PART XIII | ALLERGY

Pediatric Allergy

Chapters 183–193

51 one-best-answer study questions with original source tables

Nelson Textbook of Pediatrics, 22nd edition, Volume 1

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A child with persistent eosinophilia has abdominal symptoms and recent travel to an endemic area. Which etiologic group belongs in the childhood-eosinophilia differential?

A. An uncomplicated viral upper-respiratory illness alone
B. Physiologic pubertal change
C. Iron-deficiency anemia alone
D. Tissue-invasive helminth infection
E. Isolated bacterial cystitis

ORIGINAL SOURCE TABLE | Table 183.1 | PDF p. 1390 | printed p. 1376

Table 183.1 Differential Diagnosis of Childhood Eosinophilia

PHYSIOLOGIC
Prematurity
Infants receiving hyperalimentation
Hereditary

INFECTIOUS
Parasitic (with tissue-invasive helminths, e.g., trichinosis, strongyloidiasis, pneumocystosis, filariasis, cysticercosis, cutaneous and visceral larva migrans, echinococcosis)
Bacterial (brucellosis, tularemia, cat-scratch disease, *Chlamydia*)
Fungal (histoplasmosis, blastomycosis, coccidioidomycosis, allergic bronchopulmonary aspergillosis)
Mycobacterial (tuberculosis, leprosy)
Viral (HIV-1, HTLV-1, hepatitis A, hepatitis B, hepatitis C, Epstein-Barr virus)

PULMONARY
Allergic (rhinitis, asthma)
Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)
Loeffler syndrome
Hypersensitivity pneumonitis
Eosinophilic pneumonia (chronic, acute)
Pulmonary interstitial eosinophilia

DERMATOLOGIC
Atopic dermatitis
Pemphigus
Dermatitis herpetiformis
Infantile eosinophilic pustular folliculitis
Eosinophilic fasciitis (Schulman syndrome)
Eosinophilic cellulitis (Wells syndrome)
Kimura disease (angiolymphoid hyperplasia with eosinophilia)

FIP1L1-PDGFRA, FIP1-like 1–platelet-derived growth factor receptor α; HTLV, human T-lymphotr

of peripheral blood eosinophilia. Increased numbers of eosinophils are s

An adolescent taking an antihistamine needs allergen-specific IgE evaluation without interrupting medication. Which test is less affected by the antihistamine?

A. Immediate wheal measurement after skin testing
B. Serum allergen-specific IgE testing
C. Skin prick testing
D. Intradermal skin testing
E. Histamine skin control

ORIGINAL SOURCE TABLE | Table 183.3 | PDF p. 1391 | printed p. 1377

Table 183.3 Determination of Allergen-Specific IgE by Skin Testing vs In Vitro Testing

VARIABLE	SKIN TEST*	sIgE ASSAY
Risk of allergic reaction	Yes (especially ID)	No
Relative sensitivity	High	High
Affected by antihistamines	Yes	No
Affected by corticosteroids	Usually not	No
Affected by extensive dermatitis or dermatographism	Yes	No
Broad selection of antigens	Fewer	Yes
Immediate results	Yes	No
Expensive	No	Yes
Lability of allergens	Yes	No
Results evident to patient	Yes	No

*Skin testing may be the prick test or intradermal (ID) injection.

A child with recurrent infections has a very high total IgE. Which nonallergic diagnosis listed in the table should be considered?

A. Isolated seasonal allergic rhinitis as the only possibility
B. Normal IgE variation as the required explanation
C. Acute appendicitis
D. Simple iron deficiency
E. DOCK8-associated immunodeficiency

ORIGINAL SOURCE TABLE | Table 183.2 | PDF p. 1391 | printed p. 1377

Table 183.2 Nonallergic Diseases Associated with Increased Serum IgE Concentrations

PARASITIC INFESTATIONS
Ascariasis
Capillariasis
Echinococcosis
Fascioliasis
Filariasis
Hookworm
Onchocerciasis
Malaria
Paragonimiasis
Schistosomiasis
Strongyloidiasis
Trichinosis
Visceral larva migrans

INFECTIONS
Allergic bronchopulmonary aspergillosis
Candidiasis, systemic
Coccidioidomycosis
Cytomegalovirus mononucleosis
HIV type 1 infections
Infectious mononucleosis (Epstein-Barr virus)
Leprosy
Pertussis
Viral respiratory infections

