First-aid treatment of anaphylaxis to food: Focus on epinephrine

Avoiding food triggers for anaphylactic reactions (severe acute systemic allergic reactions) is easier said than done. Most episodes of anaphylaxis to food occur unexpectedly in the community in the absence of a health care professional. All individuals at risk should therefore have an emergency action plan in place. The cornerstone of first-aid treatment of anaphylaxis is epinephrine injected intramuscularly in the vastus lateralis muscle (lateral aspect of the thigh). In this review, we focus on epinephrine. We examine a therapeutic dilemma: the issue of epinephrine dose selection in an individual for whom no optimal fixed-dose auto-injector formulation exists, and a therapeutic controversy: the issue of epinephrine injection versus an oral H1-antihistamine in anaphylaxis episodes that appear to be mild. The pharmaceutical industry could address the first of these issues by providing a wider range of epinephrine fixed doses in easy-to-use auto-injectors, or by providing adjustable epinephrine doses in auto-injectors. The second issue could be addressed in part by development of alternative routes of epinephrine administration for the first-aid, out-of-hospital treatment of anaphylaxis. (J Allergy Clin Immunol 2004;113:837-44.)

Key words: Acute allergic reaction, adrenaline, adults, anaphylaxis, auto-injector, children, epinephrine, EpiPen, EpiPen Jr, food allergy, peanut allergy, H1-antihistamines, activated charcoal

Historically, anaphylaxis was triggered mainly by biological substances such as antitoxin or by medications, and usually occurred in a health care setting. Currently, food is the most common trigger of anaphylaxis, and most episodes occur unexpectedly in the community in the absence of a trained health care professional. Anaphylaxis is defined as an acute systemic allergic reaction that varies in severity from mild to life-threatening or fatal and may be rapidly progressive. Individuals who have had such a reaction (or for children, their caregivers) should be equipped with an anaphylaxis emergency action plan and with injectable epinephrine for first-aid treatment, defined as treatment before or during transport to an emergency department.

Here, we review the current scientific evidence on which the first-aid treatment of anaphylaxis is based. We focus chiefly on epinephrine and address 2 difficult issues in first-aid treatment with this life-saving medication: the dilemma of epinephrine dose selection in individuals for whom no optimal fixed-dose auto-injector formulation exists, and the controversial issue of epinephrine injection versus an oral H1-antihistamine in anaphylaxis episodes that appear to be mild.

EPINEPHRINE IN THE FIRST-AID TREATMENT OF ANAPHYLAXIS

Pharmacologic activity

Epinephrine is a direct-acting sympathomimetic α-adrenergic and β-adrenergic agonist with cyclic adenosine monophosphate-mediated, complex, bidirectional pharmacologic effects on many target organs (Fig 1). Achieving high plasma and tissue epinephrine concentrations rapidly appears to be critical for reversal of hypotension and possibly for survival. Epinephrine has a narrow toxic-therapeutic index (risk-to-benefit ratio). Administered to individuals of any age, in therapeutic doses, by any route, including inhalation, it may cause pharmacologic adverse effects such as anxiety, fear, restlessness, headache, dizziness, palpitations, pallor, and tremor. Rarely, and especially after overdose, it may lead to ventricular arrhythmias, angina, myocardial infarction, pulmonary edema, sudden sharp increase in blood pressure, and intracranial hemorrhage. The risk of epinephrine adverse effects may be increased in individuals with some pre-existing cardiovascular, central nervous system, or thyroid diseases; in persons using monoamine oxidase inhibitors, which block epinephrine metabolism; or in those using tricyclic antidepressants or cocaine, in whom epinephrine duration of action is prolonged. There is, however, no absolute contraindication to epinephrine use in anaphylaxis.

Evidence base for epinephrine use in anaphylaxis

Recommendations for epinephrine dosing in the first-aid, out-of-hospital treatment of anaphylaxis are based on anecdotal experience and vary with regard to maximum initial dose (0.2 mg to 0.5 mg in adults; 0.01 mg/kg to a maximum of 0.3 mg in children), route of injection (subcutaneous vs intramuscular), and interval between doses (5-30 minutes). Prospective, randomized, double-blind, placebo-controlled clinical trials of epinephrine in individuals actually experiencing anaphylaxis are...
unethical because prompt treatment with epinephrine is deemed critically important for survival. Also, such studies would be difficult to conduct because anaphylaxis episodes usually occur without warning in a nonmedical setting and differ in severity among individuals and from one episode to another in the same individual; consequently, baseline measurements and frequent timed measurements would be hard to obtain.

