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In Reply:—The communication to which Dr. Baum refers was not a response to a letter to the editor; instead, it was a *de novo* submission. Dr. Baum also seems confused about the purpose and nature of the Outcomes Research™ Group.

The group was founded in 1990 and now includes 65 members in 10 countries. Our primary interest is large outcome studies; however, we continue to conduct smaller studies. Fewer than half are thermoregulatory, and a fair number are unrelated to anesthesia. We have more than 60 studies in progress, and the group typically publishes more than 20 full articles each year. Most of our funding is derived from peer-reviewed sources, including the National Institutes of Health. However, we are neither a corporation nor a foundation; consequently, all funds are administered by host universities.

Additional information about the group, including a list of members, is available from our web site: outcomes-research.org. This site includes a searchable bibliography of more than 2,500 references related to thermoregulation and other group interests. The entire database can be downloaded in EndNote® format. The web site similarly includes various slide presentations; these PowerPoint® files also can be downloaded for teaching purposes.

Dr. Baum wonders how we can trademark a relatively common term, and what the implication might be. There is no particular difficulty obtaining trademark protection for common terms. Consider, for example, "Palm" of the Palm III organizer. We registered "Outcomes Research" rather than "Outcomes Research Group" for the same reason that the Apple Computer, Inc. registered the term "Apple" rather than "Apple Computer Company": So we can refer to the group using the shorter and more convenient term. A trademark on Outcomes Research does not preclude casual or descriptive use of the words *outcomes research*. However, it would be inappropriate to use the proper noun Outcomes Research to solicit grants or corporate contracts, especially if doing so engendered any confusion with our group.

We appreciate Dr. Baum's interest in supporting our efforts. Checks can be made payable to the Regents of the University of California and mailed to my attention. Small unmarked bills will also be accepted.

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A Reliable Diagnosis of Porphyria Should Preceed any Conclusion Concerning the Safety of a Drug in Porphyria

To the Editor:—Asirvatham *et al.*[1] reported a case of "prolonged loss of consciousness and elevated porphyrins following propofol administration." In view of this, the authors conclude that it might be the first report to doubt whether propofol, an agent considered to be safe in porphyric patients, is indeed safe. However, we doubt the diagnosis of porphyria in this case and, therefore, doubt the conclusion.

To the best of our knowledge, increases in urinary aminolevulinic acid, porphobilinogen, and coproporphyrin III are not necessarily compatible with the diagnosis of coproporphyria, a diagnosis suggested by the authors. These biochemical findings may be compatible with any type of neurogenic porphyria (coproporphyria, acute intermittent, variegate) as a primary cause and also may reflect secondary changes due to other clinical states (*e.g.*, liver injury, effect of drugs, lead poisoning).[2,3] Various biochemical tests, related to porphyrin biosynthetic pathway, conducted in the urine, feces, and blood may indicate which of the aforementioned causes leads to the abnormalities reported in the urine.[4] According to the case report, none of these tests was performed. The diagnosis of coproporphyria, for example, should be based on disturbed fecal porphyrin profile with elevated coproporphyrin that predominates and a reversed ratio of coproporphyrin III/I (from < 1 in normal patients to up to 30 in coproporphyric patients).[5,6] To complete the

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