that the case of Harden *et al.*[4] should not be regarded as a true Lazarus phenomenon because there is no indication that CPR had been stopped at the time when spontaneous conversion of ventricular fibrillation occurred.

With regard to prevention of Lazarus phenomena, Frölich suggests to continue CPR until ineffectiveness has been shown by a decreasing *p*H with adequate ventilation.[1] Although this approach is probably correct, there are no defined *p*H values below which resuscitation can be considered futile. In the case of Fumeaux *et al.*,[5] the patient survived neurologically intact after cessation of CPR at a *p*H of 6.54. An alternative approach might be end-tidal carbon dioxide. Its use for therapeutic and prognostic decisions during CPR was first proposed by Eisenmenger,[6,7] first used in humans by Leigh *et al.*,[8] and studied in detail by Smalhout.[9] In the last 20 years there have been several studies on capnography during CPR.[10] Values greater than 10–15 mmHg indicate a favorable prognosis and should preclude termination of CPR. Unfortunately, there are no capnography data in the Lazarus cases published to date, including ours and that of Frölich.[1,2]

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(Accepted for publication March 25, 1999.)

Anesthesiology
1999; 91:585–6

Spontaneous Recovery after Discontinuation of Cardiopulmonary Resuscitation

To the Editor:—I read with interest the case report from Dr. Frölich on spontaneous recovery after discontinuation of intraoperative cardiopulmonary resuscitation (CPR).[1] This rare and unsettling occurrence was also observed recently in our intensive care unit, although the postulated etiology differs from the published case.

A 76-yr-old man with severe bullous chronic obstructive pulmonary disease had been admitted *in extremis* requiring urgent intubation and ventilation. Within minutes he had suffered cardiac arrest from which he was resuscitated, although with evidence of residual hypoxic encephalopathy. He was resistant to attempts to wean him from mechanical ventilatory support. On the eighth day, while the patient was undergoing synchronized intermittent mandatory ventilation with pressure support, it was noted that the ventilator pressures were fluctuating widely, although delivered tidal volume was constant. He rapidly developed a profound bradycardia and increasing cyanosis. The ventilator was disconnected and manual ventilation with a self-inflating bag and chest compressions were started. In response to 0.6 mg atropine and 1 mg epinephrine, he developed a ventricular tachycardia that progressed to ventricular fibrillation. Direct current defibrillation led to a wide complex rhythm that progressed to asystole despite further pharmacologic intervention (including additional epinephrine, dopamine, bicarbonate, and lignocaine). It was noted throughout that ventilatory compliance was poor, although there was bilateral air entry, the trachea was central, and the ready passage of a large bore suction catheter suggested tube patency was not compromised. An arterial blood gas analysis during CPR showed *p*H 6.92, Pa_{CO_2} 117 mmHg, Pa_{O_2} 327 mmHg, and base excess −10 M.

After 30 min of CPR with no evidence of spontaneous circulation and asystole in all electrocardiogram leads, resuscitative efforts were discontinued. The endotracheal tube was removed, and examination of it showed nothing untoward; the electrocardiogram remained connected. After 5 min return of cardiac electrical activity was noted, which progressed to sinus tachycardia accompanied by good volume pulses and spontaneous respiratory effort.

Arterial blood gas analysis shortly thereafter, with the patient breathing spontaneously with $F_{I_{O_2}}$ of 0.24, showed *p*H 7.19, Pa_{CO_2} 64 mmHg,

Pa_{O_2} 53 mmHg, and base excess −3.7 mM. Chest radiograph confirmed that there was no tension pneumothorax. However, neurologic assessment showed exacerbation of the hypoxic encephalopathy. After consultation with the family, it was agreed that supportive measures only would be continued. These included biphasic positive airway pressure ventilation with $F_{I_{O_2}}$ 0.7 *via* nasotracheal tube, giving blood gas analysis of *p*H 7.30, Pa_{CO_2} 57 mmHg, Pa_{O_2} 68 mmHg, base excess −3.8 mM. Twenty-four hours later there was further deterioration in his condition, and he died without active resuscitation being performed.

The sequence of events strongly suggested a respiratory cause for both arrests. Although it may be difficult to rule out tension pneumothorax clinically, there is considerable risk in arbitrary placement of an intercostal needle or drain in a patient with severe bullous chronic obstructive pulmonary disease. A subsequent radiograph showed no pneumothorax. A more plausible explanation is the development of dynamic hyperinflation as a consequence of excessive end-expiratory pressure in the respiratory system, or auto-postive end-expiratory pressure; this may lead to significant impedance to venous return and reduction in cardiac output.[2] Although modern ventilators can be programmed to provide optimum cycle lengths, inspiratory-expiratory time ratios, variable inspiratory flow patterns, and external positive end-expiratory pressure to counter auto-postive end-expiratory pressure, such refinements are not available during manual ventilation in the heat of CPR. It is suggested that in this patient pressure in the respiratory system was so high that it shut down venous return. When CPR was discontinued and the endotracheal tube was removed, over the ensuing 5 min there was a gradual return of pressure in the respiratory system to atmospheric pressure, which allowed the heart to fill once more, leading to a return of spontaneous function.

Like Dr. Frölich's case, our patient was fully monitored and chest compressions were observed to be effective in creating a waveform on the arterial line, yet the *p*H was 6.9 and there was asystole. This may be a further argument for monitoring for an additional 10 min after discontinuation of resuscitation, as suggested in a previous report.[3]

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(Accepted for publication March 25, 1999.)

Anesthesiology
1999; 91:586

In Reply:—In his letter, Dr. Maleck refers to previously reported cases of spontaneous recovery after unsuccessful resuscitation, including one of his own cases in which a patient was resuscitated for asystolic arrest for approximately 30 min and showed return of spontaneous circulation 5 min after cessation of resuscitation efforts. Dr. MacGillivray adds another case of asystolic arrest with spontaneous recovery after cardiopulmonary resuscitation (CPR). In both cases, resuscitative efforts were discontinued after an arbitrary time frame of 30 min.

The apparent question in the reported cases of the "Lazarus phenomenon," including my own, is whether resuscitation had been terminated prematurely. To date, there seems to be no consensus with respect to the timing of termination of CPR. The manual on advanced cardiac life support (ACLS) by the American Heart Association[1] states that resuscitation may be discontinued in the prehospital setting after an adequate trial of ACLS. An "extremely short attempt to reverse the arrest" is recommended for the intensive or critical care unit because of close monitoring by personnel and immediate attempts to reverse the arrest. In most clinical situations, CPR is discontinued after a full course of the relevant ACLS algorithm.

There are limited data on the predictability of outcome after CPR. Levine *et al.*[2] recommended the discontinuation of CPR if an end-tidal carbon dioxide level of ≤ 10 mmHg is measured 20 min after the initiation of ACLS. Dr. Maleck, in his letter, also makes reference to this approach. I would use end-tidal CO_2 as a parameter to gauge the effectiveness of resuscitation rather than to predict outcome. Nevertheless, it seems to be reasonable to discontinue CPR if effective circulation cannot be re-established over an extended period of time.

From my own and similar reported cases, I have learned that CPR should not automatically be discontinued if the end of the ACLS algorithm has been reached. Resuscitation may have to be continued until proven ineffective by parameters such as end-tidal CO_2 or metabolic deterioration.

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(Accepted for publication March 25, 1999.)