



Complete Summary

GUIDELINE TITLE

The diagnosis and management of anaphylaxis: an updated practice parameter.

BIBLIOGRAPHIC SOURCE(S)

The diagnosis and management of anaphylaxis: an updated practice parameter. J Allergy Clin Immunol 2005 Mar;115(3 Suppl):S483-523. [232 references] [PubMed](#)

GUIDELINE STATUS

This is the current release of the guideline.

This guideline updates a previous version: Joint Council of Allergy, Asthma and Immunology. The diagnosis and management of anaphylaxis. J Allergy Clin Immunol 1998 Jun;101(6 Pt 2):S465-S528.

COMPLETE SUMMARY CONTENT

SCOPE
METHODOLOGY - including Rating Scheme and Cost Analysis
RECOMMENDATIONS
EVIDENCE SUPPORTING THE RECOMMENDATIONS
BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS
CONTRAINDICATIONS
QUALIFYING STATEMENTS
IMPLEMENTATION OF THE GUIDELINE
INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES
IDENTIFYING INFORMATION AND AVAILABILITY
DISCLAIMER

SCOPE

DISEASE/CONDITION(S)

Anaphylaxis and anaphylactoid reactions, including:

- Anaphylaxis to foods
- Latex-induced anaphylaxis
- Anaphylaxis during general anesthesia, the intraoperative period, and the postoperative period
- Seminal fluid-induced anaphylaxis
- Exercise-induced anaphylaxis
- Idiopathic anaphylaxis

- Anaphylaxis and allergen immunotherapy vaccine
- Anaphylaxis to drugs

GUIDELINE CATEGORY

Diagnosis
Evaluation
Management
Prevention
Risk Assessment
Treatment

CLINICAL SPECIALTY

Allergy and Immunology
Emergency Medicine
Family Practice
Internal Medicine
Pediatrics

INTENDED USERS

Physicians

GUIDELINE OBJECTIVE(S)

To improve the care of patients by providing the practicing physician with an evidence-based approach to the diagnosis and management of anaphylactic reactions

TARGET POPULATION

Patients at risk for anaphylactic or anaphylactoid reactions

INTERVENTIONS AND PRACTICES CONSIDERED

Evaluation/Diagnosis/Risk Assessment

1. Patient history
2. Signs and symptoms
3. Diagnostic tests (e.g., skin prick tests, food challenges, in vitro testing)
4. Laboratory tests (e.g., serum tryptase)
5. Intensive evaluation, including a meticulous history (idiopathic anaphylaxis)

Prevention/Management

1. Patient education (early treatment, self-administration of epinephrine, and use of medical condition identification bracelet)
2. Avoidance measures
3. Prophylaxis with glucocorticosteroids and antihistamines
4. Allergen immunotherapy

5. Desensitization to medications

Treatment

1. Epinephrine and oxygen
2. Volume replacement with colloid or crystalloids
3. Immunotherapy
4. Cardiopulmonary resuscitation and advanced cardiac life support for cardiopulmonary arrest during anaphylaxis
5. Transport to emergency or intensive care facility
6. Individualized follow-up
7. Consultation with allergist-immunologist

MAJOR OUTCOMES CONSIDERED

- Mortality due to anaphylaxis
- Recurrence of anaphylaxis or anaphylactoid reactions
- Utility of diagnostic interventions

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

A comprehensive search of the medical literature was conducted with various search engines, including PubMed, using appropriate search terms.

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Ia Evidence from meta-analysis of randomized controlled trials

Ib Evidence from at least one randomized controlled trial

IIa Evidence from at least one controlled study without randomization

IIb Evidence from at least one other type of quasiexperimental study

III Evidence from nonexperimental descriptive studies, such as comparative studies

IV Evidence from expert committee reports or opinions or clinical experience of respected authorities or both

METHODS USED TO ANALYZE THE EVIDENCE

Systematic Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Published clinical studies were rated by category of evidence and used to establish the strength of the clinical recommendations.

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

Not stated

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

- A. Directly based on category I evidence
- B. Directly based on category II evidence or extrapolated recommendation from category I evidence
- C. Directly based on category III evidence or extrapolated recommendation from category I or II evidence
- D. Directly based on category IV evidence or extrapolated recommendation from category I, II, or III evidence

NR Not rated

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

A workgroup chaired by Phillip Lieberman, MD, prepared the initial draft. The Joint Task Force then reworked the initial draft into a working draft of the document. The working draft of this updated parameter was reviewed by a large number of

experts on anaphylaxis selected by the sponsoring organizations. This document represents an evidence-based and broadly accepted consensus viewpoint on the diagnosis and management of anaphylaxis.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

This practice parameter includes algorithms for the initial evaluation and management of a patient with a history of an episode of anaphylaxis and for the treatment of acute anaphylaxis accompanied by annotations (numbered to correspond to the algorithms). Guideline recommendations are presented in the form of summary statements. After each statement is a letter that indicates the strength of the recommendation. Grades of recommendations are defined at the end of the Major Recommendations field.

Algorithm for Initial Evaluation and Management of a Patient with a History of Anaphylaxis

Annotation 1: Is the history consistent with a previous episode of anaphylaxis?

All individuals who have had a known or suspected anaphylactic episode require a careful and complete review of their clinical history. This history might elicit manifestations, such as urticaria, angioedema, flushing, pruritus, upper airway obstruction, gastrointestinal symptoms, syncope, hypotension, lower airway obstruction, and/or dizziness.

Of primary importance is the nature of the symptoms characterizing the event. Essential questions to be asked are as follows:

1. Were there cutaneous manifestations, specifically pruritus, flush, urticaria, and angioedema?
2. Was there any sign of airway obstruction involving either the upper airway or the lower airway?
3. Were there gastrointestinal symptoms (i.e., nausea, vomiting, or diarrhea)?
4. Were syncope or presyncopal symptoms present?

At this point, it should be noted that the absence of cutaneous symptoms puts the diagnosis in question because the majority of anaphylaxis includes cutaneous symptoms, but their absence would not necessarily rule out an anaphylactic or anaphylactoid event.

The history should concentrate on agents encountered before the reaction. Whenever appropriate, the information should be obtained from not only the patient but also family members or other witnesses. The complete sequence of events must be reviewed, with special attention paid to the cardiorespiratory symptoms. Medical records, including medication records, can often be useful in evaluating the history, physical findings, and treatment of the clinical event. In addition, the results of any previous laboratory studies (e.g., serum tryptase

levels) might be helpful in making the diagnosis of anaphylaxis or distinguishing it from other entities.

