When these channels are activated, it typically produces the sensation of cold and an appropriate physiologic response. However, menthol activation of the TRP-M8 ion channel in the nose is interpreted in the brain as a sensation of increased airflow. While opioids truly block pain, this sensation of increased airflow triggered by menthol is associated with no change in airflow. The clearest description of this would be “fooling the brain.”

Finally, on January 9, 2009, while this article was under embargo, Dr. Paul wrote to Mr. Ray Kotera, Director of Technical and Medical Services at the American Academy of Pediatrics (AAP), asking that he share comments similar to those in his letter with key physicians in the AAP. Dr. Paul admitted having a conflict of interest and said he would not be commenting to the media but that his talking points might help others respond to the media. However, within days Dr. Paul gave many media interviews. He told MSNBC “this article is at best incomplete and at worst irresponsible.” He told National Public Radio that he takes issue with this study stating, “People for one hundred years have been using VVR and I hear from parents that their parents used it when they were kids and their parents when they were kids.” Dr. Paul was quoted by the Business Courier of Cincinnati that this manuscript was a “real stretch.” “It’s really unbelievable... and it really calls into question for me that they had an agenda here,” he said.

Unlike Dr. Paul, who has received funding from P&G to study VVR in 150 children to determine if this makes then feel better, I could not obtain funding to conduct these studies. This research was performed by Drs. Abanses and Arima using discretionary funds that I pulled together. I am delighted that both of these men are now academic clinician-scientists. If this was my “agenda,” I am happy to confess. I have been impressed with the response of P&G confirming that VVR should never be used in children under the age of 2 nor placed under or in the nose of anyone. They have expressed a genuine interest in learning more about the phenomena that we described. Since the time of publication of this article, there have been nearly 30 additional cases reported to me by parents and physicians from around the world. With the cooperation of P&G, I have urged each of these physicians and parents to report their observations directly to the company.

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Acute Management of Atrial Fibrillation

To the Editor:

Khoo and Lip (March 2009) have reviewed the therapy of acute atrial fibrillation (AAF) extensively. Patients presenting with AAF are very heterogeneous; however, all may show spontaneous conversion to sinus rhythm. The heterogeneity of the disease and its underlying mechanisms makes studies, reviews, and guidelines very complicated and confusing. The review by Khoo and Lip suggests that AAF leading to critical illness is the same as critical illness leading to AAF, and that, therefore, the therapeutic approach can also be the same. In our view, this is not correct.

AAF is a multifactorial disease involving structural cardiac abnormalities, inflammation, electrolyte disturbances, hormonal and autonomous nervous system dysregulation, and fluid imbalance, among others, as underlying causes. A difference in balance between causes in the two types of patients is more than likely. For example, arrhythmias can only develop by the combination of a trigger and a substrate. In outpatient clinic patients with AAF, there might be a larger role for the substrate, while in critically ill patients with AAF the trigger is of utmost importance. In critically ill patients, the underlying trigger should be treated first. Treatment of the underlying disease, pain and anxiety relief, oxygenation, and correction of hemodynamics are mandatory, and this essential therapy leads to conversion to sinus rhythm in the majority of cases without further intervention. Direct current cardioversion, based on our experience and supported by the literature, has no sustainable result if the underlying triggers are not eliminated. Direct current cardioversion therefore, should only be used in really desperate situations, although, as mentioned before, the efficacy remains debatable.

The best pharmacotherapeutic approach is also a point of discussion. However, much experience with a few drugs is probably better than little experience with many drugs, and amiodarone might be the drug with the best range. Most antiarrhythmic drugs, and especially class I drugs, have never been tested in critically ill patients, and their potential adverse effects might be exaggerated just in these patients. Whether magnesium is really effective or just buys time to spontaneous conversion remains to be elucidated. Critically ill patients may have thrombophilia, but they certainly have a high risk of bleeding. This means that studies and guidelines about prophylaxis for the prevention of thromboembolism for outpatients or other patients cannot be extrapolated. There is no evidence we can rely on for anticoagulant therapy in patients with AAF due to critical illness.

In conclusion, the paucity of data about AAF in critically ill patients should not lead to the extrapolation of guidelines from...
The consequences of acute AF mainly relate to hemodynamic and thromboembolic effects. Patients who are already critically ill are frequently at risk for hemodynamic instability and thromboembolism. The risk for these clinical sequelae with the development of acute AF is even higher. Hence, the appropriate management of AF in these patients is paramount. Pharmacologic antiarrhythmic agents that can be used in these patients are limited especially if heart failure and structural heart disease are present.

The development of AF in critically ill patients would also increase the risk of thromboembolism in this “high-risk” group. Unfortunately, there are no clinical trials assessing the role of anticoagulation in these patients per se, and the clinical assessment of stroke risk using current risk stratification schema have not been validated in this situation. However, thromboprophylaxis using heparin (low molecular weight or unfractionated) should be initiated, pending stabilization of the clinical situation and appropriate investigation and more long-term treatment decisions.2

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Response

To the Editor:

We thank Sleeswijk et al1 for their comments on our recent review article “Acute Management of Atrial Fibrillation.”2 Atrial fibrillation (AF) is the most commonly encountered arrhythmia in clinical practice and is also common in critically ill patients.3,4 The development of AF is multifactorial and often involves the combination of triggers and substrates. Approximately 50% of the patients who present with acute AF will revert back to sinus rhythm within 48 h,6 and this is more likely if the underlying etiology (trigger) can be identified and treated.

We do agree that acute AF that leads to critical illness may be different from critical illness leading to acute AF. The former involves proactive treatment of the arrhythmias to prevent the development of critical illness or complications, while the latter involves treatment of the underlying critical illness to prevent the development of AF. Our review is particularly directed toward the management of critical illness as a consequence of acute AF.2

REFERENCES