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Syncope confusion

Soong and colleagues' intent was to highlight the overuse of investigations, particularly neuroimaging, among patients with syncope.¹ Although Soong and colleagues cited the 2009 European Society of Cardiology guideline,² they failed to differentiate syncope from other causes of transient loss of consciousness. Syncope is caused by global cerebral hypoperfusion, and none of the listed neurologic diagnoses cause syncope.

This confusion has led to great research efforts, consensus conferences, guidelines and statements developed by the European Society of Cardiology, the Gargnano multidisciplinary consensus conference (led by internists), and the Canadian Cardiovascular Society, all of which uniformly exclude neurologic conditions causing transient loss of consciousness from syncope.²⁻⁵

Based on current evidence, syncope is defined as a transient loss of consciousness due to global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery. Sadly, this article¹ worsens the confusion by clearly stating that neurologic causes of syncope exist. This could cause practising physicians to include neurologic causes in the differential diagnosis for true syncope, and to not differentiate syncope from other causes of transient loss of consciousness. This is important, as the literature evidence regarding the risk of "cardiac syncope," and use of neuroimaging exist only for true syncope patients and cannot be applied to all patients with transient loss of consciousness. The authors could also have cited new evi-

dence for high-risk features for "cardiac syncope" that have been summarized, based on evidence.^{6,7}

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The authors respond

We thank Thiruganasambandamoorthy and Sheldon for clarifying the definition of syncope as a transient loss of consciousness due to global cerebral hypoperfusion characterized by rapid onset, short duration and spontaneous complete recovery.¹ However, experts acknowledge that this approach has pragmatic limitations, particularly when applied to undifferentiated patient presentations in the emergency department. To guide clinicians in the emergency department, our approach focuses on all potential causes of transient loss of consciousness, including syncope "mimickers." We agree that stroke, transient loss of consciousness, seizure and metabolic disturbances do not represent true syncope.

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Group A streptococcus

It was gratifying to read that the authors of this commentary¹ on group A streptococcus mention the significant rate of colonization versus infection. Surprisingly, this was not mentioned in the related research article.² This continuing uncertainty, so appropriate in science, highlights the need for the art of medicine — the art in which clinicians dance with the complexity of uncertainty, balance their sense of whether the child is quite ill ("toxic") or otherwise medically fragile, converse with parents to assess their resourcefulness and preferences, and balance all of this with the public health issues. I would appreciate a review of the implications of the treatment of carrier states, with respect to group A streptococcus in particular.

Another *CMAJ* paper,³ examining the potential harms of the use of amoxicillin and amoxicillin-clavulanic acid, also surprised me by treating the two drugs as if they were similar. I understand that the latter is one of the broadest spectrum agents, and one I reserve for very specific situations. I am of the old school, and I still do not even use amoxicillin for group A streptococcus, preferring penicillin V (which is often not even available in the suspension form).

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