

Correspondence

Asthma mortality in patients treated by differently trained physicians

To the Editor:

I have read with interest the initial article by R. Michael Sly, MD,¹ entitled, "Mortality from Asthma, 1979-1984." It appears to me that the real answer to the subject matter at hand would lie in what percentage of patients with asthma that are dying are treated by trained allergists/immunologists and what percentage are not being treated by allergists/immunologists before their death. I have been practicing allergy/immunology for more than 20 years, and I am board certified by the American Board of Allergy and Immunology. In my large practice I do not recall when I last had a death from asthma. In addition, since I am also certified by the American Board of Internal Medicine, Subspecialty Board—Pulmonary Disease, my practice is weighted with subjects with asthma. It would appear to me that, if all patients with major head trauma were treated by obstetricians/gynecologists, the death rate would differ from patients treated by neurologists/neurosurgeons. This is not to cast any aspersions on obstetricians/gynecologists but merely to state that one treats best what one is trained to treat. I would be extremely interested if Dr. Sly has done any investigation regarding changing death rates in asthma by those patients treated by trained allergists/immunologists.

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Reply

To the Editor:

Dr. Krumholz has raised an interesting question regarding the relationship of asthma mortality to medical care. These data I reviewed were derived from information supplied on death certificates.¹ Accordingly, no information about sources of treatment was available.

Other investigators have identified inadequate care as a risk factor for death from asthma.²⁻⁴ Some deaths apparently have been unavoidable despite optimal medical care by current standards.

No data relating asthma mortality to care by medical specialty have been published, but deaths from asthma have occurred in patients cared for by trained allergists. Asthma mortality rates are so low that fatalities in single physician's practices are rare.

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REFERENCES

1. Sly RM. Mortality from asthma, 1979-1984. *J ALLERGY CLIN IMMUNOL* 1988;82:705-17.
2. Ormerod LP, Stableforth DE. Asthma mortality in Birmingham 1975-7: 53 deaths. *Br Med J* 1980;280:687-90.
3. Rea HH, Scragg R, Jackson R, Beaglehole R, Fenwick J, Sutherland DC. A case-control study of deaths from asthma. *Thorax* 1986;41:833-9.
4. Barger LW, Vollmer WM, Felt RW, Buist AS. Further investigation into the recent increase in asthma death rates: a review of 41 asthma deaths in Oregon in 1982. *Ann Allergy* 1988;60:31-9.

Relapsing anaphylaxis to bee sting in a patient treated with β -blocker and Ca blocker

To the Editor:

Severe anaphylaxis has been previously reported in patients receiving β -blocker therapy.¹⁻³ The attacks have been characterized by severe and transient hypotension, bronchospasm, and urticaria-angioedema, which are usually refractory to treatment. Calcium channel blockers (Ca blockers) were reported to worsen anaphylactic shock to radio contrast media.⁴ We would like to report an unusual reaction in a patient who developed recurrent anaphylaxis (triphasic) reaction to bee sting while the patient was being treated with both β -blocker and Ca-blocker drugs.

The patient was a 52-year-old farmer who was frequently stung by honeybee and yellow jackets with only local reaction. A year before his attack, he was evaluated for chest pain of unknown origin, and because of arteriosclerotic narrowing found on cardiac catheterization, he started receiving 80 mg/day of propranolol (slow release), together with 30 mg of nifedipine. Before his severe attack, he was stung by a honeybee on his neck. A few minutes later, he developed generalized itching and urticaria-angioedema, which in a few minutes progressed to severe anaphylactic shock (undetected blood pressure). He was immediately brought to the local emergency room where he was administered a large amount of intravenous fluids, adrenaline and hydrocortisone. ECG done during the treatment demonstrated mild tachycardia. During the next 30 minutes, he gradually improved and was generally well except for urticaria-angioedema, which still prevailed. Two hours after the initial event, the patient was preparing to leave the emergency room when he started feeling generalized itching, flushing, and dizziness. His blood pressure was again unmeasurable with rapid pulse. He was immediately administered intravenous fluids, adrenaline and hydrocortisone. Once again he gradually improved, gaining complete recovery 20 minutes later. At approximately 4 hours after the initial event, the patient again experienced an anaphylactic reaction similar to reactions he had earlier. With the same

treatment, he recovered 15 minutes later. No evidence for cardiac ischemia could be found.

Two weeks after this event, the patient was referred to the allergy clinic at Ichilov Hospital for evaluation and treatment. He was found to be highly sensitive to intradermal honeybee venom (0.01 mg/ml). At this stage, after consulting with his cardiologist, propranolol was stopped and immunotherapy to bee venom was initiated. Within 6 weeks, maintenance dose was reached without side effects.

The course of anaphylaxis to bee sting was described very elegantly by Smith et al.⁵ They reported only a monophasic response lasting no longer than 40 minutes, but biphasic or protracted anaphylaxis has been observed.⁶ Late anaphylaxis to bee sting has also been recently described.⁷

Our patient had an unusual anaphylactic reaction after a bee sting, first observed as recurrent triphasic drop in blood pressure, together with urticaria-angioedema, while the patient was receiving a β -blocker and a Ca blocker, and in whom previous stings had been uneventful.

The question arises whether the treatment was responsible for this reaction. Severe anaphylaxis has been previously reported in patients receiving β -blockers.¹⁻³ Combined therapy with β -blockers and Ca blockers is frequently used in the treatment of patients with arteriosclerotic heart disease. This combination has been previously implicated in causing severe hypotension.⁸ Recently, Morris et al.⁴ reported that patients, receiving either nifedipine or diltiazem and undergoing left ventriculography with an ionic contrast agent, experienced more severe hypotensive response than patients not receiving these drugs.

Although this finding has not been proved, it is highly likely that this unusual reaction was partially due to this

treatment. To the best of our knowledge, this unusual reaction has not been described, and studies of this nature are essential for physicians caring for patients who develop anaphylaxis for various reasons.

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REFERENCES

1. Awai LW, Mekori YA. Insect-sting anaphylaxis and beta-adrenergic blockade: a relative contraindication. *Ann Allergy* 1984;53:48-9.
2. Hannaway PJ, Hopper GDK. Severe anaphylaxis and drug-induced beta-blockade. *N Engl J Med* 1983;308:1536.
3. Ingall M, Goldman G, Page LB. Beta-blockade in stinging-insect anaphylaxis. *JAMA* 1984;251:1432.
4. Morris DL, Wisneski JA, Gertz EW, Wexman M, Axelrod R, Lanberg J. Potentiation by nifedipine and diltiazem of the hypotensive response after contrast angiography. *J Am Coll Cardiol* 1985;6:785-91.
5. Smith PS, Sobotka AK, Bleccher ER, et al. Physiological manifestation of human anaphylaxis. *J Clin Invest* 1980;66:1072-80.
6. Stark BJ, Sullivan RJ. Biphasic and protracted anaphylaxis. *J ALLERGY CLIN IMMUNOL* 1986;78:76-83.
7. Nicklas RA, Bochner BS, Scanlon RT. Delayed insect sting anaphylaxis [Abstract]. *J ALLERGY CLIN IMMUNOL* 1988;81:237.
8. Opie LH, White DA. Adverse interaction between nifedipine and beta-blockade. *Br Med J* 1980;281:1462.