The Lazarus Phenomenon Revisited

To The Editor.—Our Cardiothoracic Anesthesia Service recently was involved in the care of a 75-year-old white man who had undergone an open cardiac surgical procedure 8 days before the current event. He had been experiencing severe ongoing refractory congestive heart failure since the operation and required continued invasive monitoring and ongoing hemodynamic support. During a routine measurement of the pulmonary artery occlusion pressure, the patient had hemoptysis. He became markedly cyanotic, hypotensive, and unresponsive. An acute rupture of the pulmonary artery was suspected, and an endotracheal tube was advanced into the right mainstem bronchus. Several doses of atropine and epinephrine were administered, and the patient was transported to the surgical suite for emergent thoracotomy and pulmonary artery ligation. There he became pulseless and the full advanced cardiac life support (ACLS) resuscitation protocol was initiated. No clinical response was observed. Asystole ensued, as demonstrated on two separate three-lead electrocardiogram (ECG) systems. Both femoral and carotid pulsations, which had been easily palpable prior to the arrest, were absent. His pupils were fixed and dilated. Resuscitation was terminated after 23 min, all infusions were stopped and the oxygen supply was disconnected.

Approximately 5 min later, as the patient was being prepared for transportation, femoral pulses were serendipitously discovered. The ECG monitor was reattached—using the same leads, cables, and machines as before—and revealed a sinus tachycardia at a rate of 115 beats/min. Cardiopulmonary resuscitation was reinstalled, and the patient was eventually returned to the intensive care unit. Though hemodynamic stability was ultimately obtained, no significant neurologic recovery was observed. All support was discontinued several days after the event and the patient subsequently expired.

This delayed return of the native circulation—the "Lazarus Phenomenon"—has been described in the literature on only two previous occasions, each from Europe and appearing as letters to the editor, one in response to the other, more than a decade ago.2,3 That these are truly the only examples of this phenomenon ever to have occurred prior to ours—both in the same country and within a few months of each other—seems unlikely. More probably, rather than being remarkably rare, this experience is simply grossly under-reported.

Such a reluctance may result from the extreme difficulty encountered when trying to explain the event in a scientifically satisfying fashion. The fear of an accusation of negligence, even though none may be evident, may contribute to the stifling of this discussion. The circumstances surrounding the care of our patient may help ameliorate such concerns. This individual could not be resuscitated using the standard ACLS protocol and this outcome was well documented. Two separate ECG systems and multiple examinations by several physicians confirmed the diagnosis of unresponsive cessation of native cardiopulmonary function. This condition, established by more evidence and supportive expert opinion than is usually present in these situations, surprisingly proved to be reversible. The native circulation resumed spontaneously some time after the cessation of our efforts directed toward stimulating it. Clearly, considering the extensive documentation of cardiopulmonary arrest resistant to treatment, an explanation other than oversight must exist to explain the observed reversal. An important physiologic mechanism remains to be elucidated.

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(Accepted for publication February 15, 1993.)

Is Nitroglycerin a Myocardial Depressant?

To the Editor.—Recently Cahalan et al.1 concluded that, in absence of the effects of anesthesia and surgery, elderly patients have a more pronounced blood pressure response to intravenous nitroglycerin infusion. This is in contrast to the findings by Gascho et al.2 who reported that nitroglycerin administration produced less venous dispersion in patients older than 60 yr than in younger patients. On the
other hand, measurements of arterial plasma concentrations of nitroglycerin and its metabolites failed to detect significant pharmacokinetic differences between elderly and young patients. The authors thereby hypothesized that an age-related difference in the effect of nitroglycerin at the active site was likely. Furthermore, they were forced to speculate that a markedly decreased circulatory compensation might be responsible for the reductions in cardiac output and blood pressure if there was a decreased venous distensibility in elderly patients. Unfortunately, they did not provide the complete hemodynamic data, particularly cardiac output and systemic vascular resistance, following nitroglycerin administration for comparison. Other than the already well known vasodilating effect of nitroglycerin, it would be interesting to know if myocardial contractility was decreased. If there was, did it have a greater effect on the elderly heart? So far, the effect of nitroglycerin on myocardial contractility has not been well defined or characterized.3

Recently, we conducted studies to investigate the direct effects of nitroglycerin on myocardial contractility in rabbits.4,5 We used myocardial septa and perfused them with nitroglycerin through the first septal perforator of the left coronary artery in different concentrations. The results showed that nitroglycerin infusions caused a significant dose-related depression of isotropic activity in rabbit myocardium that differed from that of nitroprusside (fig. 1). Of course, we cannot extrapolate our data directly to humans. However, since the relationship between age and relaxation of vascular smooth muscle is still controversial,4,6 it would have been helpful had they studied the effects of nitroprusside in addition to those of nitroglycerin.

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(Accepted for publication February 15, 1993.)

In Reply.—As Lee and colleagues correctly note, we were unable to definitively determine the cause of our patients' blood pressure responses to nitroglycerin, because we were unwilling to place invasive monitors that were not required for clinical care.

Lee and colleagues propose a cause for nitroglycerin's blood pressure effects we had not seriously considered, namely, myocardial depression. In their letter, they provide us with data demonstrating a dose-dependent effect of nitroglycerin on peak developed tension (PDT) and the maximal acceleration of tension (dT/dt).

However, we question the relevance of their findings to ours for

Anesthesiology 78:992–993, 1993
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J. B. Lippincott Company, Philadelphia

Anesthesiology, V 78, No 5, May 1993

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