Despite the absence of clinical trials, evidence from clinical pharmacology studies, epidemiologic studies, and other investigations supports the use of epinephrine in anaphylaxis. Based on the observation that subcutaneous administration of epinephrine causes skin blanching at the injection site as a result of the powerful $\alpha_1$-vasoconstrictor effect of the drug, it was hypothesized that retention of epinephrine at the site of subcutaneous injection might lead to a delay in absorption into the systemic circulation. This hypothesis was initially tested in a randomized, blind study in children at risk for anaphylaxis in whom the time to peak plasma epinephrine concentration ($t_{\text{max}}$), accompanied by prompt physiologic effects, was $8 \pm 2$ minutes after intramuscular injection, significantly shorter than the $t_{\text{max}}$ of $34 \pm 14$ minutes (range, 5 to 120) after injection of epinephrine 0.01 mg/kg subcutaneously in the deltoid region. Based on data from Simons et al.

Limitations of epinephrine first-aid treatment in anaphylaxis

Epinephrine is usually, but not always, effective in the first-aid treatment of anaphylaxis. Many potential reasons for lack of response can be identified. These include rapid progression of the episode, and failure
to give epinephrine in a timely manner or to administer it correctly: in 1 survey, only 30% of individuals at risk for anaphylaxis to food, or their caregivers, could demonstrate how to use an auto-injector. In addition, epinephrine may not be given in an optimal dose or administered by an optimal route. As noted previously, subcutaneous injection may lead to a delay in epinephrine absorption; inhalation of a few puffs of epinephrine from a pressurized metered-dose inhaler will be inadequate for treatment of nonrespiratory symptoms, and supplying individuals with an epinephrine ampule, syringe, and needle may lead to delayed dose, overdose, underdose, or no dose at all. In 1 study, parents without health care training who were instructed on how to draw up an infant dose of epinephrine from an ampule took significantly longer to get the dose into the syringe than physician or nurse controls did (Fig 3); moreover, the epinephrine content of the parents’ doses ranged 40-fold, and their speed and accuracy did not correlate. Out-of-date EpiPen and EpiPen Jr auto-injectors (Dey, Napa, Calif) may not provide an optimal dose of epinephrine, even if their contents appear to be clear and within

### TABLE I. Potential reasons for lack of response to epinephrine in anaphylaxis

<table>
<thead>
<tr>
<th>Potential reason</th>
<th>Relevant studies</th>
<th>Comments</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient/caregiver/physician</td>
<td>Case reports, autopsy reports</td>
<td>In 10% of anaphylaxis episodes, epinephrine does not work even if given promptly</td>
<td>25-28</td>
</tr>
<tr>
<td>Rapid anaphylaxis progression</td>
<td>Case reports, autopsy reports</td>
<td>Cause-and-effect hard to prove</td>
<td>25-28</td>
</tr>
<tr>
<td>Epinephrine given too late</td>
<td>Cross-sectional surveys, demonstrations</td>
<td>Frequently reported</td>
<td>7-9,16</td>
</tr>
<tr>
<td>Individuals do not know how to use epinephrine auto-injectors</td>
<td>Few dose-response studies</td>
<td>Optimal dose unknown; based on tradition, 0.3 mg is used for adults in many countries, 0.5 mg in some countries</td>
<td>7-9,13</td>
</tr>
<tr>
<td>Dose too low</td>
<td>Intramuscular vs subcutaneous</td>
<td>Intramuscular, thigh preferred to subcutaneous, arm</td>
<td>14,15</td>
</tr>
<tr>
<td>Lack of availability of fixed doses 0.05, 0.1, 0.2, 0.25 mg in auto-injectors</td>
<td>Ampule and syringe</td>
<td>Non-medical personnel lack speed and accuracy</td>
<td>18</td>
</tr>
<tr>
<td>Route/site not optimal</td>
<td>Pressurized metered-dose inhaler</td>
<td>For systemic effects, adults need 20-30 puffs (children 10-20); possible phase-out by 2006</td>
<td>17</td>
</tr>
<tr>
<td>Past expiration date</td>
<td>Bioavailability measured in animal models; content measured in vitro</td>
<td>Epinephrine content inversely related to number of months past expiration date</td>
<td>19</td>
</tr>
<tr>
<td>Other</td>
<td>Autopsy reports</td>
<td>If the individual is standing, venous return is decreased, the ventricles are empty, and death may occur despite timely epinephrine-induced reversal of vasodilation and shock</td>
<td>31</td>
</tr>
<tr>
<td>Individual not supine</td>
<td>Case reports</td>
<td>There is more information about lack of effect in asthma than in anaphylaxis</td>
<td>9</td>
</tr>
<tr>
<td>Individual taking medications (β-blocker or α-blocker, angiotensin-converting enzyme inhibitor) that prevent optimal epinephrine effect</td>
<td>Case reports</td>
<td>Additional studies needed, because sulfite-sensitive asthmatics tolerate epinephrine</td>
<td>20</td>
</tr>
<tr>
<td>Adverse effects of sodium metabisulfite (antioxidant in epinephrine)</td>
<td>Case reports</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*In obese individuals, intramuscular injections of epinephrine may inadvertently end up being subcutaneous injections unless a needle at least 2.5 cm (1 in) is used to penetrate the fat pad over the vastus lateralis muscle.*

