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Overview of allergic disease: Anaphylaxis – WAO White Book on Allergy 2026 – 2.11

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ABSTRACT

Anaphylaxis is a rapid-onset, potentially fatal systemic hypersensitivity reaction marked by airway, breathing, or circulatory compromise, which may occur even without skin symptoms. It is driven primarily by mast-cell activation—usually through IgE-mediated pathways—leading to the release of mediators such as tryptase and histamine. Basophils can also contribute. Although traditionally considered rare, anaphylaxis has a lifetime prevalence of 0.3–5.1%, with incidence rising globally in both adults and children, likely due to increased allergic diseases. Fatalities remain relatively low. Common triggers vary by age and geography: foods such as milk, peanuts, and tree nuts dominate in children; medications—especially beta-lactam antibiotics and non-steroidal anti-inflammatory drugs (NSAIDs)—are more common in adults; and insect venoms are major causes in some regions.

Diagnosis is clinical, based on rapid multisystem involvement, despite sometimes isolated cardiovascular or non-inhaled respiratory symptoms may occur. First measures should be to remove the allergen if possible and position the patient appropriately. Immediate intramuscular epinephrine injected into the lateral thigh is the first-line treatment, with repeat dosing as needed. Intranasal epinephrine is emerging as a needle-free alternative. Adjunctive measures include oxygen, intravenous fluids, and bronchodilators for bronchospasm. Antihistamines and corticosteroids may alleviate some symptoms but do not reverse anaphylaxis and must not delay epinephrine administration.

Long-term management focuses on identifying triggers through detailed history, IgE testing, skin testing, or challenge procedures; however, up to 10% of cases remain idiopathic. Patients require action plans and access to self-administered epinephrine, though availability is limited in many regions. Key unmet needs include global device access, harmonized definitions and severity scoring, improved education, digital health integration, and deeper investigation into mechanisms, biomarkers, genetics, and cofactors to optimize diagnosis, prevention, and care.

Keywords: Anaphylaxis, Severe anaphylaxis, Clinical diagnosis, Anaphylaxis triggers, Adrenaline, Epinephrine, Epinephrine auto-injectors

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Key statements

- Anaphylaxis is a serious systemic hypersensitivity reaction that is usually rapid in onset and may cause death.
- Severe anaphylaxis is characterized by potentially life-threatening compromise in the airway, breathing, and/or the circulation, and may occur without typical skin features or circulatory shock being present.
- Intramuscular epinephrine (adrenaline), administered in the lateral mid-thigh, or via intranasal spray, is currently the first-line treatment for anaphylaxis.
- The diagnosis of anaphylaxis is based on clinical criteria.
- The 3 most common causes of anaphylaxis are food, drugs, and insect venoms.

Unmet needs in anaphylaxis include the need for universal availability of self-administered epinephrine eg, auto-injectors and intranasal devices.

OVERVIEW

In this concise narrative review on anaphylaxis, information has been drawn from the literature of the last decade. It is aligned with previously published World Allergy Organization (WAO) documents for the text and figures.

Anaphylaxis is a generalized allergic reaction that can be severe and, in extreme cases, may even be fatal. A trigger initiates a reaction in which mediators are rapidly released, causing a variety of symptoms that may affect multiple organ systems. The following is the latest definition of anaphylaxis proposed by the WAO:

> "Anaphylaxis is a serious systemic hypersensitivity reaction that is usually rapid in onset and may cause death. Severe anaphylaxis is characterized by potentially life-threatening compromise in airway, breathing, and/or the circulation, and may occur without typical skin features or circulatory shock being present."[1]

Mast cells are the primary effector cells for driving anaphylaxis in humans, and increased levels of mast-cell mediators such as tryptase and histamine have been detected during episodes. Additionally, basophils, when activated, may theoretically contribute to symptom development by secreting mediators including histamine and leukotrienes. The major activator of these cells is specific immunoglobulin E (IgE) through its high-affinity receptor, upon cross-linking by the allergen. Other pathways have also been described (direct mast-cell activation, other receptors).[2]

Major inequities have been found in different geographical regions regarding the recognition, diagnosis, and acute and long-term management of anaphylaxis, especially in provider training, access to epinephrine devices, registries, and systematic follow-up.[1,3–6]