Annotation 1A: Consider consultation with an allergist-immunologist

Patients with anaphylaxis might be first seen with serious and life-threatening symptoms. Evaluation and diagnosis, as well as long-term management, can be complex. The allergist-immunologist has the training and expertise to obtain a detailed allergy history, coordinate laboratory and allergy testing, evaluate the benefits and risks of therapeutic options, and counsel the patient on avoidance measures. For these reasons, patients with a history of anaphylaxis should be considered for referral to an allergy-immunology specialist.

Annotation 2: Pursue other diagnoses or make appropriate referral

Other conditions that should be considered in the differential diagnosis include the following: (1) vasodepressor (vasovagal-neurocardiogenic) syncope; (2) syndromes that can be associated with flushing (e.g., metastatic carcinoid); (3) postprandial syndromes (e.g., scombroid poisoning); (4) systemic mastocytosis; (5) psychiatric disorders that can mimic anaphylaxis, such as panic attacks or vocal cord dysfunction syndrome; (6) angioedema (e.g., hereditary angioedema); (7) other causes of shock (e.g., cardiogenic); and (8) other cardiovascular or respiratory events.

Annotation 3: Is cause readily identified by history?

The history is the most important tool to establish the cause of anaphylaxis and takes precedence over diagnostic tests. A detailed history of all ingestants (both foods and drugs) several hours before the episode should be obtained. In addition, the labels for all packaged foods ingested by the patient in this period of time should be reviewed because a substance added to the food (e.g., carmine) could be responsible. A history of any preceding bite or sting should be obtained. The patient's activities (e.g., exercise, sexual activity, or both) preceding the event should be reviewed. Patient diaries might be a useful adjunct in confirming and identifying the cause of anaphylaxis.

Annotation 4: Consider idiopathic anaphylaxis

Idiopathic anaphylaxis is a diagnosis of exclusion that should be made only after other causes of anaphylaxis and other differential diagnoses have been considered.

Annotation 5: Are further diagnostic tests indicated: immediate hypersensitivity or in vitro tests, challenge tests?

Immediate hypersensitivity tests or in vitro specific immunoglobulin E (IgE) tests and/or challenge tests might be appropriate to help define the cause of the anaphylactic episode. However, the history might be so specific that none of the above tests are necessary.

Annotation 6: Diagnosis established on basis of history, risk of testing, limitation of tests, patient refuses test, other management options available, management

There might be circumstances in which allergy skin tests, in vitro specific IgE tests, and/or challenge tests might not be warranted. In general, this might apply when the clinician (with the consent of the patient) decides to proceed with management on the basis of the history and physical examination.

For example, the clinical history of anaphylaxis to a specific agent might be so strong that testing is unnecessary (or dangerous). Conversely, the medical history of anaphylaxis might be sufficiently mild or weak that management can proceed in the absence of testing. If avoidance can be easily and safely accomplished, testing might not be necessary.

Furthermore, testing or challenge with reagents to a suspected allergen might not be available, or the accuracy of the test might be in question. In addition, for patients with a history of anaphylaxis, challenge tests (and, to a lesser extent, skin tests) might be hazardous.

Annotation 7: Testing identifies specific cause of anaphylaxis

Skin tests or in vitro tests that determine the presence of specific IgE antibodies can identify specific causes of anaphylaxis. Causes of anaphylaxis that can be defined in this way include foods, medications (e.g., penicillin and insulin), and stinging insects. For the majority of medications, standardized testing either by in vivo or in vitro means is not available. Such tests are only valid when the reaction is due to a true anaphylactic event (IgE-mediated reaction) and not as a result of an anaphylactoid (non-IgE-mediated) reaction.

In general, skin testing is more sensitive than in vitro testing and is the diagnostic procedure of choice for evaluation of most potential causes of anaphylaxis (e.g., penicillin, insect stings, and foods). It is essential that the correct technique for skin testing be used to obtain meaningful data regarding causative agents of anaphylaxis. When possible, standardized extracts should be used (occasionally fresh food extracts will be superior to available standardized extracts). If the skin testing extract has not been standardized (e.g., latex, protamine, or antibiotics other than penicillin), the predictive value is uncertain. If skin testing is performed, it should be done under the supervision of a physician who is experienced in the procedure in a setting with appropriate rescue equipment and medication.

The accuracy of in vitro testing depends on the reliability of the in vitro method, the ability to interpret the results, and the availability of reliable testing material. The clinical significance of skin test or in vitro test results depends on the ability to correlate such results with the patient's history.

If tests for specific IgE antibodies (i.e., skin tests, in vitro tests, or both) do not provide conclusive evidence of the cause of anaphylaxis, challenge with the suspected agent can be considered. Challenge procedures might also be appropriate in patients with anaphylactoid reactions (e.g., reactions to aspirin or other nonsteroidal anti-inflammatory drugs). Challenges with suspected agents

must be done carefully by individuals knowledgeable in the challenge procedure and with expertise in managing reactions to the challenge agent if they should occur.

Annotation 8: Reconsider clinical diagnosis, reconsider idiopathic anaphylaxis, consider other triggers, consider further testing, management

At this stage in the patient's evaluation, it is particularly important to consider other trigger factors and diagnoses. The medical history and laboratory test results should be reviewed. Further testing for specific IgE antibodies should be considered. Laboratory studies that might be helpful include serum tryptase measurement, as well as urinary 5-hydroxyindoleacetic acid, methylhistamine, and catecholamine measurement. Idiopathic anaphylaxis is a diagnosis of exclusion (see "Idiopathic anaphylaxis").

Management of anaphylaxis episodes should follow annotation 10 (see algorithm).

Annotation 9: Diagnosis made of specific cause of anaphylaxis

The diagnosis of a specific cause of anaphylaxis might be supported by the results of skin tests, in vitro IgE tests, and/or challenge tests (particularly double-blind, placebo-controlled challenge tests).

Annotation 10: Management of anaphylaxis

When anaphylaxis has occurred because of exposure to a specific agent (e.g., food, medication, or insect sting), patients should be educated about agents or exposures that would place them at risk for future reactions and be counseled on avoidance measures that might be used to reduce risk for such exposures. Patients who have had anaphylactic reactions to food should be instructed on how to read food ingredient labels to identify foods that they should avoid. Patients with anaphylaxis to medications should be informed about all cross-reacting medications that should be avoided. Should there be a future essential indication for use of incriminated medications, it might be helpful to educate patients about applicable management options (e.g., medication pretreatment and use of low-osmolarity agents in patients with a history of reactions to radiographic contrast media or desensitization for drugs, such as antibiotics). Patients who have had anaphylactic reactions to insect stings should be advised about avoidance measures to reduce the risk of insect stings and are candidates for insect venom immunotherapy (see "Stinging insect hypersensitivity: a practice parameter update"). Patients who have had anaphylaxis should carry self-injectable epinephrine for use if anaphylaxis develops. There might be exceptions to this (e.g., anaphylaxis to penicillin). Patients should also carry identification indicating that they are prone to anaphylaxis and indicating the responsible agent. Patients taking beta-blockers are at increased risk during anaphylaxis.

