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.

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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.

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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