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Central Anticholinergic Syndrome: Does It Exist?

To the Editor:—I was fascinated with Grum and Osborne's case report.[1] The number of preoperative tests were impressive, and their work-up for possible use of "drugs" was commendable.

From the signs and symptoms presented, I could not help but suspect that their patient was suffering from symptoms related to pheochromocytoma. Hypertension, tachycardia, pupillary dilation, agitation, and severe headaches all point to increased circulating catecholamines. The "dry and warm" skin is not typical of pheochromocytoma, but the patient had received glycopyrrolate, which causes dry and at times flushed skin.

It is difficult to believe that atropine, which has been in use for more than 100 yr and is still prescribed for millions of patients every day, would suddenly become so vicious and bring about the frightful syndrome called central anticholinergic syndrome! I for one, if confronted with such a clinical episode, will not diagnose "central anticholinergic syndrome" unless I have ruled out pheochromocytoma.

The author thanks Dr. Benjamin G. Covino for reviewing and correcting this correspondence.

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In Reply:—As Dr. Shamsai points out, pheochromocytoma can be considered in the differential diagnosis of a patient who suddenly presents with sudden onset of severe hypertension, tachycardia, headache, and agitation. However, there were reasons not to suspect this diagnosis in our patient, and we did not test for it. Whereas symptoms of pheochromocytoma are often provoked by activity, our patient was resting in bed while awaiting surgery. The patient had a negative family history for systemic diseases that are often associated with pheochromocytoma. During her preoperative work-up, she denied a history of cardiovascular symptoms. More compelling is her denial of previously having had a similar incident. This negative history was corroborated by a family member. The sweating that commonly occurs during an acute attack was absent, and we believe that glycopyrrolate is likely to have produced her dry skin and mucous membranes within a few minutes of administration. Although symptoms of acute catecholamine release from a pheochromocytoma may last only a few minutes, as stated in the case report the first dose of physostigmine given at the height of the patient's symptoms resulted in an immediate and dramatic decrease in her blood pressure and in the severity of her headache. A second dose given 10 min later virtually completely ablated her symptoms and physical findings. The suggestion that the episode self-terminated over a period of one half hour is not supported by the case description.

Thus, although we cannot rule out the presence of pheochromocytoma in this patient, we believe that the immediate temporal relationship between physostigmine and the cessation of the patient's symptoms argues for acute central anticholinergic syndrome following glycopyrrolate. I personally have seen two prior cardiovascular and neurologic crises immediately following administration of an antimuscarinic drug that were promptly terminated by physostigmine, and I doubt that it was mere coincidence in either case. Our report emphasizes that central anticholinergic syndrome, like pheochromocytoma, does exist and may occur more often than is commonly suspected.

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Epidural Opioid Requirements

To the Editor:—We read with interest the case report by Kreitzman and Samuels.[1] While we understand that the main point in the report is to document this patient's response to a high dose of epidural hydromorphone, we feel some issues must be clarified.

First, there was no mention as to whether this patient used opioids for pain control in the preoperative period. If he had, which drug was used, what dose, and for how long? Second, was the catheter placed in the thoracic or in the lumbar area, and was correct position of the