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☐ REPLY

☐ To the Editor:

We thank De Bernardo et al. for their response to our recent publication about the impact of head of bed elevation on optic nerve sheath diameter (ONSD) measured by ultrasound (US) (1).

De Bernardo et al.'s first concern was the so-called blooming effect that might affect measurement accuracy, so they recommended using the standardized A-scan sonography instead of the B-scan method.

We have previously noted the blooming effect and tried to avoid it in our research. Two blinded sonographers performed 450 ONSD measurements each and the interrater reliability of the sonographers at all 5 time points was >0.9. Therefore, we think that the blooming effect did not influence our results.

Although De Bernardo et al. suggest that measurements with A mode are more precise than measurements with B mode, the majority of the current literature in sonographic ONSD measurements affirms the B mode technique (2–4). However, suggesting that A mode is superior to B mode, or vice versa, requires studies where both methods are tested concurrently and compared with intracranial pressur monitoring, which are lacking (5).

The authors recently stated that standardized A mode is more difficult to perform and it requires some skill (6). This might increase the risk of measurement errors in less skilled hands. We are not experienced with the standardized A scan technique, but we agree with the current literature that A scan and B scan are complementary methods (2).

As their secondary concern, De Bernardo et al. claim that the probe was slightly misplaced in our Figure 2. We disagree. However, ONSD measurements with A scan and B scan are both user-dependent methods (2). Many studies have reported the interrater concordance of sonographic ONSD measurements (7–9). According to these studies, we think that our results measured by B mode, with high interrater reliability between the sonographers, were not influenced by these 2 concerns.

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☐ REPRINT OF: REMEMBER ATROPINE FOR "KILLER B'S"***

☐ To the Editor:

With great interest we read the article from Sercan and Selahattin titled "Respiratory failure due to plant poisoning: *Nicotiana glauca* Graham" in the latest issue of *The Journal of Emergency Medicine* (1). Anabasine is an alkaloid found in the plant *Nicotiana glauca* and

is chemically similar to nicotine. Toxicity manifests as a cholinergic syndrome that includes weakness, hypertension, tachycardia, convulsions, and muscle fasciculations, due to overstimulation of nicotinic acetylcholine receptors in the central and peripheral nervous system. However, prolonged depolarization at the receptor diminishes the responses and results in hypotension, bradycardia, paralysis, and coma (2). We are not informed about pupil size nor occurrence of bradycardia and hypotension when the patient deteriorates. In that case, atropine can be used for treatment of bradycardia. The authors describe some symptoms that may also indicate muscarinic overstimulation (vomiting, salivation, and bronchorrhea). Muscarinic symptoms can be remembered by the mnemonic DUMBELS, which is an acronym for Diarrhea, Urination, Miosis, Bronchospasm/bronchorrhea/bradycardia, Emesis, Lacrimation, and Salivation/sweating/secretion. Atropine is a muscarinic agonist and is able to reverse muscarinic symptoms. Atropine is especially useful when facing high-risk symptoms like bronchorrhea and bradycardia (killer B's). Although treatment in this case was supportive, please remember atropine as an antidote, especially when confronted with a cholinergic crisis with possible signs of muscarinic symptoms, including the killer B's!

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☐ RESPONSE TO LETTER TO THE EDITOR

☐ To the Editor:

Thank you for the opportunity to respond to Dr. Olgers and colleagues (1). We appreciate their acknowledgement of our case report.

Firstly, we did not detect bradycardia or hypotension when the general condition of our patient began to deteriorate. The patient's blood pressure during intubation was 140/84 mm Hg, and pulse rate was 134 beats/min. Also, hypotension was not detected during the patient's observation in the Emergency Department, and the pulse rate remained at 98 to 143 beats/min. In addition, pupil size was within normal limits although the patient had ptosis.

Secondly, we did not use atropine in the treatment because bradycardia and bronchorrhea were not detected during the management of the patient.

Gastrointestinal, cardiovascular, respiratory, and neurological symptoms may occur due to nicotinic stimulation (2). In the literature, different types of clinical presentations related to *Nicotiana glauca* Graham poisoning have been reported (3). In our case, toxicity was characterized mainly by neuromuscular blockade and respiratory failure. Therefore, respiratory support and close follow-up were applied to the patient.

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☐ UTERINE ARTERY PSEUDOANEURYSM: SOME PRACTICAL CONSIDERATIONS

☐ To the Editor:

Jennings et al. demonstrated a typical course of uterine artery pseudoaneurysm (UAP) (1). Massive vaginal