IMMUNODEFICIENCY
Autosomal dominant hyper-IgE syndrome (*STAT3* variants)
Autosomal recessive hyper-IgE syndrome (*DOCK8, TYK2* variants)
IgA deficiency, selective
Nezelof syndrome (cellular immunodeficiency with immunoglobulins)
Thymic hypoplasia (DiGeorge anomaly)
Wiskott-Aldrich syndrome

NEOPLASTIC DISEASES
Hodgkin disease
IgE myeloma
Bronchial carcinoma

OTHER DISEASES AND DISORDERS
Alopecia areata
Bone marrow transplantation
Burns
Cystic fibrosis
Dermatitis, chronic acral
Erythema nodosum, streptococcal infection
Guillain-Barré syndrome
Kawasaki disease
Liver disease
Medication related
Nephritis, drug-induced interstitial
Nephrotic syndrome
Pemphigus, bullous
Polyarteritis nodosa, infantile
Primary pulmonary hemosiderosis
Juvenile idiopathic arthritis

A toddler has unilateral foul-smelling nasal discharge without seasonal symptoms. Which nonallergic cause in the rhinitis table deserves assessment?

A. Food-protein enterocolitis
B. Allergic conjunctivitis
C. Nasal foreign body
D. Pollen-driven seasonal rhinitis
E. Perennial dust-mite allergy

ORIGINAL SOURCE TABLE | Table 184.1 | PDF p. 1395 | printed p. 1380

Table 184.1 Causes of Rhinitis

ALLERGIC RHINITIS
Seasonal
Perennial
Perennial with seasonal exacerbations
NONALLERGIC RHINITIS
Structural/Mechanical Factors
Deviated septum/septal wall anomalies
Hypertrophic turbinates
Adenoidal hypertrophy
Foreign bodies
Nasal tumors
Benign
Malignant
Choanal atresia
CSF leakage
Infectious
Acute infections
Chronic infections
Congenital syphilis
Inflammatory/Immunologic
Granulomatosis with polyangiitis
Sarcoidosis
Midline granuloma
Systemic lupus erythematosus
Sjögren syndrome
Nasal polyposis
Physiologic
Primary ciliary dyskinesia
Atrophic rhinitis
Hormonally induced
Hypothyroidism
Pregnancy
Oral contraceptives
Menstrual cycle
Exercise
Atrophic
Drug induced
Rhinitis medicamentosa
Oral contraceptives
Antihypertensive therapy
Aspirin
Nonsteroidal antiinflammatory drugs
Cocaine
Reflex induced
Gustatory rhinitis
Chemical or irritant induced
Posture reflexes
Nasal cycle
Environmental factors
Odors
Temperature (cold air)
Weather/barometric pressure
Occupational (irritants)
OTHER
Nonallergic rhinitis with eosinophilia syndrome
Perennial nonallergic rhinitis (vasomotor rhinitis)
Emotional factors

From Skoner DP. Allergic rhinitis: definition, epidemiology, pathophysiology, detection, and diagnosis. *J Allergy Clin Immunol*. 2001;108(1 Suppl);108:S2–S8.

A child with rhinorrhea predominant rhinitis asks about an intranasal drug that reduces secretions through anticholinergic activity. Which table-listed spray fits?