*It is difficult to inhale the large number of epinephrine puffs required because of vasoconstriction of the oropharyngeal mucosa, causing tingling and burning sensations.*

*Compendial limits for epinephrine content of formulations are 90% to 115% of labeled strength (United States Pharmacopeia), but in some countries, the stated content of epinephrine in auto-injectors may range from 0.23 mg to 0.37 mg. Epinephrine should be stored at room temperature (15°C to 30°C) to prevent oxidation and inactivation. In an EpiPen auto-injector, it is supplied in light-resistant packaging, and each 0.3-mL dose contains 0.3 mg epinephrine, 1.8 mg sodium chloride, 0.5 mg sodium metabisulfite, and hydrochloric acid to adjust the pH from 2.2 to 5.0. An EpiPen Jr contains epinephrine 0.15 mg and the same nonmedicinal ingredients in the same amounts as in the EpiPen.
out apparent pink or brown discoloration from oxidation of epinephrine to adrenochrome or melanin. Significant reduction in epinephrine bioavailability from out-of-date auto-injectors has been documented, and their measured epinephrine content correlates inversely with the length of time past the expiration date ($r = 0.63$).\textsuperscript{19}

Lack of an optimal range of epinephrine doses in easy-to-use auto-injectors for first-aid use, and lack of ability to adjust the epinephrine dose in auto-injectors, may also contribute to underdosing with epinephrine and to lack of response in some individuals.

**Epinephrine dose dilemma: 2 fixed-dose auto-injectors do not suffice**

An important factor that needs to be addressed by regulatory agencies and ultimately by the pharmaceutical industry is the availability of only 2 fixed doses of epinephrine (0.15 mg and 0.3 mg) in easy-to-use auto-injectors.
TABLE II. Clinical dilemma: Selecting an appropriate epinephrine fixed-dose auto-injector for infants and children

<table>
<thead>
<tr>
<th>Body weight</th>
<th>≤5 kg</th>
<th>10 kg</th>
<th>15 kg</th>
<th>20 kg</th>
<th>25 kg</th>
<th>≥30 kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at which this weight is 50th percentile</td>
<td>2 mo</td>
<td>14 mo</td>
<td>3 y</td>
<td>6 y</td>
<td>9 y</td>
<td>12 y</td>
</tr>
<tr>
<td>Optimal epinephrine dose</td>
<td>0.05 mg</td>
<td>0.1 mg</td>
<td>0.15 mg</td>
<td>0.2 mg</td>
<td>0.25 mg</td>
<td>0.3 mg</td>
</tr>
<tr>
<td>Is optimal dose available in an auto-injector?</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Situation acceptable?</td>
<td>No</td>
<td>No</td>
<td>Yes, use EpiPen Jr</td>
<td>No</td>
<td>Yes, use EpiPen</td>
<td></td>
</tr>
</tbody>
</table>

*The difficulty of giving a precise epinephrine dose of 0.01 mg/kg to a child weighing <15 kg or weighing between 15 and 30 kg, either by using the EpiPen Jr (0.15 mg) or the EpiPen (0.3 mg), is outlined, including the magnitude of overdose or underdose that must be accepted if 1 of the currently available fixed-dose auto-injectors is recommended.