EPIDEMIOLOGY

Anaphylaxis has been considered a rare event, but data indicate that the lifetime prevalence may be around 0.3%–5.1%.[7,8] Despite low mortality (<1 death per million population per year),[9] patients at risk of anaphylaxis experience a deleterious impact on their quality of life.[10] The global incidence of anaphylaxis is reported to be from 50 to 112 episodes per 100,000 person-years,[7] while in children reported incidence ranges widely between 1 and 761 per 100,000 person-years.[11] According to a recent metanalysis, the estimated global incidence is approximately 46 cases per 100,000 people per year (95% CI 21–103), with anaphylaxis incidence increasing annually by about 7.4% (95% CI 7.3–7.6; $p < 0.05$).[12] Wide variation in reported prevalence and incidence of anaphylaxis arises from differences in case definitions, diagnostic criteria, and coding practices, as well as underrecognition and misclassification in clinical settings. Additional variability reflects differences in study design, populations, healthcare access, and reporting systems across regions.

Based on recent studies, the worldwide incidence of anaphylaxis and admission rates are increasing both in children and adults.[13–16] This may be partly due to an improvement in recognition but is most likely due more to a rise

Organ/system	Signs/symptoms
Skin	Itch (especially palms-soles, genitalia, scalp), erythema, urticaria, angioedema, morbilliform rash
Respiratory	Dyspnea, wheezing, cough, rhinoconjunctivitis, hypoxemia, throat itching, chest tightening, hoarseness, dysphonia, stridor, increased respiratory rate, decreased peak flow, cyanosis, respiratory arrest
Cardiovascular (or end-organ symptoms)	Palpitations, hypotension, chest pain, tachycardia, bradycardia (less common), dizziness, collapse, loss of consciousness, incontinence
Digestive	Nausea, vomiting, abdominal cramps, diarrhea, dysphagia
Other	Uterine cramps, uterine bleeding, sense of impending doom, metallic taste, confusion

Table 1. Signs and symptoms in anaphylaxis.

in allergic diseases in general and of food and drug allergies in particular. In contrast, fatality rates seem to be stable or to be decreasing slightly.[17]

Triggers of anaphylaxis vary depending on the age of the studied population and the geographical area; the 3 most important are food, drugs, and insect venom. Food is the main anaphylaxis trigger in children, with cow's milk, peanuts, and tree nuts being the most notable.[18,19] Drug allergy is more relevant in adults; antibiotics (beta-lactams) and nonsteroidal anti-inflammatory drugs (NSAIDs) are the main triggers. Hymenoptera insects (bees, wasps, and red ants) are the leading causes of anaphylaxis in some countries.[20]

DIAGNOSIS

Anaphylaxis should be suspected upon the development of a clinical picture suggestive of an allergic reaction usually affecting multiple organ systems, despite it can also manifest as isolated breathing or cardiocirculatory symptoms (Table 1). The onset is usually rapid, occurring in minutes. The diagnosis of anaphylaxis is made clinically as there are no diagnostic biomarkers. The diagnosis can be especially challenging in young children who cannot express symptoms.

Clinical criteria have been defined for the diagnosis of anaphylaxis (Fig. 1).[1]

In 2025, the Global Allergy and Asthma Excellence Network (GA^{2}LEN) proposed a set of clinical criteria (clinical support tool) (Fig. 2), fundamentally aligned with WAO 2020, especially in recognizing isolated respiratory or cardiovascular presentations after likely allergen exposure, with only minor contextual and editorial differences.[21]

Some laboratory assays are used to support the diagnosis of anaphylaxis, as part of the follow-up evaluation. The most reliable marker is serum tryptase, despite it may not rise in some cases. An increase of 20% above basal levels plus 2 mcg/L is suggestive of acute mast cell activation.[22] Also, it is helpful to evaluate other conditions (see "Prevention").