A. Ipratropium bromide
B. Azelastine
C. Cromolyn sodium
D. Oxymetazoline
E. Phenylephrine

ORIGINAL SOURCE TABLE | Table 184.5 | PDF p. 1399 | printed p. 1384

Table 184.5 Miscellaneous Intranasal Sprays

DRUG	INDICATIONS (I), MECHANISM(S) OF ACTION (M), AND DOSING	COMMENTS, CAUTIONS, ADVERSE EVENTS, AND MONITORING
Ipratropium bromide	*I:* Symptomatic relief of rhinorrhea *M:* Anticholinergic	Atrovent inhalation aerosol is contraindicated in patients with hypersensitivity to soy lecithin Safety and efficacy of use beyond 4 days in patients with the common cold have not been established *Adverse effects:* Epistaxis, nasal dryness, nausea
Atrovent nasal spray (0.06%)	Colds (symptomatic relief of rhinorrhea): 5-12 yr: 2 sprays in each nostril tid ≥12 yr and adults: 2 sprays in each nostril tid-qid	
Azelastine	*I:* Treatment of rhinorrhea, sneezing, and nasal pruritus *M:* Antagonism of histamine H_1 receptor	May cause drowsiness *Adverse effects:* Headache, somnolence, bitter taste
Astelin	6-12 yr: 1 spray bid >12 yr: 1-2 sprays bid	
Cromolyn sodium	*I:* AR *M:* Inhibition of mast cell degranulation	Not effective immediately; requires frequent administration
NasalCrom	>2 yr: 1 spray tid-qid; max 6 times daily	
Oxymetazoline Afrin Nostrilla	*I:* Symptomatic relief of nasal mucosal congestion *M:* Adrenergic agonist, vasoconstricting agent 0.05% solution: instill 2-3 sprays into each nostril bid; therapy should not exceed 3 days	Excessive dosage may cause profound CNS depression Use in excess of 3 days may result in severe rebound nasal congestion Do not repeat more than once a month Use with caution in patients with hyperthyroidism, heart disease, hypertension, or diabetes *Adverse effects:* Hypertension, palpitations, reflex bradycardia, nervousness, dizziness, insomnia, headache, CNS depression, convulsions, hallucinations, nausea, vomiting, mydriasis, elevated intraocular pressure, blurred vision
Phenylephrine	*I:* Symptomatic relief of nasal mucosal congestion *M:* Adrenergic, vasoconstricting agent	Use in excess of 3 days may result in severe rebound nasal congestion Do not repeat more than once a month 0.16% and 0.125% solutions are not commercially available *Adverse effects:* Reflex bradycardia, excitability, headache, anxiety, dizziness
Neo-Synephrine	2-6 yr: 1 drop every 2-4 hr of 0.125% solution as needed *Note:* Therapy should not exceed 3 continuous days 6-12 yr: 1-2 sprays or 1-2 drops every 4 hr of 0.25% solution as needed *Note:* Therapy should not exceed 3 continuous days >12 yr: 1-2 sprays or 1-2 drops every 4 hr of 0.25% to 0.5% solution as needed; 1% solution may be used in adults with extreme nasal congestion *Note:* Therapy should not exceed 3 continuous days	

bid, 2 times daily; CNS, central nervous system; tid, 3 times daily; qid, 4 times daily.

A preschooler has recurrent wheeze. Which history is a major early-childhood risk factor for persistent asthma in the table?

A. Female sex
B. High birthweight
C. Absence of atopic sensitization
D. Parental asthma
E. A single isolated cold

ORIGINAL SOURCE TABLE | Table 185.1 | PDF p. 1402 | printed p. 1387

Table 185.1 Early Childhood Risk Factors for Persistent Asthma

Parental asthma*

Allergy
- Atopic dermatitis (eczema)*
- Allergic rhinitis
- Food allergy
- Inhalant allergen sensitization*
- Food allergen sensitization

Severe lower respiratory tract infection
- Pneumonia
- Bronchiolitis requiring hospitalization

Wheezing apart from colds

Male sex

Low birthweight

Environmental tobacco smoke exposure

Reduced lung function at birth

Formula feeding rather than breastfeeding

*Major risk factors.

A preschooler wheezes only with common viral colds and has no early atopic disease. Which childhood pattern in the table most closely fits?

A. Exercise-induced laryngeal obstruction
B. Transient nonatopic wheezing
C. Persistent atopy-associated asthma
D. Asthma with declining lung function
E. Occupational asthma

ORIGINAL SOURCE TABLE | Table 185.2 | PDF p. 1403 | printed p. 1388

Table 185.2 Asthma Patterns in Childhood, Based on Natural History and Asthma Management

TRANSIENT NONATOPIC WHEEZING
Common in early preschool years
Recurrent cough/wheeze, primarily triggered by common respiratory viral infections
Usually resolves during the preschool and lower school years, without increased risk for asthma in later life
Reduced airflow at birth, suggestive of relatively narrow airways; AHR near birth; improves by school age

PERSISTENT ATOPY-ASSOCIATED ASTHMA
Begins in early preschool years
Associated with atopy in early preschool years
- Clinical (e.g., atopic dermatitis in infancy, allergic rhinitis, food allergy)
- Biologic (e.g., early inhalant allergen sensitization, increased serum IgE, increased blood eosinophils)
- Highest risk for persistence into later childhood and adulthood

