diagnosis, the activity of lymphocyte coproporphyrinogen oxidase may be determined.[2] The test, which is accurate and reliable, is being conducted in a few porphyria reference laboratories. Both tests are used to establish a diagnosis also in the latent phase and may therefore be performed in the patient after recovery.

We suggest that the patient be checked by a reference laboratory authorized for the biochemical diagnosis of porphyria before any conclusion concerning the use of propofol in porphyric patients is drawn.

In addition, we would like to point out a few mistakes in the report of the biochemical findings in the urine: aminolevulinic acid and porphobilinogen are not porphyrins but precursors in the porphyrin biosynthetic pathway; the excretion of aminolevulinic acid and porphobilinogen is determined in micromoles per 24 h and that of porphyrins in nanomoles per 24 h, not in millimoles as reported in the article.

Rivka Mamet, Ph.D.
Nili Schoenfeld, Ph.D.
Porphyria Reference Laboratory
Rabin Medical Center, Beilinson Campus
Petah-Tikva, Israel

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In Reply:—We would like to thank Drs. Mamet and Schoenfeld for their instructive comments and corrections. We completely agree that porphyria had not been definitively diagnosed and that the pattern of porphyrin and porphyrin precursor elevation is consistent with any neurogenic porphyria. However, it should be noted that the patient's liver function tests had returned to the normal range (except for a minimally elevated alanine transaminase level) on the day before the urine porphyrin collection. Furthermore, we noted that propofol (which could have interfered with the colorimetric assay) was at near-undetectable levels at the time of urine collection, thus making both liver dysfunction or drug effect unlikely as the cause of the abnormal laboratory results. Regrettably, testing for lead poisoning was not performed at that time; however, clinically, there was no feature to suggest this as a possibility.

The patient was referred to our center for his ablation and has not followed up with us. We strongly recommended that a fecal porphyrin profile as well as a coproporphyrinogen oxidase level analysis be performed by his primary physician.

Despite the above discussion, in our opinion, the clinical syndrome and abnormal tests as outlined in our report make latent neurogenic porphyria manifested by propofol an important and likely possibility. We believe that this observation should be considered when administering large amounts of propofol to porphyric patients until larger studies have demonstrated otherwise.

Samuel J. Asirvatham, M.D.
Department of Electrophysiology
St. Mary's Hospital
Rochester, Minnesota 55902
Asirvatham.samuel@mayo.edu

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Recovery after Discontinuation of Cardiopulmonary Resuscitation ("Lazarus Phenomenon")

To the Editor:—The case report by Frölich is one of the best documented cases of spontaneous recovery after discontinuation of cardiopulmonary resuscitation (CPR).[1] More than 25 such cases have been reported, including at least eight patients discharged neurologically intact.[2] One case published in ANESTHESIOLOGY introduced the term "Lazarus phenomenon" for such events.[3] However, we would argue

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]

Wolfgang H. Maleck, A.R.Z.T.
Swen N. Piper, M.D.
Department of Anesthesiology
Klinikum
D-67063 Ludwigshafen, Germany
wolfgang_maleck@hotmail.com

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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).[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,