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# Catecholamine Surge in Opioid-addicted Patients Undergoing Detoxification under General Anesthesia

*To the Editor:*—We read with interest the article by Kienbaum et al.[1] regarding the hypothesis that u-opioid receptor blockade by naloxone induces cardiovascular stimulation mediated by the sympathoadrenal system.

This study confirms that clinicians who have detoxified heroin addicts for 20 yr have known, *i.e.,* clonidine, an $\alpha_2$-agonist, is essential to avoiding hyperadrengic crisis and pulmonary edema. Riordan and Kleber[2] in 1980 first demonstrated that utility of clonidine in controlling the hemodynamic changes seen in the withdrawal syndrome. Naloxone has also been previously associated with pulmonary edema[3,4] presently because of this sympathoadrenal surge.

It is very dramatic to document the extent of catacholamine secretion. This has been elegantly demonstrated in the study by Kienbaum et al.[1] We also agree with the authors that because of this cardiovascular stimulation secondary to a surge in sympathoadrenal system, the procedure of acute opioid detoxification should be done by trained anesthesiologists in an intensive care setting. However, in order to make this procedure safe, it is imperative to use an $\alpha_2$-agonist, such as clonidine before $\mu$-opioid receptor blockade, even if the patient is under general anesthesia.

**Clifford Gevirtz, M.D.**
Associate Professor
**Dilip V. Subhedar, M.D.**
Assistant Professor

**Chang S. Choi, M.D.**
Assistant Professor
Metropolitan Hospital Center
New York Medical College
Department of Anesthesiology
New York, New York

## References

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*In Reply:*—We appreciate the interest of Drs. Gevirtz, Subhedar, and Choi in our recent publication.[1]

On the clinical side, consistent with our experience, clonidine reduces withdrawal symptoms during detoxification. Accordingly, it has been favourably administered during detoxification from opioids in *awake* addicts for more than 20 yr.[2] However, it has been claimed by psychiatrists that cardiovascular responses and withdrawal symptoms to detoxification from opioids both can be minimized by administration of large amounts of $\mu$-opioid receptor antagonists alone when administered during sedation or general anesthesia.[3,4] In our study, we had to reject this hypothesis by demonstrating extensive increases in catecholamine concentrations in plasma and cardiovascular stimulation associated with $\mu$-opioid receptor blockade by naloxone during general anesthesia. Furthermore, the need for additional drugs such as $\alpha_2$-adrenergic agonists, *e.g.,* clonidine, was described.[1]

We pointed attention to the fact that (1) marked sympathoadrenal activation and cardiovascular stimulation may be observed during detoxification from opioids by administration of $\mu$-opioid receptor antagonists alone, despite deep general anesthesia, and (2) the need for trained anesthesiologists/intensivists in performing this treatment, as also proposed by Dr. Gevirtz and coworkers. Whether and why clonidine helps in minimizing cardiovascular and sympathoadrenal stimulation during detoxification even during conditions of general anesthesia is the subject of ongoing investigation.

**Peter Kienbaum, M.D.**
Research Fellow and Resident
**Jürgen Peters, M.D.**
Professor of Anesthesiology and Intensive Care Therapy and Chairman
Abteilung für Anästhesiologie und Intensivmedizin
Universitätsklinikum GH
Essen, Germany
peter.kienbaum@uni-essen.de