Algorithm for the Treatment of Acute Anaphylaxis

Annotation 1. Anaphylaxis preparedness

It is important to stress that management recommendations are subject to physician discretion and that variations in sequence and performance rely on physician judgment. Additionally, a determination of when a patient should be transferred to an emergency facility depends on the skill, experience, and clinical decision making of the individual physician. Preparedness, prompt recognition, and appropriate and aggressive treatment are integral to parts of successful management of anaphylaxis. A treatment log will assist in accurately recording progress.

Recommendations depend on practice resources and the proximity to other emergency assistance. Stocking and maintaining anaphylaxis supplies with regular written documentation of contents and expiration dates and ready availability of injectable epinephrine, intravenous fluids and needles, oxygen and mask cannula, airway adjuncts, and stethoscope and sphygmomanometer are bare essentials. (An example of a supply checklist is included in Figure 4 in the original guideline document. Not all items need to be present in each office.)

Regular anaphylaxis practice drills, the contents of which are left to the discretion and qualifications of the individual physician, are strongly recommended. Essential ingredients are identification of a person who will be responsible for calling emergency medical services and the person who will document treatment and time each is rendered. The emergency kit should be up to date and complete. Everyone who will be directly involved in patient care should, for example, be able to easily locate necessary supplies and rapidly assemble fluids for intravenous administration.

Annotation 2. Patient presents with possible-probable acute anaphylaxis

Anaphylaxis is an acute life-threatening reaction, usually but not always mediated by an immunologic mechanism (anaphylactoid reactions are IgE independent), that results from the sudden systemic release of mast cells and basophil mediators. It has varied clinical presentations, but respiratory compromise and cardiovascular collapse cause the most concern because they are the most frequent causes of fatalities. Urticaria and angioedema are the most common manifestations of anaphylaxis but might be delayed or absent in rapidly progressive anaphylaxis. The more rapidly anaphylaxis occurs after exposure to an offending stimulus, the more likely the reaction is to be severe and potentially life-threatening.

Anaphylaxis often produces signs and symptoms within minutes of exposure to an offending stimulus (see comments in text in the original guideline document), but some reactions might develop later (e.g., >30 minutes after exposure). Late-phase or biphasic reactions, which occur 8 to 12 hours after the initial attack, have also been reported. Protracted and severe anaphylaxis might last up to 32 hours, despite aggressive treatment.

Increased vascular permeability, a characteristic feature of anaphylaxis, allows transfer of as much as 50% of the intravascular fluid into the extravascular space within 10 minutes. As a result, hemodynamic collapse might occur rapidly with little or no cutaneous or respiratory manifestations.

Annotation 3. Initial assessment supports potential anaphylaxis

Initial assessment should determine whether history and physical findings are compatible with anaphylaxis. The setting of the episode and the history might suggest or reveal the source of the reaction. Evaluation should include level of consciousness (impairment might reflect hypoxia), upper and lower airways (dysphonia, stridor, cough, wheezing, or shortness of breath), cardiovascular system (hypotension with or without syncope and/or cardiac arrhythmias), the skin (diffuse or localized erythema, pruritus, urticaria, and/or angioedema), and the gastrointestinal system (nausea, vomiting, or diarrhea). In addition, some patients might have symptoms of lightheadedness, headache, uterine cramps, feeling of impending doom, and unconsciousness.

The vasodepressor (vasovagal) reaction probably is the condition most commonly confused with anaphylactic and anaphylactoid reactions. In vasodepressor reactions, however, urticaria is absent, the heart rate is typically bradycardic, bronchospasm or other breathing difficulty is generally absent, the blood pressure is usually normal or increased, and the skin is typically cool and pale. Tachycardia is the rule in anaphylaxis, but it might be absent in patients with conduction defects, with increased vagal tone caused by a cardioinhibitory (Bezold-Jarisch) reflex, or who take sympatholytic medications.

Annotation 4. Consider other diagnosis

Other diagnoses that might present with signs and/or symptoms characteristic of anaphylaxis should be excluded. Like anaphylaxis, several conditions might cause abrupt and dramatic patient collapse. Among conditions to consider are vasodepressor (vasovagal) reactions, acute anxiety (e.g., panic attack or hyperventilation syndrome), myocardial dysfunction, pulmonary embolism, systemic mast cell disorders, foreign-body aspiration, acute poisoning, hypoglycemia, and seizure disorder. Specific signs and symptoms of anaphylaxis might present singly in other disorders. Examples are urticaria-angioedema, hereditary angioedema, and asthma.

Annotation 5. Immediate intervention

The clinician should remember that anaphylaxis occurs as part of a continuum. Symptoms not immediately life-threatening might progress rapidly unless treated promptly. Treatment recommendations are subject to physician discretion, and variations in sequence and performance rely on physician judgment. Additionally, a determination of when a patient should be transferred to an emergency or intensive care facility depends on available resources and the skill, experience, and clinical decision making of the individual physician.

1. Assess airway, breathing, circulation, and level of consciousness (altered mentation might suggest the presence of hypoxia).
2. Administer epinephrine. Aqueous epinephrine 1:1000 dilution (1 mg/mL), 0.2 to 0.5 mL (0.01 mg/kg in children, maximum 0.3-mg dosage) intramuscularly or subcutaneously every 5 minutes, as necessary, should be used to control symptoms and increase blood pressure. Consider dose-response effects. Note: If the clinician deems it appropriate, the 5-minute interval between injections can be liberalized to permit more frequent injections. Intramuscular epinephrine injections into the thigh have been reported to provide more rapid absorption and higher plasma epinephrine levels in both children and

adults than intramuscular or subcutaneous injections administered in the arm. However, similar studies comparing intramuscular injections with subcutaneous injections in the thigh have not yet been done. Moreover, these studies were not performed in patients experiencing anaphylaxis. For this reason, the generalizability of these findings to the clinical setting of anaphylaxis has not been established. Although intuitively more rapid absorption and higher epinephrine levels would seem desirable, the clinical significance of this finding is not known. No data support the use of epinephrine in anaphylaxis through a nonparenteral route. However, alternative routes of administration have been anecdotally successful. These include, for example, inhaled epinephrine in the presence of laryngeal edema or sublingual administration if an intravenous route cannot be obtained. Endotracheally administered dosages have also been proposed for use when intravenous access is not available in intubated patients experiencing cardiac arrest.

