that the case of Harden et al. 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 pH with adequate ventilation. Although this approach is probably correct, there are no defined pH values below which resuscitation can be considered futile. In the case of Fumeaux et al., the patient survived neurologically intact after cessation of CPR at a pH 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, first used in humans by Leigh et al., and studied in detail by Smalhout. In the last 20 years there have been several studies on capnography during CPR. 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.

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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). 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 pH 6.92, PaCO2 117 mmHg, PaO2 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 Pao2 of 24, showed pH 7.19, PaCO2 64 mmHg,
Twenty-four hours later there was further deterioration in his condition, dynamic hyperinflation as a consequence of excessive end-expiratory pressure. After consultation with the family, it was agreed that supportive measures only would be continued. These included biphasic positive airway pressure ventilation with Fio2 0.7 via nasotracheal tube, giving blood gas analysis of pH 7.30, PaO2, 57 mmHg, PaCO2, 68 mmHg, base excess -3.8 m. 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–positive end-expiratory pressure; this may lead to significant impedance to venous return and reduction in cardiac output. 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–positive 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.

References


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