


The author replies:

To the Editor:

Dr. Dianzumba is quite correct in pointing out that we did not mention that the hypertensive groups were given antihypertensive medication during the prolonged followup stage. Indeed, they were treated vigorously when possible, but compliance and infrequent during the prolonged followup stage. Indeed, they were given antihypertensive

The interesting facet is not the evolution of hypertension so skillfully summarized by Dr. Dianzumba, but rather that this self-induced heart failure occurs in normotensives as well as hypertensives. Again, we apologize for our ambiguity.

SIDNEY J. FILLMORE, M.D.
Maricopa County General Hospital
Phoenix, Arizona 85008

MI After CABG

To the Editor:

Bulkley and Hutchins, in their analysis of myocardial infarction in patients who died after coronary bypass surgery, note that infarction was usually found in the distribution of "widely patent vessels." Their discussion therefore appropriately refers to the influence of "transient interruption of coronary artery perfusion . . . periods of nonpulsatile pump perfusion, aortic cross clamp, anoxic arrest, ventricular fibrillation . . . and exposure to pressor amines." Clinicians have usually held these latter aspects of intraoperative myocardial protection (or abuse) responsible for most instances of postoperative myocardial depression and frank infarction. Bulkley and Hutchins, however, focus attention on coronary reflow after these ischemic insults as the most likely cause of postoperative infarction. This interesting suggestion is disturbing, because it implies that infarction may be an obligatory and unavoidable consequence of coronary bypass in a relatively high percentage of cases.

For these reasons, Bulkley and Hutchins' total failure to describe the methods of intraoperative myocardial protection employed in their study is a crucial omission. Failure to specify the duration of aortic cross-clamping, ventricular fibrillation, etc., to correlate these clinical data with necropsy findings, and to compare these findings with similar analyses in survivors of operation, limits the relevance of their study.

Many of the patients they studied apparently died after surgery some years ago, when surgical techniques were less refined, and morbidity and mortality were much greater than now. For example, in our own current experience with 325 consecutive coronary bypass operations (including all patients with unstable angina, left main disease, poor LV function, etc.), there have been five hospital deaths (1.5%), with none due to low cardiac output. Four of these five patients had necropsy, and no fresh myocardial necrosis was found by a senior cardiac pathologist. There were six perioperative infarctions in the survivors (1.9%) by ECG. While some unrecognized infarctions may have occurred, these could not have been frequent, as only six patients in the entire series briefly required postoperative inotropic support. This experience is cited only to support our feeling that perioperative infarction is usually a preventable occurrence related predominantly to technical factors. Our operative techniques have been described elsewhere, and are based on common surgical principles.

If the conclusions of Bulkley and Hutchins' study are to be widely applicable, detailed data about intraoperative myocardial management and the results in survivors and non-survivors are required.

LAWRENCE I. BONCHEK, M.D.
Medical College of Wisconsin
Milwaukee, Wisconsin 53226

References


The authors reply:

To the Editor:

Dr. Bonchek raises three major issues with regard to our study on myocardial infarction in autopsied patients after coronary artery bypass surgery.¹

1. His concern that the findings make myocardial infarction an obligatory consequence of this procedure in a relatively high percentage of cases represents a misinterpretation of the study. By its very nature an autopsy study does not provide incidence data; unless all patients die and are autopsied, it is inappropriate to extrapolate these "percentages" to the living, surviving, population. As a study of pathophysiology and not epidemiology, its aim was to focus on the nature and possible mechanisms of myocardial injury after coronary surgery. Although we know from animal studies that under certain conditions of transient ischemia myocardial injury may be worsened by reflow,² our study suggests that a similar injury, under certain circumstances, may occur in the human as well.

2. With regard to Dr. Bonchek's second concern about intraoperative methods, I would suggest that he read the article again, noting specifically: table 4; the last paragraph of the results section (p. 910); and the second paragraph of the discussion (p. 911). Techniques of myocardial protection were not significantly different between the autopsied group that developed necrosis and those that did not. In the first four years of bypass surgery at this institution, normothermic anoxic arrest was used; subsequently, hypothermic arrest. In the past year we have used cold potassium cardioplegia, but none of the patients in this study had this kind of arrest. Since the autopsy numbers are small, and the patient population heterogeneous (as we are operating on more and sicker patients today than five or ten years ago), that some infarcts seen at autopsy have developed despite hypothermic protection does not mean that the latter is not superior to normothermic arrest. The entire operative experience is necessary to answer this type of question, and not the handful of patients that come to autopsy for a variety of reasons.

3. Dr. Bonchek is correct that this study included patients that died some time ago and in fact the patients span close to ten years,
MI after CABG.
L I Bonchek

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