Annotation 6. Subsequent emergency care that might be necessary depending on response to epinephrine

1. Place patient in the recumbent position and elevate the lower extremities, as tolerated symptomatically. This slows progression of hemodynamic compromise, if present, by preventing orthostatic hypotension and helping to shunt effective circulation from the periphery to the head and to the heart and kidneys.
2. Establish and maintain airway. Ventilatory assistance through a 1-way valve facemask with an oxygen inlet port (e.g., Pocket-Mask [Laerdal, Preparedness Industries, Ukiah, Calif] or similar device) might be necessary. Ambubags of less than 700 mL are discouraged in adults in the absence of an endotracheal tube because ventilated volume will not overcome 150 to 200 mL of anatomic dead space to provide effective tidal volume. (Ambubags can be used in children, provided the reservoir volume of the device is sufficient.) Endotracheal intubation or cricothyroidotomy might be considered where appropriate and provided that clinicians are adequately trained and proficient in this procedure.
3. Administer oxygen. Oxygen should be administered to patients with anaphylaxis who have prolonged reactions, have pre-existing hypoxemia or myocardial dysfunction, receive inhaled beta-agonists as part of therapy for anaphylaxis, or require multiple doses of epinephrine. Continuous pulse oximetry and/or arterial blood gas determination (where available) should guide oxygen therapy where hypoxemia is a concern.
4. Consider a normal saline intravenous line for fluid replacement and venous access. Lactated Ringer's solution might potentially contribute to metabolic acidosis, and dextrose is rapidly extravasated from the intravascular circulation to the interstitial tissues. Increased vascular permeability in anaphylaxis might permit transfer of 50% of the intravascular fluid into the extravascular space within 10 minutes. Crystalloid volumes (e.g., saline) of up to 7 L might be necessary. One to 2 L of normal saline should be administered to adults at a rate of 5 to 10 mL/kg in the first 5 minutes. Patients with congestive heart failure or chronic renal disease should be observed cautiously to prevent volume overload. Children should receive up to 30 mL/kg in the first hour. Adults receiving colloid solution should receive 500 mL rapidly, followed by slow infusion. Aqueous epinephrine 1:1000, 0.1

- to 0.3 mL in 10 mL of normal saline, can be administered intravenously over several minutes and repeated as necessary in cases of anaphylaxis not responding to epinephrine injections and volume resuscitation. Alternatively, an epinephrine infusion can be prepared by adding 1 mg (1 mL) of a 1:1000 dilution of epinephrine to 250 mL of D5W to yield a concentration of 4.0 micrograms/mL. This solution is infused at a rate of 1 to 4 micrograms/min (15 to 60 drops per minute with a microdrop apparatus [60 drops per minute = 1 mL = 60 mL/h]), increasing to a maximum of 10.0 micrograms/min. If an infusion pump is available, an alternative 1:100,000 solution of epinephrine (1 mg [1 mL] in 100 mL of saline) can be prepared and administered intravenously at an initial rate of 30 to 100 mL/h (5 to 15 micrograms/min), titrated up or down depending on clinical response or epinephrine side effects (toxicity). A dosage of 0.01 mg/kg (0.1 mL/kg of a 1:10,000 solution; maximum dose, 0.3 mg) is recommended for children. Alternative pediatric dosage by the "rule of 6" is as follows: 0.6 times body weight (in kilograms) = the number of milligrams diluted to a total of 100 mL of saline; then 1 mL/h delivers 0.1 microgram/kg/min. Note: Because of the risk for potentially lethal arrhythmias, epinephrine should be administered intravenously only during cardiac arrest or to profoundly hypotensive patients who have failed to respond to intravenous volume replacement and several injected doses of epinephrine. In situations in which hemodynamic monitoring is available (e.g., emergency department or intensive care facility), continuous hemodynamic monitoring is essential. However, use of intravenous epinephrine should not be precluded in a scenario in which such monitoring is unavailable if the clinician deems administration is essential after failure of several epinephrine injections. If intravenous epinephrine is considered essential under these special circumstances, monitoring by available means (e.g., every-minute blood pressure and pulse measurements and electrocardiographic monitoring, if available) should be conducted.
5. Consider diphenhydramine, 1 to 2 mg/kg or 25 to 50 mg per dose (parenterally). Note: H₁ antihistamines are considered second-line therapy to epinephrine and should never be administered alone in the treatment of anaphylaxis.
 6. Consider ranitidine, 50 mg in adults and 12.5 to 50 mg (1 mg/kg) in children, which might be diluted in 5% dextrose to a total volume of 20 mL and injected intravenously over 5 minutes. Cimetidine (4 mg/kg) can be administered intravenously to adults, but no pediatric dosage in anaphylaxis has been established. Note: In the management of anaphylaxis, a combination of diphenhydramine and ranitidine is superior to diphenhydramine alone. However, these agents have a much slower onset of action than epinephrine and should never be used alone in the treatment of anaphylaxis. Both alone and in combination, these agents are second-line therapy to epinephrine.
 7. Bronchospasm resistant to adequate doses of epinephrine: consider inhaled beta-agonist (e.g., nebulized albuterol, 2.5 to 5 mg in 3 mL of saline and repeat as necessary).
 8. Hypotension refractory to volume replacement and epinephrine injections: consider vasopressor infusion. Continuous hemodynamic monitoring is essential. For example, dopamine (400 mg in 500 mL of 5% dextrose) can be infused at 2 to 20 micrograms/kg/min and titrated to maintain systolic blood pressure of greater than 90 mm Hg.
 9. Consider glucagon infusion when concomitant beta-adrenergic blocking agent complicates treatment. Glucagon dosage is 1 to 5 mg (20 to 30

- micrograms/kg [maximum dose, 1 mg] in children) administered intravenously over 5 minutes and followed by an infusion (5 to 15 micrograms/min) titrated to clinical response.
10. Consider systemic glucocorticosteroids for patients with a history of idiopathic anaphylaxis or asthma and patients who experience severe or prolonged anaphylaxis. Glucocorticosteroids usually are not helpful acutely but potentially might prevent recurrent or protracted anaphylaxis. If given, intravenous glucocorticosteroids should be administered every 6 hours at a dosage equivalent to 1.0 to 2.0 mg/kg/d. Oral administration of glucocorticosteroids (e.g., prednisone, 0.5 mg/kg) might be sufficient for less critical anaphylactic episodes.
 11. Consider transportation to emergency department or intensive care facility.