There is a possibility that underdosing (<0.01 mg/kg) may occur when some larger children, adolescents, and adults use the EpiPen (0.3 mg).

The main examples used are EpiPen Jr and EpiPen, which is also distributed under other trade names; however, similar limitations apply to other auto-injectors. In addition to the issue highlighted in Table II, manufacturer/distributor recommendations for weight-appropriate and age-appropriate use of auto-injectors containing 0.15 mg and 0.3 mg epinephrine differ in different countries—eg, in some countries, an auto-injector containing 0.15 mg is recommended only for individuals weighing ≤15 kg, and in other countries, an auto-injector containing 0.15 mg epinephrine is recommended only for individuals weighing 15 to 30 kg.12,21

In a geographically defined population, using an administrative claims database, both EpiPen Jr (0.15 mg) and EpiPen (0.3 mg) auto-injectors were found to be dispensed (for anaphylaxis from all triggers) over nearly the entire pediatric age range: EpiPen Jr from 2 months to 16 years, 10 months; and EpiPen from 1 year, 8 months, to 16 years, 11 months.22 The age of transition from EpiPen Jr to EpiPen ranged from 1 year, 10 months, to 16 years, 11 months, with a mean of 6 years, 6 months, at which time fewer than 3% of children weigh 30 kg and would receive an optimal dose of 0.01 mg/kg from an EpiPen.22

In a randomized, double-blind, parallel group study, children age 5 to 8 years and weighing 16 to 30 kg self-injected epinephrine by using either an EpiPen Jr or an EpiPen with the aid of a physician.13 Children who received a dose of 0.01 to 0.014 mg/kg from an EpiPen had a significantly higher mean systolic blood pressure 30 minutes after injection; however, in every child, this was accompanied by pallor, tremor, anxiety, and palpitations. Some children also developed headache and nausea, and 1 child had an increase in the QTc interval. In contrast, children who received 0.008 to 0.009 mg/kg from an EpiPen Jr did not achieve a significant increase in blood pressure and had fewer, milder, and more transient adverse effects limited to pallor, tremor, or anxiety.13

Until additional fixed doses of epinephrine are available in auto-injector formulations, physicians should carefully weigh the benefits versus the risks of the two available doses, 0.15 mg versus 0.3 mg, for each child (Table III). Lack of appropriate dose options should not deter them from recommending epinephrine for the first-aid, out-of-hospital treatment of anaphylaxis.

Some adolescents and adults may not be optimally treated with the maximum epinephrine dose of 0.3 mg available in an auto-injector. In addition, the 14.29 mm length needle on currently available auto-injectors may be

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<td>0.3 mg</td>
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<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Situation acceptable?</td>
<td>No</td>
<td>No</td>
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Epinephrine is widely dispensed, but not widely used in anaphylaxis

Epinephrine is widely dispensed in the community; however, in retrospective studies of individuals dying from anaphylaxis, it has been consistently reported to be underused, and failure to inject it at all, delayed use, inappropriate dose, or inappropriate route of administration have been identified as contributing factors to death. In 1 autopsy series, although epinephrine was given in 62% of fatal anaphylactic reactions triggered by a variety of agents, it was given before respiratory arrest in only 14% of these reactions. In a study of 32 individuals dying from peanut or tree nut allergy, 12 did not receive epinephrine at all, 10 received it too late, 4 died despite receiving it in a timely manner, and for 6, no information was available. In studies of individuals surviving anaphylaxis episodes, it has been reported that only 30% to 40% of subjects who required epinephrine actually received it.