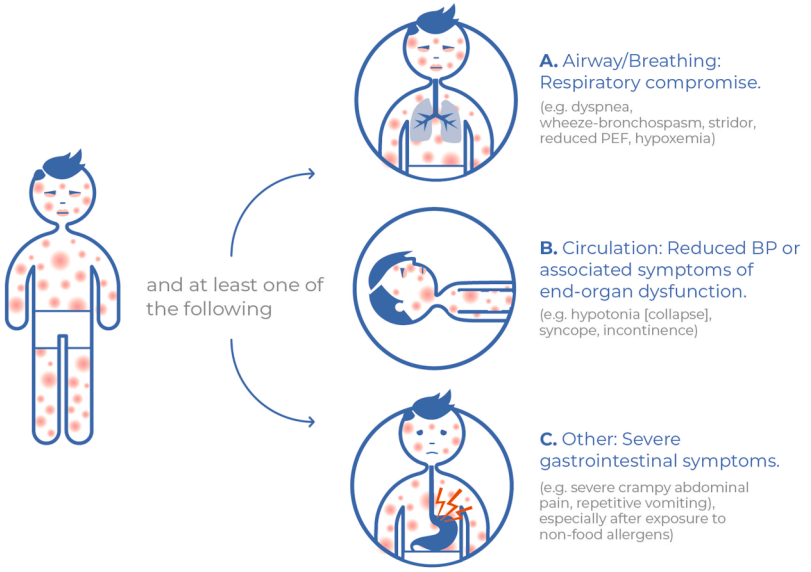
TREATMENT

Patients should be placed lying down with legs elevated to support venous return or semi-recumbent, in case of breathing difficulty or nausea. Women in advanced pregnancy should lie on the left side, to avoid compression of the cava by the uterus. A quick assessment of the patient's status should be performed, and help requested. If possible, triggers should be removed. Vital parameters should be monitored promptly.

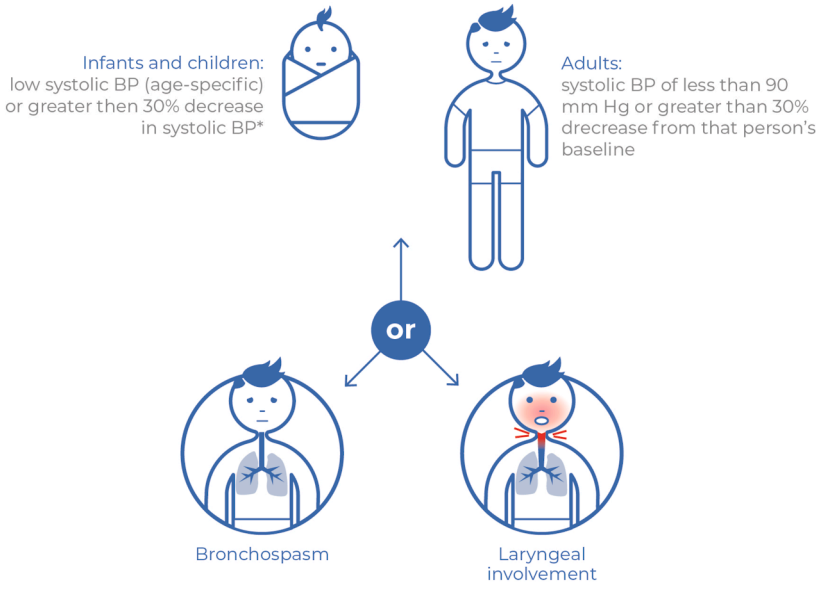
Currently, intramuscular and intranasal epinephrine (adrenaline) are accepted choices for the treatment of anaphylaxis. Intramuscular epinephrine in the lateral mid-thigh has been considered the first-line treatment for anaphylaxis until now (Fig. 3).[1] It allows a rapid pharmacological action and overcomes the potential risks inherent to intravenous

Anaphylaxis is highly likely when any one of the following **two criteria is fulfilled**

1 Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g. generalized hives, pruritus or flushing, swollen lips-tongue-uvula)



2 Acute onset of **hypotension*** or **bronchospasm** or **laryngeal involvement#** after exposure to a known or highly probable allergen for that patient (minutes to several hours), **even in the absence of typical skin involvement.**



PEF, Peak expiratory flow; BP, blood pressure.