Annotation 7. Cardiopulmonary arrest during anaphylaxis

1. Cardiopulmonary resuscitation and advanced cardiac life support measures.
2. High-dose epinephrine administered intravenously (i.e., rapid progression to high dose). A common sequence is 1 to 3 mg (1:10,000 dilution) slowly administered intravenously over 3 minutes, 3 to 5 mg administered intravenously over 3 minutes, and then a 4 to 10 microgram/min infusion. For children, the recommended initial resuscitation dosage is 0.01 mg/kg (0.1 mL/kg of a 1:10,000 solution up to 10 mg/min rate of infusion) repeated every 3 to 5 minutes for ongoing arrest. Higher subsequent dosages (0.1 to 0.2 mg/kg; 0.1 mL/kg of a 1:1,000 solution) might be considered for unresponsive asystole or pulseless electrical activity.
3. Rapid volume expansion.
4. Atropine and transcutaneous pacing if asystole and/or pulseless electrical activity are present.
5. Prolonged resuscitation is encouraged, if necessary, because efforts are more likely to be successful in anaphylaxis.
6. Transport to emergency department or intensive care facility, as setting dictates.

Annotation 8. Observation and subsequent follow-up

Observation periods must be individualized because there are no reliable predictors of biphasic or protracted anaphylaxis on the basis of initial clinical presentation. Follow-up accordingly must be individualized and based on such factors as clinical scenario and distance from the patient's home to the closest emergency facility. After resolution of the acute episode, patients should be provided with an epinephrine syringe and receive proper instruction for self-administration in case of a subsequent episode. In circumstances in which an allergist-immunologist is not already involved, it is strongly recommended that individuals who have experienced acute anaphylaxis should receive consultation from an allergist-immunologist regarding diagnosis, prevention, and treatment.

Annotation 9. Consider consultation with an allergist-immunologist

After acute anaphylaxis, patients should be assessed for future risk for anaphylaxis. The allergist-immunologist can obtain a detailed history, coordinate allergy diagnostic testing, evaluate the risks and benefits of therapeutic options, train and retrain in self-administration of epinephrine, and provide counseling on

avoidance measures (the most effective treatment for most causes of anaphylaxis). Consultation with an allergist-immunologist is recommended when:

1. The diagnosis is doubtful or incomplete.
2. The symptoms are recurrent or difficult to control.
3. Help is needed in evaluation and management of medication use or side effects.
4. Help is needed in medical management or adherence to treatment.
5. Help is needed in the diagnosis or management of IgE-mediated reactions or identification of allergic triggers.
6. The patient is a candidate for desensitization (e.g., penicillin) or immunotherapy (e.g., venom-specific immunotherapy).
7. The patient requires daily medications for prevention.
8. The patient requires intensive education regarding avoidance or management.
9. Help is needed with new or investigative therapy.
10. Treatment goals have not been met.
11. Anaphylaxis is complicated by one or more comorbid conditions or concomitant medications.
12. The patient has requested a subspecialty consultation.

Summary Statements

Evaluation and Management of the Patient with a History of Episodes of Anaphylaxis

1. The history is the most important tool to determine whether a patient has had anaphylaxis and the cause of the episode. (C)
2. A thorough differential diagnosis should be considered, and other conditions should be ruled out. (C)
3. Laboratory tests can be helpful to confirm a diagnosis of anaphylaxis or rule out other causes. Proper timing of studies (e.g., serum tryptase) is essential. (B)
4. In the management of a patient with a previous episode, education is necessary. Emphasis on early treatment, specifically the self-administration of epinephrine, is essential. (C)
5. The patient should be instructed to wear and/or carry identification denoting his or her condition (e.g., Medic Alert jewelry). (C)

Management of Anaphylaxis

6. Medical facilities should have an established protocol to deal with anaphylaxis and the appropriate equipment to treat the episode. In addition, telephone numbers for paramedical rescue squads and ambulance services might be helpful to have on hand. (B)
7. Anaphylaxis is an acute, life-threatening systemic reaction with varied mechanisms, clinical presentations, and severity that results from the sudden systemic release of mediators from mast cells and basophils. (B)
8. Anaphylactic (IgE-dependent) and anaphylactoid (IgE-independent) reactions differ mechanistically, but the clinical presentations are identical. (C)
9. The more rapidly anaphylaxis develops, the more likely the reaction is to be severe and potentially life threatening. (C)

10. Prompt recognition of signs and symptoms of anaphylaxis is crucial. If there is any doubt, it is generally better to administer epinephrine. (C)
11. Any health care facility should have a plan of action for anaphylaxis should it occur. Physicians and office staff should maintain clinical proficiency in anaphylaxis management. (D)
12. Epinephrine and oxygen are the most important therapeutic agents administered in anaphylaxis. Epinephrine is the drug of choice, and the appropriate dose should be administered promptly at the onset of apparent anaphylaxis. (A/D)
13. Appropriate volume replacement either with colloid or crystalloids and rapid transport to the hospital is essential for patients who are unstable or refractory to initial therapy for anaphylaxis in the office setting. (B)

Anaphylaxis to Foods

14. Severe food reactions have been reported to involve the gastrointestinal, cutaneous, respiratory, and cardiovascular systems. (D)
15. The greatest number of anaphylactic episodes in children has involved peanuts, tree nuts (i.e., walnuts, pecans, and others), fish, shellfish, milk, and eggs (C). The greatest number of anaphylactic episodes in adults is due to shellfish (C). Clinical cross-reactivity with other foods in the same group is unpredictable (B). Additives can also cause anaphylaxis (C).
16. Anaphylactic reactions to foods almost always occur immediately. Symptoms might then subside, only to recur several hours later. (A)
17. The most useful diagnostic tests include skin tests and food challenges. In vitro testing with foods might be appropriate as an alternative screening procedure. (C)
18. Double- or single-blind placebo-controlled food challenges can be done in patients with suspected food allergy in a medical facility by personnel experienced in performing the procedure and prepared to treat anaphylaxis. (B)
19. Patient education should include discussion about avoidance and management of accidental ingestion. (C)
20. Schools might present a special hazard for the student with food allergy. Epinephrine should be available for use by the individuals in the school trained to respond to such a medical emergency. (C)

Latex-Induced Anaphylaxis

21. Latex (rubber) hypersensitivity is a significant medical problem, and 3 groups are at higher risk of reaction: health care workers, children with spina bifida and genitourinary abnormalities, and workers with occupational exposure to latex. (C)
22. Skin prick tests with latex extracts should be considered for patients who are members of high-risk groups or who have a clinical history of possible latex allergy to identify IgE-mediated sensitivity. Although a standardized, commercial skin test reagent for latex is not available in the United States, many allergy centers have prepared latex extracts from gloves to be used for clinical testing. It should be noted, however, that such extracts prepared from gloves demonstrate tremendous variability in content of latex antigen. In vitro assays for IgE to latex might also be useful, although these tests are generally less sensitive than skin tests. (C)

