Incomplete Reporting in a
Case Report of
Corticosteroids in the
Treatment of Alcohol-Induced
Rhabdomyolysis

To the Editor: In the October 2011 issue of Mayo Clinic Proceedings, we reported on a 55-year-old man who had an association between substance abuse and muscle disease. We interpreted this as an example of alcohol-induced rhabdomyolysis and shared that interpretation with readers of the Proceedings. We further speculated that improvements in the patient's condition while under our care might have been the result of corticosteroid treatment.

Shortly after the publication of our report, other physicians who also cared for this patient during other hospitalizations brought to our attention that our report did not accurately represent the complexity of the case. Specifically, we did not report multiple confounding events that could have meaningfully influenced readers' interpretation of the underlying pathophysiology and our claims of a positive response to corticosteroid treatment in this patient.

Based on this critique, we again reviewed the complete collection of the patient's record at the Medical Center of Louisiana at New Orleans. These additional findings were submitted to experts provided both by Tulane University and by the Editorial Board of Mayo Clinic Proceedings. Some factors that the authors and reviewers felt should be shared with readers include:

- On rereview of the available records, the hospital course reported was the fourth, and not the second, in a series of 5 admissions for similar symptoms.
- In at least one prior admission, the patient experienced rhabdomyolysis that resolved over the course of 4 days without the use of corticosteroids.
- 3. Poorly differentiated, metastatic carcinoma was diagnosed 1 week after the diagnosis of polymyositis. Ultimately, immunohistochemistry results from a biopsy specimen of a pelvic lesion suggested that lung cancer was the likely primary tumor.

- 4. Polymyositis is known to occur as a paraneoplastic syndrome and may respond to corticosteroids,²⁻⁴ but a direct relationship between lung cancer and rhabdomyolysis is not well described.
- 5. The extremely high levels of creatine kinase (>400,000 U/L) and history of fluctuating muscle pain may speak against polymyositis being the cause of the rhabdomyolysis, although we did not rule it out with certainty. Based on the patient's history, other possible etiologies include cocaine and alcohol abuse; however, on both hospital admissions, the patient's urine drug screen was negative for cocaine.
- 6. Finally, we should make it clear that we cannot rule out the possibility of alcohol-induced rhabdomyolysis coexisting with polymyositis.

In the final analysis, the authors and expert reviewers were uncertain whether the new information changed the conclusions and diagnosis presented in our case report. However, all agree that we were remiss in not being more precise with these facts in the manuscript accepted for publication in *Mayo Clinic Proceedings*.

We apologize for any confusion our actions may have caused, and we thank those individuals who brought this oversight to our attention and helped us clarify the facts of the case.

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Opioid Substitution Therapy for Dependent Health Care Practitioners: Approach With Caution

To the Editor: In a recent article in Mayo Clinic Proceedings, Hamza and Bryson visit the difficult decisions involved in returning addicted health care practitioners (HCPs) back to work; their article focuses on maintenance therapy of addiction disorders with opioid maintenance therapy. Specifically, they propose that "abstinence-based recovery should be recommended until studies demonstrate that it is safe to allow this population to practice while undergoing opioid substitution therapy."1 They review the current (and limited) research on the cognitive effects of opioid medications, including buprenorphine. Opioids are indeed potent drugs with primary targets in the central nervous system; it should come as no surprise that they alter brain functioning. Collateral information from a completely different angle comes from the magnetic resonance imaging research of Younger et al.2 They describe changes in neuronal structures when opioid-naive individuals are prescribed morphine for 1 month. These 2 widely divergent angles of study provide a compelling case for caution and further investigation.

But what should addicted HCPs do while the research sorts itself out? Two different points of view emerge. One view is that proposed by Hamza and Bryson: to hold off on prescribing buprenorphine and other opioid agonists until we know more about the effects of these opioids on critical thinking. The other point of view comes from the vast clinical experience addicted HCPs have with partial or full μ -opioid agonists. Clinicians who have been using opioid replacement therapy for years may be tempted to see the Hamza and Bryson position as withholding humane care or even acting in a prejudicial or punitive fashion toward our colleagues who develop an addictive disease. I believe that careful consideration of that landscape of addiction care in this population upholds the cautious approach proposed in the article by Hamza and Bryson.

My conclusion comes from 4 lines of reasoning. First, as prevalent as opioid replacement therapy is, we do not have a clear