either diphenhydramine or a nonsedating antihistamine such as cetirizine (10 mg daily for 3 days, given orally) would be appropriate. Although H_2 receptors are involved to a limited extent in the pathophysiology of anaphylaxis, we rarely administer H_2 blockers in this capacity and consider their use optional. With respect to prophylactic corticosteroids, we usually begin with a single oral dose of 50 mg prednisone, followed by reduced doses of 40 mg on days 2 and 3, then 20 mg on days 4 and 5, then discontinuation. The medication is given in the morning to optimize effect and minimize adrenocortical suppression.

The questions surrounding cross-reactivity of penicillin and third-generation cephalosporins remain unresolved. Allergic reactivity to cephalosporins (of any type) is 4 to 8 times greater in patients with a history of allergy to penicillin than in those without,[2,3] and the rate of reactivity to cephalosporins is 4% to 7% in patients with previous reactivity to penicillin.[3] Recent evidence suggests that variable side-chains on the β-lactam ring, rather than the β-lactam nucleus, induce this cross-reactivity.[4,5] Indeed, several patients with documented skin test reactivity to penicillin who were given doses of second- and third-generation cephalosporins had no reactivity.[6] Nevertheless, caution is advised in the administration of cephalosporins to patients with known anaphylactic reactivity to penicillin.

We agree that the designation 1:1000 or 1:10 000 can be confusing, but this description facilitates rapid dosing and administration of epinephrine, which is essential in managing anaphylaxis.[7] In addition, this presentation displays the dilution much more prominently than if the dose is given as milligrams per millilitre (mg/mL).

Patrick Potter raises the issue of empiric use of glucagon for treatment-resistant anaphylaxis. Epinephrine resistance in anaphylaxis does suggest concomitant β-blockade and hence an indication for glucagon administration. However, glucagon may be associated with nausea, vomiting, hyperglycemia and allergic reactivity, which precludes its general use in anaphylaxis. If repeat doses of epinephrine yield inadequate clinical response during an episode of anaphylaxis, especially when there is evidence of increasing systolic hypertension due to unopposed α-adrenergic activation and bradycardia signifying reflex vagotonic effect, further epinephrine is contraindicated and glucagon should be administered.

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Competing interests: None declared.

Corrections

In a recent commentary summarizing the updated recommendations for the management of dyslipidemia and the prevention of cardiovascular disease,[1] the year in which the report of the US National Cholesterol Education Program Adult Treatment Panel-III was published was given incorrectly as 2002; this report was in fact published in 2001.

In addition, in the appendix to the commentary, certain symbols are missing from the 3 tables on page 923 and, in the paragraph on diet (page 924), the body mass index to be achieved and maintained should be less than 25 kg/m^2. A corrected version of the appendix has been posted online at www.cmaj.ca/cgi/content/full/169/9/921/DC2.

Reference

1. Genest J, Frohlich J, Fodor G, McPherson R (the Working Group on Hypercholesterolemia and Other Dyslipidemias). Recommendations for the management of dyslipidemia and the prevention of cardiovascular disease: summary of the 2003 update [editorial]. *CMAJ* 2003;169(9):921-4.

In the recent article on high-altitude decompression illness by Michael Allan and David Kenny,[1] the map showing locations of hyperbaric facilities in Canada did not include the Hyperbaric Medical Centre of the Hôtel-Dieu de Lévis, Centre hospitalier affilié à l'Université Laval, located in Lévis, Que. Physicians at this centre can be reached at 418 835-7121.

The Undersea and Hyperbaric Medicine Society offers a directory of hyperbaric chambers and facilities in North and Central America through its Web site (www.uhms.org).

Reference

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