23. Delage JM, Bergeron P, Simard J, et al. Hereditary C7 deficiency. Diagnosis and HLA studies in a French-Canadian family. *J Clin Invest* 60:1061–1069, 1977.
24. Zeitz HJ, Miller GW, Lint TF, et al. Deficiency of C7 with systemic lupus erythematosus: Solubilization of immune complexes in complement-deficient sera. *Arthritis Rheum* 24:87–93, 1981.
25. Alcalay M, Bontoux D, Peltier A, et al. C7 deficiency, abnormal platelet aggregation, and rheumatoid arthritis. *Arthritis Rheum* 24:102–103, 1981.
26. Kojima K, Sasaki A, Yokomatsu Y, et al. Deficiency of the seventh component of complement with systemic lupus erythematosus. *Osaka City Med J* 31:121–128, 1985.
27. Segurado OG, Arnaiz-Villena AA, Iglesias-Casarrubios P, et al. Combined total deficiency of C7 and C4B with systemic lupus erythematosus (SLE). *Clin Exp Immunol* 87:410–414, 1992.
28. Amano T, Miyashima H, Mitsuhashi Y, et al. Deficiency of the 9th component of complement associated with rheumatoid arthritis. *Jpn J Clin Immunol* 4:206–211, 1981.
29. Sugimoto M, Nishikai M, Sato A, et al. SLE-like and sicca symptoms in late component (C9) complement deficiency. *Ann Rheum Dis* 46:153–155, 1987.
30. Kawai T, Katoh K, Narita M, et al. Deficiency of the 9th component of complement (C9) in a patient with systemic lupus erythematosus. *J Rheumatol* 16:542–543, 1989.
31. Takeda I, Igarashi S, Nishimaki T, and Kasukawa R. A case of systemic lupus erythematosus in late component (C9) complement deficiency. *Ryumachi* 34:628–632, 1994.
32. Ichikawa E, Furuta J, Kawachi Y, et al. Hereditary complement (C9) deficiency associated with dermatomyositis. *Br J Dermatol* 144:1080–1083, 2001.
33. Inai S, Akagaki Y, Moriyama T, et al. Inherited deficiencies of the late-acting complement components other than C9 found among healthy blood donors. *Int Arch Allergy Appl Immunol* 90:274–279, 1989.
34. Fukumori Y, Yoshimura K, Ohnoki S, et al. A high incidence of C9 deficiency among healthy blood donors in Osaka, Japan. *Int Immunol* 1:85–89, 1989.
35. Zhu Z, Atkinson TP, Hovanky KT, et al. High prevalence of complement component C6 deficiency among African-Americans in the south-eastern USA. *Clin Exp Immunol* 119:305–310, 2000.
36. Orren A, Owen EP, Henderson HE, et al. Complete deficiency of the sixth complement component (C6Q0), susceptibility to *Neisseria meningitidis* infections and analysis of the frequencies of C6Q0 gene defects in South Africans. *Clin Exp Immunol* 167:459–471, 2012.. □

Erratum

Assessment of fractionated exhaled nitric oxide as a biomarker for the treatment of eosinophilic esophagitis

John Leung, M.D., Ann Nguyen-Traxler, M.D., Erika M. Lee, M.D., Jason S. Yip, M.D., Joel V. Weinstock, M.D., Walter W. Chan, M.D., Peter Ngo, M.D., Barbara J. Weinstein, M.D., and Peter A. Bonis, M.D.

On p 519 of the November-December 2012 issue, the Abstract printed incorrectly. The following is the correct version of the Abstract:

Diagnosis of eosinophilic esophagitis (EoE) and determination of response to therapy is based on histological assessment of the esophagus, which requires upper endoscopy. In children, in whom a dietary approach is commonly used, multiple endoscopies are needed, because foods are eliminated and then gradually reintroduced. Ideally, noninvasive methods could supplement or replace upper endoscopy to facilitate management. Fractionated exhaled nitric oxide (FeNO) has been proposed as a useful measure for monitoring disease activity in studies of patients with eosinophil-predominant asthma and in other atopic disorders. Thus, we evaluated whether FeNO levels could be a useful biomarker to assess the response to therapy in EoE patients. This study was designed to determine whether there is a change in FeNO levels during treatment with topical corticosteroids and whether changes correlated with clinical response. This was a prospective, multicenter study that enrolled nonasthmatic patients with established EoE. FeNO levels and symptom scores were measured at baseline, biweekly during 6-week swallowed fluticasone treatment, and 4 weeks posttreatment. Twelve patients completed the trial. We found a statistically significant difference between median pre- and posttreatment FeNO levels [20.3 ppb (16.0–29.0 ppb) vs 17.6 ppb (11.7–27.3 ppb), $p=0.009$]. However, neither the pretreatment FeNO level, a change of FeNO level after 2 weeks of treatment, nor the FeNO level at the end of treatment confidently predicted a clinical or histological response. Although our findings suggest nitric oxide possibly has a physiological role in EoE, our observations do not support a role of FeNo determination for management of EoE.

(doi: 10.2500/aap.2013.34.2233)