23. Patients with spina bifida (regardless of a history of latex allergy) and other patients with a positive history of latex allergy ideally should have all medical-surgical-dental procedures performed in a latex-safe environment and as the first case of the day. (D)
24. A latex-safe environment is an environment in which no latex gloves are used in the room or surgical suite and no latex accessories (catheters, adhesives, tourniquets, and anesthesia equipment) come into contact with the patient. (D)
25. In health care settings general use of latex gloves with negligible allergen content, powder-free latex gloves, and nonlatex gloves and medical articles should be considered in an effort to minimize exposure to latex allergen. Such a combined approach might minimize latex sensitization of health care workers and patients and should reduce the risk of inadvertent reactions to latex in previously sensitized individuals. (C)

Anaphylaxis during General Anesthesia, the Intraoperative Period, and the Postoperative Period

26. The incidence of anaphylaxis during anesthesia has been reported to range from 1 in 4,000 to 1 in 25,000. Anaphylaxis during anesthesia can present as cardiovascular collapse, airway obstruction, flushing, and/or edema of the skin. (C)
27. It might be difficult to differentiate between immune and nonimmune mast cell-mediated reactions and pharmacologic effects from the variety of medications administered during general anesthesia. (B)
28. Thiopental allergy has been documented by using skin tests. (B)
29. Neuromuscular blocking agents, such as succinylcholine, can cause nonimmunologic histamine release, but there have been reports of IgE-mediated mechanisms in some cases. (B)
30. Reactions to opioid analgesics are usually caused by direct mast cell-mediator release rather than IgE-dependent mechanisms. (B)
31. Antibiotics that are administered perioperatively can cause immunologic or nonimmunologic generalized reactions. (B)
32. Protamine can also cause severe systemic reactions through IgE-mediated or nonimmunologic mechanisms. (B)
33. Latex is a potent allergen, and IgE-mediated reactions to latex during anesthesia have been clearly documented. Patients with multiple surgical procedures (e.g., patients with spina bifida) and health care workers are at greater risk of latex sensitization. Precautions for latex-sensitive patients include avoiding the use of latex gloves and latex blood pressure cuffs, as well as latex intravenous tubing ports and rubber stoppers from medication vials. (B)
34. Blood transfusions can elicit a variety of systemic reactions, some of which might be IgE mediated or mediated through other immunologic mechanisms. (B)
35. Methylmethacrylate (bone cement) has been associated with hypotension and various systemic reactions, although no IgE mechanism has yet been documented. (C)
36. The evaluation of IgE-mediated reactions to medications used during anesthesia can include skin testing to a variety of anesthetic agents. (B)

37. The management of anaphylactic or anaphylactoid reactions that occur during general anesthesia is similar to the management of anaphylaxis in other situations. (B)

Seminal Fluid-Induced Anaphylaxis

38. Anaphylaxis caused by human seminal fluid has been shown to be due to IgE-mediated sensitization by proteins of varying molecular weights. (B)
39. Localized seminal plasma hypersensitivity has been well described and is likely IgE mediated on the basis of successful response to rapid seminal plasma desensitization. (C)
40. History of atopic disease is the most consistent risk factor. However, anecdotal case reports have been associated with gynecologic surgery, injection of anti-RH immunoglobulin, and the postpartum state. (C)
41. The diagnosis is confirmed by means of skin and/or in vitro tests for serum-specific IgE by using proper reagents obtained from fractionation of seminal fluid components. (C)
42. Prevention of reactions to seminal fluid can be accomplished by barrier use of condoms. (C)
43. Immunotherapy to properly fractionated seminal fluid proteins has been universally successful in preventing anaphylaxis to seminal fluid, provided the sensitizing seminal fluid fractions are used as immunogens. Successful intravaginal graded challenge with unfractionated seminal fluid has been reported in a few cases, but the duration of protection is unknown. (C)
44. Localized and/or systemic seminal plasma hypersensitivity is not associated with infertility. (D)

Exercise-Induced Anaphylaxis

45. Exercise-induced anaphylaxis is a form of physical allergy. Premonitory symptoms can include diffuse warmth, itching, and erythema. Urticaria generally ensues, with progression to confluence and often angioedema. Episodes can progress to include gastrointestinal symptoms, laryngeal edema, and/or vascular collapse. (B)
46. Factors that have been associated with exercise-induced anaphylaxis include medications (e.g., aspirin and other nonsteroidal anti-inflammatory drugs) or food ingestion before and after exercise. (C)
47. Patients with exercise-induced anaphylaxis might have a higher incidence of personal and/or family history of atopy. (C)
48. Medications used prophylactically are not useful in preventing exercise-induced anaphylaxis. (C)
49. If exercise-induced anaphylactic episodes have been associated with the ingestion of food, exercise should be avoided in the immediate postprandial period. (C)
50. Patients with exercise-induced anaphylaxis should carry epinephrine and should wear and/or carry Medic Alert identification denoting their condition. They should have a companion with them when exercising. This companion should be versed in the use of an EpiPen. (D)

Idiopathic Anaphylaxis

51. The symptoms of idiopathic anaphylaxis are identical to those of episodes related to known causes. (C)
52. Patients with idiopathic anaphylaxis should receive an intensive evaluation, including a meticulous history to rule out a definite cause of the events. (C)
53. There might be a need for specific laboratory studies to exclude systemic disorders, such as systemic mastocytosis. This might include a serum tryptase level when the patient is asymptomatic, a ratio of beta-tryptase to total tryptase during an event, and selective allergy skin testing. (C)

Anaphylaxis and Allergen Immunotherapy Vaccines

54. There is a small risk of near-fatal and fatal anaphylactic reactions to allergen immunotherapy injections. (C)
55. Patients with asthma, particularly poorly controlled asthma, are at higher risk for serious systemic reactions to allergen immunotherapy injections (C). Patients taking beta- adrenergic blocking agents are at higher risk for serious systemic reactions to allergen immunotherapy injections. (B)
56. Allergen immunotherapy vaccines should be administered only by health care professionals trained in the recognition and treatment of anaphylaxis, only in health care facilities with the proper equipment for the treatment of anaphylaxis, and in clinics with policies and procedures that minimize the risk of anaphylaxis. (D)

