Clarification — "Pretreatment" in RSI

To the Editor: In the July issue of *CJEM*, Drs. Zed, Harrison and Kuzak[1] expressed disappointment with our opinion[2] that pre-treatment for rapid sequence intubation (RSI) is contraindicated. We would like to clarify that our correspondence regarding this practice in no way implied that pretreatment for RSI is contraindicated. The use of this term misrepresents material taught in our course. Our point is that, athough pretreatment may have benefit in certain circumstances, these remain poorly defined and the risk:benefit ratio of this therapy remains unproven. Pretreatment for RSI should not be allowed to complicate or delay timely airway management. Athough it is not "contraindicated," it should not be used as an indicator of "quality of care."

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References
1. Zed PJ, Harrison DW, Kuzak N. Pretreatment in rapid sequence intubation: Indicated or contraindicated? [letter]. Can J Emerg Med 2006;8(4):243-4.
2. Kovacs G, MacQuarrie K. Campbell S; and the AIME Instructors. Pretreatment in rapid sequence intubation: Indicated or contraindicated? [letter]. Can J Emerg Med 2006;8(4):243.
3. Grant: Should Kuzak et al's original article be cited along with ref 2? (i.e., pages 80 to 84 of the March issue).

AMI after epinephrine

To the Editor: From the point of view of a seasoned veteran with little academic training but a lot of experience working in the trenches I would like to comment on the case of myocardial infarction after epinephrine that was reported in the July issue of *CJEM*.[1]

I believe it is quite a stretch to think that anaphylaxis was caused by a medication ingested 24 hours prior. I also disagree with the author that ibuprofen infrequently cause severe allergic reactions. I have intubated a young man who arrived apneic after ingesting 200 mg of ibuprofen.

Despite the discussion in the article, I would not use IV epinephrine in a patient who is walking and talking, with normal vital signs. Furthermore, I don't believe this patient had a myocardial infarction. I think a young man that exhibited impressive ST changes on his ECG secondary to an MI would have equally impressive changes in his CK and troponin. More likely, this patient had an episode of coronary vasospasm with minor myocardial injury that caused a slight troponin rise. To elucidate this further, I would think it mandatory to send this patient for urgent angiograms. Finally, I would not give someone in the throes of an anaphylactic reaction ASA. And by the way, would you give this person epinephrine for their next allergic reaction?

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Reference
1. Shaver KJ, Adams C, Weiss SJ. Acute myocardial infarction after administration of low-dose intravenous epinephrine for anaphylaxis. Can J Emerg Med 2006;8(4):289-94.

To the Editor: I have two comments to offer with regard to the recent case report of acute myocardial infarction after administration of low-dose IV epinephrine for anaphylaxis.[1] A variety of routes of administration for epinephrine for anaphylaxis are mentioned in the introduction, including subcutaneous, intravenous and sublingual. The preferred route of administration of epinephrine for most cases of anaphylaxis is now felt to be intramuscular (IM). This is based on a 2001 study showing higher peak plasma epinephrine concentrations with the IM route as opposed to the subcutaneous route (SC).[2]

A more controversial consideration is when to use epinephrine intravenously. This was highlighted for me a few years ago when I sat as an examiner for one of the national qualification examinations. I trust I won't be breaching my exam confidentiality agreement by relaying (without detail) that one of our "exam cases" involved a scenario very much like the one presented by Shaver and colleagues.[1] The examinee had to ascertain that the young, healthy patient was presenting with moderate anaphylaxis, and treat that person with epinephrine. To validate the exam, we had a room full of experienced emergency physicians who would be serving as examiners. We discussed whether administration of IV epinephrine would be an acceptable response. Half of the physicians in the room thought that IV epinephrine would be far too risky a route for this patient. Most in this camp had stories to tell of severe complications with IV epinephrine use in the nonmoribund patient, either personal or relayed by colleagues. I recall administering IV epinephrine to a healthy 40-year-old woman with severe anaphylaxis, as I felt her airway swelling was progressing rapidly. This lady was still awake when she received her 0.1 mg of epinephrine IV, and promptly started screaming and clutching her head. Several of my organs returned to their native position after the CT showed no intracranial bleed, and her

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systolic blood pressure slowly receded from 230. This put me squarely in the first group of nay-sayers to IV epinephrine for the non-moribund patient group.

Interestingly, there were an equal number of physicians who were in the alternative camp of considering IV epinephrine as a first-line agent, even in the moderate anaphylaxis patient. These were experienced emergency physicians, many from teaching or academic settings. The difference in practice was quite striking. In the Discussion section of this article,[1] I was surprised to see the relative paucity of case reports and research on this topic, as well as the conflicting recommendations available on when to move to IV epinephrine. This is certainly an area that bears some further research and clinical scrutiny. I thank Dr. Shaver and colleagues for presenting this interesting case report.

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References

1. Shaver KJ, Adams C, Weiss SJ. Acute myocardial Infarction after administration of low-dose intravenous epinephrine for anaphylaxis. Can J Emerg Med 2006;8(4):289-94.
2. Simons FE, Gu X, Simons KJ. Epinephrine absorption in adults: intramuscular versus subcutaneous injection. J Allergy Clin Immunol 2001;108(5): 871-3.

Erratum

There was an error in the Diagnostic Challenge[1] in the July issue of *CJEM*. In the last paragraph of the Commentary the article stated: "Interestingly, there have been no reports of SIPE [swimming-induced pulmonary edema] in Olympic swimmers, but there is one case published of a triathelete who developed dyspnea with slight hypoxia and right-sided pulmonary crackles some 8–9 hours after the swim." Eight to nine hours was in fact, when the patient presented to the ED, not the time frame during which symptoms developed.

Reference

1. Deady B, Glezos J, Blackie S. A swimmer's wheeze. Can J Emerg Med 2006; 8(4):281; 297-8.

Letters will be considered for publication if they relate to topics of interest to emergency physicians in urban, rural, community or academic settings. Letters responding to a previously published *CJEM* article should reach *CJEM* head office in Vancouver (see masthead for details) within 6 weeks of the article's publication. Letters should be limited to 400 words and 5 references. For reasons of space, letters may be edited for brevity and clarity.

Les lettres seront considérées pour publication si elles sont pertinentes à la médecine d'urgence en milieu urbain, rural, communautaire ou universitaire. Les lettres en réponse à des articles du *JCMU* publiés antérieurement devraient parvenir au siège social du *JCMU* à Vancouver (voir titre pour plus de détails) moins de six semaines après la parution de l'article en question. Les lettres ne devraient pas avoir plus de 400 mots et cinq références. Pour des raisons d'espace et par souci de concision et de clarté, certaines lettres pourraient être modifiées.

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