Alternative routes of epinephrine administration in anaphylaxis

Many individuals with anaphylaxis and many caregivers of children with anaphylaxis are reluctant to inject epinephrine because of anxiety about using a needle. Administration of epinephrine through chlorofluorocarbon-containing pressurized metered-dose inhalers, in countries where these are still approved for use, contributes to relief of respiratory symptoms but is impractical for the treatment of other systemic effects, which requires 20 to 30 inhalations over a period of 4 minutes in an adult. Oral epinephrine administration is ineffective because of metabolism by catechol-O-methyltransferase in the wall of the gastrointestinal tract and by monoamine oxidase in the wall of the gastrointestinal tract and in the liver. Based on the precedent of using sublingual nitroglycerin for treatment of angina, the feasibility of sublingual epinephrine administration for the first-aid treatment of anaphylaxis is being explored in clinical pharmacology studies. Other approaches to the first-aid treatment of anaphylaxis

Supportive treatment

Individuals with anaphylaxis, especially those who feel faint or dizzy because of impending shock, should be kept in the supine position unless they are vomiting or experiencing severe respiratory distress. During extreme vasodilation, blood return to the vena cava, right and left chambers of the heart, and coronary arteries is more likely to be maintained if they are supine than if they are seated or standing.

The epinephrine injection versus oral H1-antihistamine controversy

Histamine is an important mediator in anaphylaxis. H1-antihistamines are commonly used to relieve cutaneous signs and symptoms such as itching, flushing, and urticaria, but play little, if any, role in relief of bronchospasm or gastrointestinal symptoms; fail to relieve upper airway edema or hypotension; and, in usual doses, do not reduce the explosive release of histamine and other mediators of inflammation from mast cells and basophils. In clinical pharmacology studies conducted in fasting individuals, onset of activity of orally ingested H1-antihistamines does not occur until 40 to 60 minutes after ingestion (Table IV), and maximal activity is not achieved for at least 4 hours. In anaphylaxis, there are no prospective, randomized, double-blind, placebo-controlled clinical trials of oral H1-antihistamines or the algorithms for their use. In advance, there is no way to identify individuals whose anaphylaxis manifestations will be limited to the skin and for whom an H1-antihistamine will suffice. The course of an anaphylaxis episode and the window of opportunity for successful epinephrine treatment cannot be predicted with certainty and may differ from one person to another, and from one episode to another in the same person.

TABLE IV. Oral H1-antihistamines have a slow onset of action

<table>
<thead>
<tr>
<th>First-generation</th>
<th>Healthy, fasting young adults, single dose*</th>
<th>Healthy, fasting children, single dose*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpheniramine</td>
<td>2.8 ± 0.8 Onset of activity (h postdose)†</td>
<td>2.5 ± 1.5 Onset of activity (h postdose)†</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>1.7 ± 1.0 Onset of activity (h postdose)†</td>
<td>1.3 ± 0.5 Onset of activity (h postdose)†</td>
</tr>
<tr>
<td>Hydroxyzine</td>
<td>2.1 ± 0.4 Onset of activity (h postdose)†</td>
<td>2.0 ± 0.9 Onset of activity (h postdose)†</td>
</tr>
<tr>
<td>Second-generation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cetirizine</td>
<td>1.0 ± 0.5 Onset of activity (h postdose)†</td>
<td>1.1 ± 0.8 Onset of activity (h postdose)†</td>
</tr>
<tr>
<td>Desloratadine</td>
<td>1-3 Onset of activity (h postdose)†</td>
<td>NA Onset of activity (h postdose)†</td>
</tr>
<tr>
<td>Fexofenadine</td>
<td>2.6 Onset of activity (h postdose)†</td>
<td>2.4 ± 0.2 Onset of activity (h postdose)†</td>
</tr>
<tr>
<td>Loratadine</td>
<td>1.2 ± 0.3 Onset of activity (h postdose)†</td>
<td>1 Onset of activity (h postdose)†</td>
</tr>
</tbody>
</table>

*H1-antihistamine tablets used in most studies; liquid formulation of cetirizine, chlorpheniramine, diphenhydramine, hydroxyzine, and loratadine was given to fasting children.
†The time stated is the first interval after the dose at which symptoms and/or histamine-induced wheal or flare were significantly decreased compared with baseline in clinical pharmacology studies.
NA, No published information.
different cohorts, adverse reactions to peanut and tree nut became more severe with time in 1/3 or more of individuals\textsuperscript{29,33,34} (Fig 5).