Anaphylaxis to Drugs

57. Low-molecular-weight medications induce an IgE-mediated reaction only after combining with a carrier protein to produce a complete multivalent antigen. (B)
58. Penicillin is the most common cause of drug-induced anaphylaxis. (C)
59. Penicillin spontaneously degrades to major and minor antigenic determinants, and skin testing with reagents on the basis of these determinants yields negative results in about 90% of patients with a history of penicillin allergy. (B)
60. The negative predictive value of penicillin skin testing (for immediate-type reactions) is between 97% and 99% (depending on the reagents used), and the positive predictive value is at least 50%. (B)
61. The extent of allergic cross-reactivity between penicillin and cephalosporins is unknown but appears to be low. Four percent of patients proved to have penicillin allergy by means of penicillin skin testing react to cephalosporin challenges. (C)
62. Patients with a history of penicillin allergy who have negative penicillin skin test responses might safely receive cephalosporins. (B)
63. Patients with a history of penicillin allergy who have positive penicillin skin test responses might (1) receive an alternate (non-beta-lactam) antibiotic, (2) receive a cephalosporin through graded challenge, or (3) receive a cephalosporin through rapid desensitization. (F)
64. Aztreonam does not cross-react with other beta lactams, except ceftazidime, with which it shares a common R-group side chain. (B)
65. Carbapenems should be considered cross-reactive with penicillin. (C)
66. Diagnosis of IgE-mediated reactions to non-beta-lactam antibiotics is limited by a lack of knowledge of the relevant allergenic determinants and/or metabolites. (C)

67. Aspirin and nonsteroidal anti-inflammatory drugs are the second most common cause of drug-induced anaphylaxis. (C)
68. Anaphylactic reactions to aspirin and nonsteroidal anti-inflammatory drugs appear to be medication specific and do not cross-react with structurally unrelated aspirin or other nonsteroidal anti-inflammatory drugs. (D)

Prevention of Anaphylaxis

69. Major risk factors related to anaphylaxis include, but are not limited to, prior history of such reactions, concomitant beta-adrenergic blocker therapy, exposure, or atopic background. Atopic background might be a risk factor for venom- and latex-induced anaphylaxis and possibly anaphylactoid reactions to radiographic contrast material but not for anaphylactic reactions to medications.
70. Avoidance measures are successful if future exposure to drugs, foods, additives, or occupational allergens can be prevented. Avoidance of stinging and biting insects is also possible in many cases. Prevention of systemic reactions during allergen immunotherapy is dependent on the specific circumstances involved.
71. Avoidance management should be individualized, taking into consideration factors such as age, activity, occupation, hobbies, residential conditions, access to medical care, and the patients' level of personal anxiety.
72. Pharmacologic prophylaxis should be used to prevent recurrent anaphylactoid reactions to radiographic contrast material, fluorescein, as well as to prevent idiopathic anaphylaxis. Prophylaxis with glucocorticosteroids and antihistamines markedly reduces the occurrence of subsequent reactions.
73. Allergen immunotherapy with the appropriate stinging insect venom should be recommended for patients with systemic sensitivity to stinging insects because this treatment is highly (90 to 98%) effective.
74. Desensitization to medications that are known to have caused anaphylaxis can be effective. In most cases the effect of desensitization is temporary, and if the medication is required some time in the future, the desensitization process must be repeated.
75. Patient education might be the most important preventive strategy. Patients should be carefully instructed about hidden allergens, cross-reactions to various allergens, unforeseen risks during medical procedures, and when and how to use self-administered epinephrine. Physicians should educate patients about the risks of future anaphylaxis, as well as the benefits of avoidance measures.

Definitions:

Strength of Recommendation

- A. Directly based on category I evidence
- B. Directly based on category II evidence or extrapolated recommendation from category I evidence
- C. Directly based on category III evidence or extrapolated recommendation from category I or II evidence
- D. Directly based on category IV evidence or extrapolated recommendation from category I, II, or III evidence

Category of Evidence

Ia Evidence from meta-analysis of randomized controlled trials

Ib Evidence from at least one randomized controlled trial

IIa Evidence from at least one controlled study without randomization

IIb Evidence from at least one other type of quasiexperimental study

III Evidence from nonexperimental descriptive studies, such as comparative studies

IV Evidence from expert committee reports or opinions or clinical experience of respected authorities or both

CLINICAL ALGORITHM(S)

Algorithms are provided in the original guideline document for:

- The initial evaluation and management of a patient with a history of an episode of anaphylaxis
- The treatment of acute anaphylaxis

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of supporting evidence is identified for each recommendation (see "Major Recommendations").

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Identification, prevention, and management of anaphylaxis and anaphylactoid reactions

POTENTIAL HARMS

- Because of the risk for potentially lethal arrhythmias, epinephrine should be administered intravenously only during cardiac arrest or to profoundly hypotensive subjects who have failed to respond to intravenous volume replacement and several injected doses of epinephrine.
- Cimetidine, 4 mg/kg in adults, should be administered slowly because rapid intravenous administration might produce hypotension. Cimetidine should not be administered to children with anaphylaxis because no dosages have been established.

- Often, but not always, pruritis of the oral tissues or nausea is the initial complaint after food challenge.
- There is a small risk of near-fatal and fatal anaphylactic reactions to allergen immunotherapy injections. Patients with asthma, particularly poorly controlled asthma, and patients taking beta-adrenergic blocking agents are at higher risk for serious systemic reactions to allergen immunotherapy injections.

CONTRAINDICATIONS

CONTRAINDICATIONS

Beta-adrenergic blockade is a relative contraindication for allergen immunotherapy.

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

- This is a complete and comprehensive document at the current time. The medical environment is a changing environment, and not all recommendations will be appropriate for all patients.
- Because this document incorporated the efforts of many participants, no single individual, including those who served on the Joint Task Force, is authorized to provide an official American Academy of Allergy, Asthma, and Immunology (AAAAI) or American College of Allergy, Asthma, and Immunology (ACAAI) interpretation of these practice parameters. Any request for information about or an interpretation of these practice parameters by the AAAAI or ACAAI should be directed to the Executive Offices of the AAAAI, the ACAAI, and the Joint Council of Allergy, Asthma, and Immunology.
- These parameters are not designed for use by pharmaceutical companies in drug promotion.
- This parameter was edited by Dr Nicklas in his private capacity and not in his capacity as a medical officer with the Food and Drug Administration. No official support or endorsement by the Food and Drug Administration is intended or should be inferred.
- The Joint Task Force on Practice Parameters recognizes that there are different, although appropriate, approaches to the diagnosis and management of anaphylactic reactions that often require flexible recommendations. Therefore the diagnosis and management of anaphylactic reactions must be individualized on the basis of unique features in particular patients.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

IMPLEMENTATION TOOLS

Clinical Algorithm

For information about [availability](#), see the "Availability of Companion Documents" and "Patient Resources" fields below.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Living with Illness
Staying Healthy

IOM DOMAIN

Effectiveness
Patient-centeredness
Safety
Timeliness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

The diagnosis and management of anaphylaxis: an updated practice parameter. J Allergy Clin Immunol 2005 Mar;115(3 Suppl):S483-523. [232 references] [PubMed](#)

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

1998 Jun (revised 2005 Mar)

GUIDELINE DEVELOPER(S)

American Academy of Allergy, Asthma and Immunology - Medical Specialty Society
American College of Allergy, Asthma and Immunology - Medical Specialty Society
Joint Council of Allergy, Asthma and Immunology - Medical Specialty Society

GUIDELINE DEVELOPER COMMENT

These guidelines were developed by the Joint Task Force on Practice Parameters for Allergy and Immunology, which is sponsored by the American Academy of Allergy, Asthma, and Immunology, the American College of Allergy, Asthma, and Immunology and the Joint Council of Allergy, Asthma and Immunology.

