LETTER TO THE EDITOR
http://dx.doi.org/10.4306/pi.2012.9.1.85
Print ISSN 1738-3684 / On-line ISSN 1976-3026
OPEN ACCESS

Comment on "Serial Monitoring of Lead aVR in Patients with Prolonged Unconsciousness Following Tricyclic Antidepressant Overdose"

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Sir,

I enjoyed reading the manuscript entitled: "Serial Monitoring of Lead aVR in Patients with Prolonged Unconsciousness Following Tricyclic Antidepressant Overdose" published in your journal.[1] Based on the single case reported by the authors, they suggested that the decrease in the height of R wave and R/S ratio in lead aVR may be related to the level of consciousness and be informative in predicting recovery from toxicity following tricyclic antidepressant (TCA) overdose.[1] I think there are a few problems with this suggestion. For instance, the reason for altered consciousness in TCA poisoning and the reason for electrocardiographic (ECG) changes results from different pharmacologic properties; ECG changes occur from sodium channel blockade during cardiac depolarization. Sodium channel blockade is unlikely to be the only mechanism responsible for altered consciousness and, therefore, the association with electrocardiographic changes of sodium channel blockade might not necessarily be a causal one.[2-4] It has been shown that the duration of coma in TCA poisoning is variable and does not necessarily correlate to or concomitantly occur with electrocardiographic abnormalities.[5]

Surrogates (like the ECG in TCA overdose) are useful when direct knowledge of something is not possible. Increment in GCS is easy to be directly observed and therefore, does not need a surrogate. Of course, this does not mean that ECG findings are not important in TCA toxicity. In overdose, changes in ECG parameters are used to determine the need for antidotal therapy[6-8] as well as the risk assessment.[2,9-13] Furthermore, few studies have shown that the level of consciousness (coma grade) at presentation is the most sensitive clinical predictor of dysrhythmia and seizure after TCA overdose.[14-16]

Also, another concern is about the use of sedatives for the patients on mechanical ventilation. Was sedation or analgesia used in this patient? If so, this could confound their observation with respect to the duration of CNS depression and correlating the ECG to the GCS. Thanks for this interesting article.

Received: July 14, 2011 **Revised:** September 9, 2011
Accepted: September 27, 2011 **Available online:** January 18, 2012

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