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In reply:

We thank Dr. Shiber for his insightful comments. He raises the excellent point that the incidence of seizure after intracerebral hemorrhage appears to be strongly associated with the location of hemorrhage. To the extent that the risk of seizure varies with bleeding location, it is further possible that the absolute risk reduction in seizure incidence achieved by antiepileptic medications similarly varies with the anatomic location of hemorrhage. Dr. Shiber further raises the possibility that the effect size of antiseizure activity may vary across different antiepileptic medications.

Regarding the relationship between anatomic intracranial bleeding location and the risk of seizure, we acknowledge that such an association is evident in the literature.[1] Dr. Shiber rightly notes that the meta-analysis we summarized in the Systematic Review Snapshot does not stratify its analysis according to bleeding location.[2] That said, the individual studies permit differentiation of patients according to bleeding anatomy.[3] It would be a compelling addition to the literature to examine whether there is any effect modification for antiepileptic effect related to bleeding location.

The existing meta-analysis also does not establish comparative effectiveness of alternative antiepileptic agents. For inclusion into the meta-analysis, the authors required that studies compare the use of prophylactic antiepileptic drugs with no prevention. Consequently, individual studies included in the meta-analysis have little to offer by way of evidence showing that any particular medication is superior to another.[2] Given these data limitations, it is not unreasonable to base the choice of drug on the adverse-effect profiles of each medication, as Dr. Shiber has done. That said, an alternative approach would be to use a network meta-analysis to compare different treatments by including indirect comparisons across different trials based on a common comparator.[4] We believe that a future network meta-analysis of these trials to better ascertain the comparative efficacy of specific antiepileptic drugs in patients with intracranial hemorrhage would be a valuable contribution to the literature.

This review does not reflect the views or opinions of the US government, Department of Defense, US Army, US Air Force, San Antonio Uniformed Services Health Education Consortium, or the Fort Carson Post Command.

Michael D. April, MD, DPhil
Department of Military and Emergency Medicine
Uniformed Services University of the Health Sciences
Bethesda, MD
2nd Brigade, 4th Infantry Division
Fort Carson, CO

Brit Long, MD
Department of Military and Emergency Medicine
Uniformed Services University of the Health Sciences
Bethesda, MD
Department of Emergency Medicine
San Antonio Uniformed Services Health Education Consortium
Fort Sam Houston, TX

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Acute Olfactory Loss Is Specific for COVID-19 at the Emergency Department

To the Editor:

Olfactory loss as a symptom of coronavirus disease 2019 (COVID-19) has been receiving increasing attention globally, with a number of international statements including it as a key symptom of the disease.[1-4]

In response, the emergency department (ED) at Sengkang General Hospital, a tertiary care hospital in Singapore, began actively inquiring about acute olfactory loss (hyposmia or anosmia of less than 14 days' duration) from April 23, 2020, for all patients who presented with

Table. Olfactory loss in patients presenting to the ED who met criteria for COVID-19 swab.

	COVID-19 Positive	COVID-19 Negative
Olfactory loss		
Hyposmia	3	8
Anosmia	4	14
No olfactory loss	24	664

acute respiratory symptoms and for those who fulfilled the prevailing Ministry of Health suspect or surveillance case definition. We then performed a cohort study to evaluate the utility of acute olfactory loss as a risk-stratifying tool for COVID-19.

A chart review was performed for all patients meeting the above criteria who presented between March 23, 2020, and April 4, 2020. All patients had a COVID-19 polymerase chain reaction oropharyngeal swab performed. We excluded patients with preexisting olfactory loss and those who were unable to give a history of olfactory loss reliably (eg, those with cognitive impairment).

A total of 717 patients met these criteria, and 31 had a positive test result for COVID-19 by polymerase chain reaction (Table). In this group, 7 (22.6%) complained of acute olfactory loss, of whom 3 (42.9%) had hyposmia and 4 (57.1%) had anosmia. One patient presented with isolated anosmia, and the rest had olfactory loss associated with other symptoms of acute upper respiratory tract infection. Of 686 patients who had a negative test result for COVID-19, 22 (3.2%) had acute olfactory loss (χ^2 test; $P<.05$). Within this group, 8 patients (36.4%) had hyposmia, whereas 14 (63.6%) had anosmia. One patient had isolated hyposmia. Acute olfactory loss as a marker for COVID-19 had a sensitivity of 22.6% and a specificity of 96.8%. In this cohort, the positive predictive value of acute olfactory loss for COVID-19 was 24.1% and the negative predictive value was 96.5%.

To the best of our knowledge, this is the first study that analyzes prospectively collected hyposmia data in a single cohort of COVID-19–positive and –negative patients. Our results echo those of other studies examining the prevalence of chemosensory dysfunction in COVID-19.[5] Our study is limited by the lack of objective olfactory testing, and although self-reported olfactory ability has been found to be not completely reliable, exigencies of service in the busy ED did not permit formal olfactory assessment. Data on olfactory loss developing in patients after their initial presentation were also not captured because our study was focused on the usefulness of acute olfactory loss for COVID-19 risk stratification at the ED.

The Ministry of Health suspect case definition has since been expanded to include anosmia as of April 15, 2020. Our findings support this new inclusion and suggest that it has a strong specificity for COVID-19, making it useful as a rule-in test especially in EDs to influence cohorting and isolation decisions for which testing is unavailable or results are pending.

Andy J. Chua, MBBS, MMed
Tze Choong Charn, MBBS, MMed
Department of Otolaryngology
Sengkang General Hospital
Singapore

Eunice C. Chan, MBBS, MCEM
Department of Emergency Medicine
Sengkang General Hospital
Singapore

Jiashen Loh, MBBS, MRCP
Department of Infectious Disease
Sengkang General Hospital
Singapore

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