* Hypotension defined as a decrease in systolic BP greater than 30% from that person's baseline, OR
  i. Infants and children under 10 years: systolic BP less than (70mmHg + [2 x age in years])
  ii. Adults: systolic BP less than < 90 mmHg

Laryngeal symptoms include: stridor, vocal changes, odynophagia.

administration (ie, overdosing). There are no absolute contraindications for epinephrine, although special care should be taken in the case of patients with higher risk of adverse effects (eg, older age, cardiovascular disease). The recommended dosing is shown in Table 2,[1] which may be repeated after 5–10 min if symptoms persist. An insufficient response may occur in patients on beta-blockers; if so, glucagon (intramuscular or intravenous) may be considered.

Intranasal epinephrine is a new needle-free option for the emergency treatment of anaphylaxis. Delivered as a nasal spray, it has been shown to provide blood levels and cardiovascular effects comparable to traditional intramuscular injections. This formulation offers potential advantages, including ease of use, avoidance of needles, and greater convenience for both patients and caregivers. Common side effects may include mild nasal or throat irritation, headache, or sneezing, but overall it represents a promising alternative for people at risk of severe allergic reactions.[23] Currently, no head to head studies have been published comparing injectable and non-injectable epinephrine and regulatory approval was based on pharmacokinetic and pharmacodynamic data.

Second-line therapy consists of intravenous fluids and oxygen supplementation. In case of bronchospasm, the addition of inhaled beta-adrenergic bronchodilators may be of help.

H1-antihistamines and corticosteroids have traditionally been recommended and used in the treatment of anaphylaxis. Their benefits are unclear; although they may decrease some symptoms, they are insufficient to reverse an anaphylactic reaction. Their use should not hinder or substitute for the administration of epinephrine.

Patients at risk of anaphylaxis need to have a written action plan in case of new reactions; epinephrine for self-treatment should be prescribed. Self-administered epinephrine devices are preferred; but unfortunately, they are not available in all countries.[6,23,24] Availability of epinephrine autoinjectors (EAIs) is widespread in Europe, the United States, Canada, and Australia, but much more limited in Asia, South America, Africa, and parts of the Middle East, where EAIs may be absent, available only via importation, or accessible on a named-patient/special-license basis.[24] Intranasal epinephrine is currently marketed in the United States and has been approved by the European Medicines Agency, ongoing market access in different countries.

Alternatives where self-administered devices are lacking include prefilled epinephrine syringes or a vial plus syringe; in such cases, thorough patient education on how to use them is required.

PREVENTION

Long-term management is aimed at preventing new episodes of anaphylaxis. Therefore, a thorough allergy work-up is needed to identify the potential trigger in order to avoid it. All patients who have experienced an anaphylactic reaction, or who may be at risk of experiencing one, should be referred to an experienced specialist in allergic diseases. Nevertheless, in up to 10% of cases, the exact cause remains unknown; such cases are labeled as idiopathic anaphylaxis, an exclusion diagnosis (Table 3).[25] In such cases, mast-cell disorders (eg, basal tryptase, C-kit mutations) should be ruled out.[1]

Diagnostic tests should be performed according to the clinical history, which in most cases will guide us on the potential triggers. Allergic sensitization is assessed through skin tests and *in vitro* determination of specific IgE. Challenge tests may be required to confirm or rule out some causes, depending on the benefit/risk balance. Certain causative allergens (alpha-gal, omega-5-gliadin, oleosins) need a high clinical suspicion, because their assessment may be less straightforward.

Avoidance of culprit agents should be based on robust data to avoid unnecessary diets or limitations in the use of drugs with high therapeutic value. Immunomodulatory approaches, such as

Fig. 1 Clinical criteria for the diagnosis of anaphylaxis. PEF, Peak expiratory flow; BP blood pressure. * Hypotension defined as a decrease in systolic BP greater than 30% from that person's baseline, OR Infants and children under 10 years: systolic BP less than (70 mmHg + [2 × age in years]) Adults: systolic BP < 90 mmHg #Laryngeal symptoms include stridor, vocal changes, odynophagia (Cardona et al,[1] with permission).