SOURCE(S) OF FUNDING

Funded by the American Academy of Allergy, Asthma, and Immunology (AAAAI), the American College of Allergy, Asthma, and Immunology (ACAAI) and the Joint Council of Allergy, Asthma and Immunology (JCAAI).

GUIDELINE COMMITTEE

Joint Task Force on Practice Parameters
Work Group on Diagnosis and Management of Anaphylaxis

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

Chief Editors: Phillip Lieberman, MD, Departments of Medicine and Pediatrics, University of Tennessee, College of Medicine, Memphis, Tennessee; Stephen F. Kemp, MD, Departments of Medicine and Pediatrics, University of Mississippi Medical Center, Jackson, Mississippi; John Oppenheimer, MD, Department of Internal Medicine, New Jersey Medical School, Pulmonary and Allergy Associates, Morristown, New Jersey; David M. Lang, MD, Allergy/Immunology Section, Division of Medicine, Director, Allergy and Immunology Fellowship, Training Program, Cleveland Clinic Foundation, Cleveland, Ohio; I. Leonard Bernstein, MD, Departments of Medicine and Environmental Health, University of Cincinnati College of Medicine, Cincinnati, Ohio; Richard A. Nicklas, MD, Department of Medicine, George Washington Medical Center, Washington, DC

Work Group on Diagnosis and Management of Anaphylaxis

Contributors: John Anderson, MD, Aspen Medical Center, Fort Collins, Colorado; David I. Bernstein, MD, Department of Clinical Medicine, Division of Immunology, University of Cincinnati College of Medicine, Cincinnati, Ohio; Jonathan A. Bernstein, MD, University of Cincinnati College of Medicine, Department of Internal Medicine, Division of Immunology/Allergy Section, Cincinnati, Ohio; Jordan N. Fink, MD, Allergy-Immunology, Departments of Pediatrics and Medicine, Medical College of Wisconsin, Milwaukee, Wisconsin; Paul A. Greenberger, MD, Division of Allergy and Immunology, Northwestern University Feinberg School of Medicine, Chicago, Illinois; Dennis K. Ledford, MD, Department of Medicine, University of South Florida College of Medicine and the James A. Haley V.A. Hospital, Tampa, Florida; James T. Li, MD, PhD, Mayo Clinic, Rochester, Minnesota; Albert L. Sheffer, MD, Brigham and Women's Hospital, Boston, Massachusetts; Roland Solensky, MD, The Corvallis Clinic, Corvallis, Oregon; Bruce L. Wolf, MD, Vanderbilt University, Nashville, Tennessee

FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Disclosure of potential conflict of interest: P. Lieberman-none disclosed. S. F. Kemp has consultant arrangements with MedPointe Pharmaceuticals. J. Oppenheimer-none disclosed. D. M. Lang-none disclosed. I. L. Bernstein-none disclosed. R. A. Nicklas-none disclosed. J. A. Anderson-none disclosed. D. I. Bernstein-none disclosed. J. Bernstein is on the Speakers' Bureau for Merck, GlaxoSmithKline, AstraZeneca, Aventis, Medpointe, Pfizer, Schering-Plough, and IVAX. J. N. Fink-none disclosed. P. A. Greenberger-none disclosed. D. K. Ledford-none disclosed. J. Li-none disclosed. A. L. Sheffer-none disclosed. R. Solensky-none disclosed. B. L. Wolf-none disclosed. J. Blessing-Moore-none disclosed. D. A. Khan has consultant arrangements with Pfizer; receives grants/research support

from AstraZeneca; and is on the Speakers' Bureau for Pfizer, Merck, Aventis, and GlaxoSmithKline. R. E. Lee-none disclosed. J. M. Portnoy-none disclosed. D. E. Schuller is on the Speakers' Bureau for Boehringer-Ingelheim. S.L. Spector has had consultant arrangements with, has received grants/research support from, and has been on the Speakers' Bureau for Abbott, Allen & Hanburys, AstraZeneca, Boehringer-Ingelheim, GlaxoSmithKline, Fisons, Forest, ICN, Key, Eli-Lilly, 3M, Miles, Muro, Pfizer, Purdue Frederick, Schering-Plough, Wallace, and Witby. S. A. Tilles-none disclosed.

GUIDELINE STATUS

This is the current release of the guideline.

This guideline updates a previous version: Joint Council of Allergy, Asthma and Immunology. The diagnosis and management of anaphylaxis. J Allergy Clin Immunol 1998 Jun;101(6 Pt 2):S465-S528.

GUIDELINE AVAILABILITY

Electronic copies: Available in Portable Document Format (PDF) from the [Joint Council of Allergy, Asthma, and Immunology \(JCAAI\) Web site](#).

Print copies: Available from JCAAI, 50 N. Brockway, Ste 3-3 Palatine, IL 60067.

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

This summary was completed by ECRI on October 1, 1998. The information was verified by the guideline developer on December 15, 1998. This NGC summary was updated by ECRI on May 10, 2005. The updated information was verified by the guideline developer on May 23, 2005.

COPYRIGHT STATEMENT

This NGC summary is based on the original guideline, which is subject to the guideline developer's copyright restrictions. Reproduced with the permission from the American College of Allergy, Asthma, and Immunology.

DISCLAIMER

NGC DISCLAIMER

The National Guideline Clearinghouse™ (NGC) does not develop, produce, approve, or endorse the guidelines represented on this site.

All guidelines summarized by NGC and hosted on our site are produced under the auspices of medical specialty societies, relevant professional associations, public or private organizations, other government agencies, health care organizations or plans, and similar entities.

Guidelines represented on the NGC Web site are submitted by guideline developers, and are screened solely to determine that they meet the NGC Inclusion Criteria which may be found at <http://www.guideline.gov/about/inclusion.aspx>.

NGC, AHRQ, and its contractor ECRI Institute make no warranties concerning the content or clinical efficacy or effectiveness of the clinical practice guidelines and related materials represented on this site. Moreover, the views and opinions of developers or authors of guidelines represented on this site do not necessarily state or reflect those of NGC, AHRQ, or its contractor ECRI Institute, and inclusion or hosting of guidelines in NGC may not be used for advertising or commercial endorsement purposes.

Readers with questions regarding guideline content are directed to contact the guideline developer.

© 1998-2008 National Guideline Clearinghouse

Date Modified: 9/15/2008