The detailed algorithms developed to help physicians decide whether to give epinephrine or an H\textsubscript{1}-antihistamine in anaphylaxis\textsuperscript{6} are useful in health care settings; however, in the first-aid treatment of anaphylaxis in the community, placing the burden of decision making ("if you observe this, do that") on individuals without medical training or resuscitation team backup may not be appropriate. Judgment may be clouded by central nervous system symptoms, fear, panic, or denial ("This can’t possibly be happening again!"). Especially if very young or very ill, individuals may have difficulty verbalizing or describing their symptoms. Moreover, it may be difficult to assess clinical signs accurately in crowded, noisy, poorly lighted public places such as restaurants or airplanes where anaphylaxis to food occurs. In out-of-hospital settings, the inherent danger in the "try an antihistamine" approach or the "wait and see whether epinephrine is needed" approach relates to the observation that the median time to respiratory or cardiac arrest in individuals with anaphylaxis from food is 30 minutes.\textsuperscript{25}

**Is there a role for activated charcoal in the first-aid treatment of anaphylaxis?**

Activated charcoal, which is given in emergency departments (usually by nasogastric tube) for gastrointestinal decontamination after poisoning, has been suggested as a useful adjunct to the treatment of individuals with anaphylaxis to peanut, based on an in vitro study in which it rapidly adsorbed peanut protein in a dose-dependent manner at pH 3.5 or 7.4.\textsuperscript{35} In vitro studies of adsorption of other foods and prospective studies of food adsorption in humans have not yet been conducted. Administration of activated charcoal may present a practical problem in the first-aid treatment of food-induced anaphylaxis. Time is of the essence, and it would need to be given immediately after ingestion of the offending food; however, it may be difficult to administer by mouth in an adequate dose, because it clumps during storage, is messy, and is often vomited. Even for poisoning, it is not recommended for routine administration in nonmedical settings because of limited published experience with it in such settings.\textsuperscript{36} In the first-aid treatment of anaphylaxis, an additional concern is that administration of activated charcoal may delay epinephrine injection.

**After first-aid treatment with epinephrine**

After first-aid treatment with epinephrine injection, individuals should be transported to the nearest hospital emergency department for monitoring over a period of 4 to 6 hours. Additional doses of epinephrine, as well as oxygen, intravenous fluids, glucocorticoids, H\textsubscript{1}-antihistamines, H\textsubscript{2}-antihistamines, vasopressors, and other interventions, may be required.\textsuperscript{6}

All individuals who have had anaphylaxis from food are at risk of subsequent reactions and therefore require follow-up. Ideally, this should include evaluation or re-evaluation by a board-certified allergist regarding the food trigger for the episode, recommendations for appropriate food avoidance measures and Medic Alert (Medic Alert Foundation International, Turlock, Calif) or other identification, coaching in the appropriate use of an epinephrine auto-injector, and development or review of an anaphylaxis emergency action plan.\textsuperscript{26-28} The essentials of the emergency action plan include photograph identification of the person at risk and a list of their specific food triggers. If there is a concurrent diagnosis of asthma, which increases the risk of death from anaphylaxis,\textsuperscript{26-28} this should be stated. The emergency action plan should also include a short list of anaphylaxis symptoms and

![FIG 5. Mild anaphylactic reactions do not always remain mild. Individuals in the Food Allergy and Anaphylaxis Network’s voluntary registry, or caregivers of children in the registry, answered a structured questionnaire about allergic reactions to peanut and tree nut. In comparison with initial reactions, a higher proportion of subsequent reactions were severe and were treated with epinephrine (first reaction vs third reaction, $P < .001$; the asterisks indicate a reaction more severe than the previous reaction).\textsuperscript{33}](image-url)
signs, pictorial instructions regarding prompt first-aid use of an epinephrine auto-injector, information about contacting the rescue squad and the family after epinephrine injection, and a reminder to transport the individual to an emergency department after first-aid treatment.

In the future, preventive treatment—for example, by injecting anti-IgE antibody at regular intervals—may be emergency department after first-aid treatment.

In summary, fatalities from anaphylaxis to food are, fortunately, uncommon; however, 90% of these deaths are preventable, and all who are involved in the care of individuals with food-triggered anaphylaxis share the responsibility for their prevention and first-aid treatment in the community.

REFERENCES