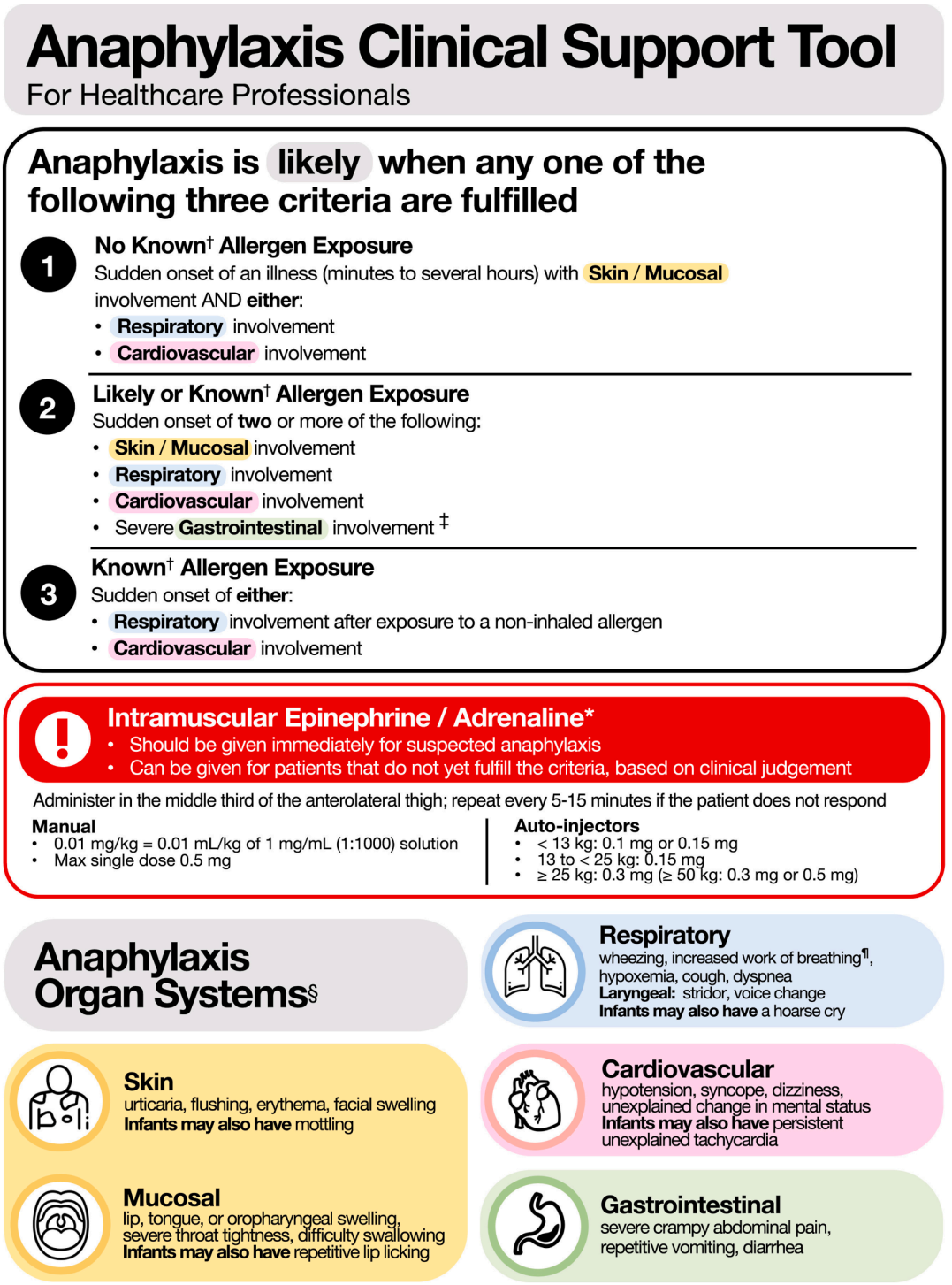


Fig. 2 Anaphylaxis clinical support tool. *Recommendations from AAAAI, ACAAI, AAP, CSACI, and EAACI. Autoinjector dosing recommendations may not be in accordance with manufacturer recommendations. ASCIA recommends transitioning to 0.3 mg autoinjector for children weighing >20 kg. Some organizations recommend age-based dosing,[27-30] as follows: <12 months, 0.1 mg; <6 years, 0.15 mg; ≥6 years, 0.3 mg; and adolescents/adults, 0.5 mg. Intranasal epinephrine (Neffy) can be provided to patients weighing ≥30 kg. Administer 1 spray (2 mg epinephrine) in 1 nostril. If symptoms do not improve or worsen after initial treatment, administer second dose in same nostril with new nasal spray starting 5 min after first dose. †*Allergen* broadly includes any anaphylaxis trigger (eg, foods, insect stings, medications, exercise), irrespective of underlying mechanism. *No known allergen exposure* means the provider cannot determine whether there was allergen exposure or cannot identify likely allergens. Known allergens do not require confirmatory testing, as when suspicious symptoms develop after insect sting in someone without existing diagnosis. ‡Gastrointestinal involvement after noningested allergen exposure suggests anaphylaxis. Gastrointestinal involvement after ingested allergen exposure may be due to local and/or systemic reactions, a distinction that may be difficult to make in clinical practice. §Table I lists possible anaphylaxis signs/

