or 100 smaller, thread-like vessels, many of which may be beyond the resolving capability of the angiographic imaging system.

In judging the effectiveness of collateral circulation, one must look beyond the mere number and size of the angiographically visible collateral vessels. A much more important factor is the quality of runoff beyond the point of obstruction. After all, the whole purpose of collateral formation is the provision of adequate blood flow to the distal tree of a blocked coronary artery. If good runoff can be demonstrated at angiography, it is safe to assume that collateral circulation is effective, regardless whether it arrives via one or two large channels or many smaller ones. Quality of runoff was the major criterion used in my study for gauging adequacy or inadequacy of collateralization. I am quite sure that this difference in classification of patients explains the difference in the conclusions of the two studies.

This subject will undoubtedly continue to be both a controversy and a source of ideas for future investigative effort. I would urge that proper criteria of classification be used, as discussed above, and am confident that others will also find that well-developed collateral circulation plays a major role in maintenance of myocardial contractility in patients with coronary disease.

DAVID C. LEVIN, M.D.
Downstate Medical Center
Brooklyn, New York 11203

References

The authors reply:

With respect to the comments of Dr. Levin, it is known that the estimation of collateral circulation is a very difficult problem. Dr. Levin proposes an interesting concept to evaluate collateral function, although it has not yet been verified by any method of direct measurement. Our classification of collaterals, based on angiographically visible vessels, is supported by a recent study which correlated angiographic appearance of collateral size and extent with direct flow measurements at surgery. Although there is no accurate technique for measuring the actual quantity of flow, we believe this study showed a significant correlation between quality of flow seen angiographically and measured directly.

It is difficult to examine Dr. Levin’s conclusions on segmental left ventricular contraction with adequate or inadequate collateral circulation. The one paragraph devoted to these results leaves several unanswered questions. Was the regional left ventricular function actually measured or estimated visually? What was the precise definition of hypokinesis; was it determined using normal left ventricular contraction? Were patients grouped according to one, two, or three vessel involvement?

We believe the differing results of Dr. Levin’s study and ours may be due not only to collateral classification but also to determination and interpretation of segmental left ventricular contraction. We are hopeful that with further investigation and advanced technology, such as microsphere technique, the role of collateral circulation in the resting and stressed heart will eventually be elucidated.

ROBYN J. CARROLL
MARIO S. VERANI, M.D.
HERMAN L. FALSETTI, M.D.
University of Iowa Hospital
Iowa City, Iowa 52242

References

Circulation, Volume 51, February 1975
D C Levin

Circulation. 1975;51:397-398
doi: 10.1161/01.CIR.51.2.397

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1975 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/51/2/397.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/