CORRESPONDENCE

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The Lazarus Phenomenon Re-revisited: I

To the Editor:-Bray recently described a delayed return of the native circulation—the "Lazarus phenomenon"—in a patient with suspected rupture of the pulmonary artery after 23 min of unsuccessful resuscitation using the standard advanced cardiac life support (ACLS) protocol.1 No mechanism could be provided for this phenomenon. One possible explanation may be related to increased airway pressures during the resuscitative effort. If high airway pressures developed as a consequence of vigorous ventilatory support, these in turn would give rise to high pleural and right atrial pressures and result in a decreased venous return to the heart and decreased cardiac output. (Although it is stated that the tracheal tube was advanced into the right mainstem bronchus, no further details concerning ventilatory management are mentioned.1) The concealed blood loss in this particular patient1 may have magnified the reduction in cardiac output associated with elevated airway pressures. At the end of ACLS, return of spontaneous ventilation (gasping) may have augmented venous return and permitted an increase in cardiac output.

In addition to the two cases of the "Lazarus phenomenon" reported in 1982 and cited by Bray, 1 there have been two recent reports of this phenomenon in the literature. ^{2,3} For each, the explanation proposed for the restoration of spontaneous circulation after cessation of ACLS was similar to that briefly outlined above.

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The Lazarus Phenomenon Re-revisited: II

To the Editor:—Bray¹ describes the Lazarus phenomenon as the spontaneous return of native circulation after cardiopulmonary resuscitation (CPR) is abandoned. His patient did not survive, but two of the three other reported cases².⁵ left the hospital to independent life after CPR was abandoned.

Also described is the Lazarus sign⁴⁻⁶ in patients who have been diagnosed as brainstem dead but who make spontaneous purposeful movements, apparently as though struggling to breathe, after artificial ventilation has been discontinued. None of these patients survived, but none were given the benefit of further CPR. His presumption of gross under-reporting also would apply to these situations and for the same reasons.

In view of these reports and the increasing mass of published evidence that the fulfillment of brainstem death criteria does not establish the total cessation of all brain activity, the question must be asked: "How many of the patients used for organ harvesting worldwide might have recovered, had CPR been continued?" The question is more urgent considering that patients used for organ harvesting

both require and receive routinely full general anesthesia with paralysis for the performance of the surgery—a situation difficult to reconcile with that of a corpse. That some operating-room personnel share these concerns is evidenced by operating-room registers that record the time of death of the patient as some time after the commencement of surgery to harvest organs.

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In Reply:—I thank Koblin for calling attention to another example of the delayed return of spontaneous circulation. The mechanism postulated in that report, however, does not appear to be the most likely explanation of the event we described. Our patient probably did have some increase in intrathoracic pressure as discussed by Martens et al.1 The tracheal tube, initially advanced into the bronchus in an attempt to staunch the pulmonary hemorrhage, was withdrawn when blood flow continued from both in and around the tube in the bronchial position. Airway pressures during manual ventilation were not excessive, although bilateral chest tubes were placed to rule out the possibility of tension pneumothorax. Our patient made no spontaneous respiratory efforts once support was discontinued, but some decrease in intrathoracic pressure, no doubt, still occurred, perhaps augmenting passive ventricular filling. Whether this augmentation can explain the return of spontaneous circulation after the cessation of all cardiac electrical activity is questionable. An improvement in ventricular filling certainly would enhance forward flow in an empty but beating heart. However, how this filling would stimulate the completely quiescent myocardium—the total absence of both cardiac electrical and mechanical activity—is not readily apparent. There had been no electrical cardiac activity—as evidenced by two separate concurrently functioning electrocardiogram systems—for several minutes at the time the efforts were terminated. This situation spontaneously reversed.

Hill raises another important issue. The sensitivity of current clinical brainstem testing as a marker for cessation of brain function is a critical question and the spontaneous movement of a patient so diagnosed would be a concerning event. However, those individuals who breathe spontaneously after the discontinuation of mechanical ventilatory support fail the so-called apnea test and therefore are not

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eligible for organ donation in our institution. Further, the harvesting process does not occur in patients receiving ongoing cardiopulmonary resuscitation. It is reserved primarily for individuals who have been successfully resuscitated to the only clinical endpoint currently available, the restoration of spontaneous circulation. Those who cannot be successfully resuscitated are not usually candidates for organ donation. The problem comes when hemodynamic function is restored in the apparent absence of neurologic function. This latter issue, the reliability of our assessment of irreversible neurologic "death," is really the crux of the point raised by Hill and remains a significant issue.

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The Proper Study of Anesthetized Humans: I

To the Editor:—Barnas et al. recently published data describing how changes in mean lung volume affect respiratory system mechanics measured during sinusoidal forcing. The authors assume that these

results obtained in healthy, awake, seated subjects can be applied to anesthetized recumbent patients. To understand why this assumption may not be valid, a bit of history is required. Pulmonary physiologists