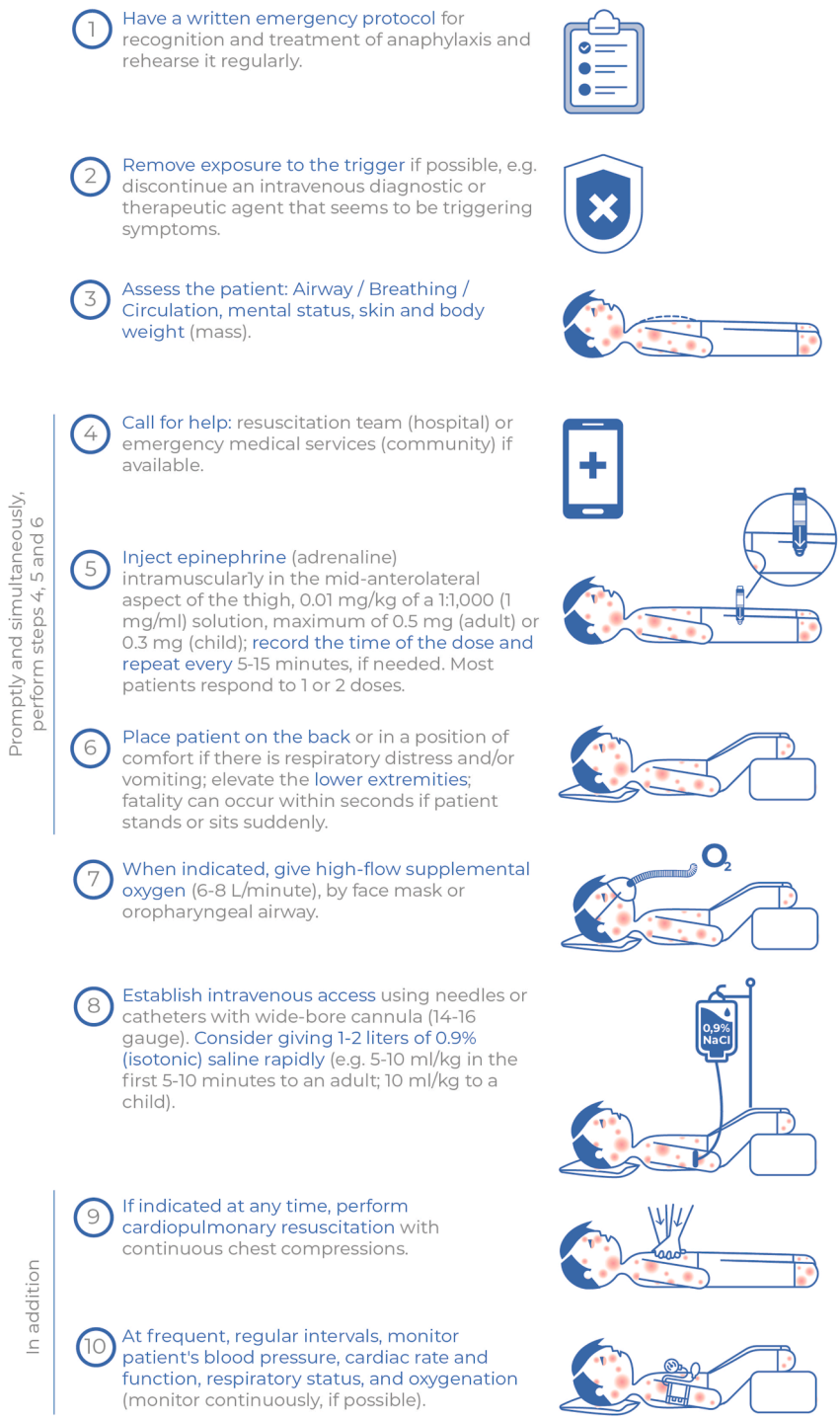


Fig. 3 Management of anaphylaxis. Cardona et al,[1] with permission.

