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In Reply:—Dr. Lam's inability to antagonize residual diplopia with neostigmine after atracurium administration is fascinating because we have had a similar experience. Before the 10 cases that we reported,[1] we did a pilot study using rocuronium as the test drug. One individual (a man aged 26 years and weighing 70 kg) complained of pronounced visual disturbances despite a measured train-of-four (TOF) fade ratio of 0.93 at the end of the study. At this time, the subject was given 0.4 mg of atropine and 5.0 mg of edrophonium intravenously. The TOF ratio promptly returned to a value of 1.00, but the subject reported that if anything his vision got worse. Blurred vision persisted for an additional 60 min.

This observation, if it can be reproduced, raises several questions. What is the effect (if any) of intravenous atropine, glycopyrrolate, neostigmine, and edrophonium alone or in combination on visual acuity and extraocular muscle function? Is it advisable to attempt to reverse diplopia if that is the sole residual effect of an administered relaxant? Is it even possible to do so? Certainly this is an area deserving of further investigation.

The question of whether persistent visual disturbances after the use of nondepolarizing relaxants represents "residual weakness" or something else is probably best left to semanticists. I would not dismiss the importance of these symptoms as lightly as Dr. Lam. The issue is not simply our comfort with the extent of neuromuscular recovery. Should not patient satisfaction enter into the equation as well?

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In Reply:—Thank you for the opportunity to comment on Dr. Lam's correspondence and very interesting observations. Dr. Lam correctly identified that subjective symptoms of visual changes such as diplopia are "obviously common, yet always overlooked, . . ." More importantly, he reports that his own symptoms of diplopia (after participation as a volunteer in an electromyographic study[1]) persisted for 60 min after self-administering anticholinesterase reversal.

Although his questions were rhetorical, I would nevertheless like to respond: the persistence of diplopia[2,3] was surprising because in some cases it was evident for up to 90 min after the train-of-four (TOF) ratio had returned to a value of 1.0. This persistence was evident after administration of a drug (mivacurium) that has a spontaneous recovery index of 7–8 min. This is as surprising as Dr. Lam's finding that atracurium-induced diplopia was not improved by anticholinesterase reversal. As to whether "it is important or necessary to have complete recovery of eye functions before we discharge patients home," it is perhaps not imperative to do so if patients received an ultra-short-acting muscle relaxant. Would we feel as comfortable discharging our patients after administration of one of the older (and cheaper), long-acting relaxants, as we are increasingly being "encouraged" to do?

Finally, as to whether we warn ambulatory patients about "persistent visual disturbances," and not interpret them as "residual weakness," it is really a matter of semantics. I doubt that the patients' subjective symptoms would be dramatically improved by our warning, regardless of what we call these symptoms. In the present era of expanding ambulatory surgery, when emphasis is placed on rapid recovery, quick discharge, and patient satisfaction scores, even "persistent visual disturbances" may be perceived by our patients (and managed care organizations) as undesirable.

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American Heart Association Recommendations for Treating Tricyclic Antidepressant-induced Hypotension

To the Editor:—Recently, in their case report "Treating Intraoperative Hypotension in a Patient on Long-term Tricyclic Antidepressants: A Case of Aborted Aortic Surgery," Sprung *et al.*[1] concluded that potent, direct-acting sympathomimetics may be the only effective management of hypotension in a patient on long-term tricyclic antidepressant (TCA) therapy.

Sprung *et al.* reason that potent direct-acting sympathomimetics may be the only effective management for TCA-induced hypotension because the adrenergic receptors are either desensitized or because catecholamine stores have been depleted in patients who have received TCAs long term.

An important recommendation of the American Heart Association (AHA) has been omitted from this case report. The AHA recommends that serum alkalinization be the mainstay for treating seriously ill patients with signs of TCA toxicity.

Cardiovascular side effects are rare when tricyclic antidepressants are taken in therapeutic dosages.[2] However, Shannon *et al.* and others found a lack of association between TCA level and blood pressure, such that hypotension, even fatal dysrhythmias, may appear with routine doses at therapeutic serum levels.[3,4] Tricyclic antidepressants are the number one cause of death from overdosage in patients who present to the hospital alive.[2]

The electrocardiographic and hemodynamic warning signs of TCA toxicity are almost identical to those signs seen with therapeutic TCA doses.[5] They are sinus tachycardia, prolonged PR, QRS, QT intervals, ST-T changes, bundle branch block, arrhythmias, second and third degree AV block, postural hypotension, decreased myocardial contractility, congestive heart failure, myocardial infarction, and sudden death.

The AHA's recommendation for managing hypotension resulting from TCAs is to first administer 1 l of intravenous saline. If this fails, the next step is to increase the serum pH to 7.5–7.55. Patients with refractory hypotension may then be treated with dopamine or norepinephrine infusion. The protocol of alkalinization of an unstable patient is the following:

1. Increase pH to 7.45–7.55 with 1 mEq/kg of sodium bicarbonate given over 1 to 2 min.
2. Analyze arterial blood gas levels to confirm pH elevation.
3. Place patient on an infusion of two ampules (50–100 mEq) of sodium bicarbonate in normal saline solution (0.9NS).
4. Run the infusion at 150–200 ml/h until the patient stabilizes, until QRS is less than 100 ms, and until arrhythmia ceases and blood pressure normalizes.
5. Maintain the patient's pH at 7.45–7.55 by routine venous or arterial pH measurements.

Alkalinization decreases the non–protein-bound form of the drug. Alkalinization is the AHA's recommended first pharmacologic maneuver for treating seriously ill patients with TCA-induced cardiovascular changes.

Although Dr. Sprung's patient was not "seriously ill" as a result of TCA toxicity, the proposed surgery was aborted because of early blood pressure changes requiring infusion of a potent vasoactive drug. After induction of anesthesia, it became important to correct the hemodynamic changes that had occurred.

The patient took his usual dose of nortriptyline the morning of surgery. Toxicity of TCAs is expected within 2 h and less than 6 h after ingestion. I suggest nortriptyline bioavailability was present. It was present in the holding room when the abnormal electrocardiographic tracing was obtained and was present in the serum after induction of anesthesia. Therefore, it is reasonable to expect some degree of cardiovascular correction with serum alkalinization.

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