symptoms, including additional organ systems and nonspecific presentations. ¶"Increased work of breathing" refers to age-defined tachypnea that is not brief or self-resolving, use of accessory muscles, retractions, nasal flaring, or grunting (infants). *AAAAI,* American Academy of Allergy, Asthma & Immunology; *AAP,* American Academy of Pediatrics; *ACAAI,* American College of Allergy Asthma and Immunology; *ASCIA,* Australasian Society of Clinical Immunology and Allergy; *CSACI,* Canadian Society of Allergy and Clinical Immunology; *EAACI,* European Academy of Allergy and Clinical Immunology (Dribin et al,[21] with permission).

0.01 mg/kg of body weight, to a maximum total dose of 0.5 mg This is equivalent to 0.5 mL of 1 mg/mL (1:1000)[a] epinephrine (adrenaline)	
Infants under 10 kg	0.01 mg/kg = 0.01 mL/kg of 1 mg/mL (1:1000)
Children ages 1–5 years	0.15 mg = 0.15 mL of 1 mg/mL (1:1000)
Children ages 6–12 years	0.3 mg = 0.3 mL of 1 mg/mL (1:1000)
Teenagers and adults	0.5 mg = 0.5 mL of 1 mg/mL (1:1000)

Table 2. Recommended doses for intramuscular epinephrine (adrenaline). *Used with permission from Cardona et al.[1] [a]1 mg/mL (1:1000) is recommended for intramuscular injections because this allows a more appropriate volume to be injected.*

Step	Focus	Key tools
1	Confirm anaphylaxis	Clinical criteria
2	Detailed history	Structured questionnaires
3	Confirm mast cell activation	Acute/baseline tryptase, urine mediators
4	Exclude common/hidden triggers	Specific IgE, skin tests, molecular diagnosis, challenges
5	Rule out mast cell/clonal disease & mimics	Tryptase, C-kit mutations, imaging, bone marrow
6	Re evaluate over time	Follow up, repeat testing

Table 3. Diagnostic workup of idiopathic anaphylaxis.

immunotherapy or desensitization, should be considered in some cases.

Factors that may influence the development and severity of anaphylaxis need to be assessed. Among endogenous factors, underlying mast-cell disease, uncontrolled asthma, or hormonal status should be considered. Exogenous factors (also known as cofactors) include exercise, infections, psychological burden, sleep deprivation, alcohol intake, and medications (eg, NSAIDs, beta-blockers, and angiotensin converting enzyme). Several therapeutic approaches have been implemented to avoid recurrence of reactions (eg, antihistamines, corticosteroids, omalizumab) despite the evidence is low, based on anecdotal cases or small series.[25]

UNMET NEEDS AND FUTURE DIRECTIONS

A number of unmet needs have been identified in anaphylaxis,[1,26] of which the following are the most relevant:

- The worldwide availability of epinephrine/adrenaline autoinjectors and/or new devices
- Universally accepted definition of anaphylaxis and clinical criteria
- Standardized and internationally accepted severity scoring system for anaphylaxis
- Further research on underlying mechanisms of anaphylaxis, potential biomarkers for diagnosis and phenotyping, and genetic determinants and the role of cofactors
- Education on anaphylaxis for professionals and the lay population, advocacy actions among regulators and policy makers, and alignment among all stakeholders
- Optimization of care pathways for patients at risk of anaphylaxis, including patient/caregiver education and training in all regions to improve global anaphylaxis management
- Implementation of digital health tools in innovative ways to aid in research and care of patients

The introduction of novel adrenaline delivery systems, such as intranasal formulations, will require updates of clinical guidelines for anaphylaxis management.

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Use of artificial intelligence (AI) and generative AI tools in the work
No use of these tools.

Abbreviations
IgE, immunoglobulin E; NSAID, nonsteroidal anti-inflammatory drugs

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