Lung function abnormalities
- Those with onset before 3 yr of age acquire reduced airflow by school age
- Those with later onset of symptoms, or with later onset of allergen sensitization, are less likely to experience airflow limitation in childhood

ASTHMA WITH DECLINING LUNG FUNCTION
Children with asthma with progressive increase in airflow limitation
Associated with hyperinflation in childhood, male gender

ASTHMA MANAGEMENT TYPES
(From national and international asthma management guidelines)

*Severity Classification**
- Intrinsic disease severity while not taking asthma medications

Intermittent
Persistent
- Mild
- Moderate
- Severe

*Control Classification**
- Clinical assessment while asthma being managed and treated

Well controlled
Not well controlled
Very poorly controlled

Management Patterns
- *Easy-to-control:* well controlled with low levels of daily controller therapy
- *Difficult-to-control:* inadequately controlled with multiple and/or high levels of controller therapies
- *Frequent exacerbators:* have severe exacerbations
- *Refractory:* continue to have poorly controlled asthma despite multiple and high levels of controller therapies

*From National Asthma Education and Prevention Program Expert Panel Report 3 (EPR3): *Guideline for the Diagnosis and Management of Asthma,* NIH Pub No 07-4051, Bethesda, MD, 2007, US Department of Health and Human Services; National Institutes of Health; National Heart, Lung, and Blood Institute Health; National Heart,

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A child with sensitized asthma has recurrent symptoms after exposure to cockroaches at home. Which trigger category in the table applies?

A. A nonallergic food intolerance
B. Exercise alone
C. Cold water immersion
D. Medication withdrawal
E. Indoor aeroallergen exposure

ORIGINAL SOURCE TABLE | Table 185.3 | PDF p. 1403 | printed p. 1388

Table 185.3 Asthma Triggers

COMMON VIRAL INFECTIONS OF THE RESPIRATORY TRACT

AEROALLERGENS IN SENSITIZED ASTHMATIC PATIENTS

Indoor Allergens
- Animal dander
- Dust mites
- Cockroaches
- Molds

Seasonal Aeroallergens
- Pollens (trees, grasses, weeds)
- Seasonal molds

AIR POLLUTANTS
- Environmental tobacco smoke
- Ozone
- Nitrogen dioxide
- Sulfur dioxide
- Particulate matter
- Wood- or coal-burning smoke
- Mycotoxins
- Endotoxin
- Dust

STRONG OR NOXIOUS ODORS OR FUMES
- Perfumes, hairsprays
- Cleaning agents

OCCUPATIONAL EXPOSURES
- Farm and barn exposures
- Formaldehydes, cedar, paint fumes
- Rhinitis
- Sinusitis
- Gastroesophageal reflux

DRUGS
- Aspirin and other nonsteroidal antiinflammatory drugs
- β-Blocking agents

OTHER
- Cold dry air
- Exercise
- Crying, laughter, hyperventilation
- Comorbid conditions

and difficulty keeping up with peers in physical activities. Asking about

A child with acute asthma is breathless at rest, prefers sitting, and speaks in phrases. Which exacerbation category best matches the table?

A. Anaphylaxis by definition
B. Intermittent stable asthma
C. Moderate exacerbation
D. Mild exacerbation
E. No exacerbation

ORIGINAL SOURCE TABLE | Table 185.4 | PDF p. 1404 | printed p. 1389

Table 185.4 Formal Evaluation of Asthma Exacerbation Severity in the Urgent or Emergency Care Setting*

	MILD	MODERATE	SEVERE	SUBSET: RESPIRATORY ARREST IMMINENT
SYMPTOMS				
Breathlessness	While walking Can lie down	While at rest (infant: softer, shorter cry, difficulty feeding) Prefers sitting	While at rest (infant: stops feeding) Sits upright	Extreme dyspnea Anxiety Upright, leaning forward
Talks in…	Sentences	Phrases	Words	Unable to talk
Alertness	May be agitated	Usually agitated	Usually agitated	Drowsy or confused
SIGNS				
Respiratory rate†	Increased	Increased	Often >30 breaths/min	
Use of accessory muscles; suprasternal retractions	Usually not	Commonly	Usually	Paradoxical thoracoabdominal movement
Wheeze	Moderate; often only end-expiratory	Loud; throughout exhalation	Usually loud; throughout inhalation and exhalation	Absence of wheeze
Pulse rate (beats/min)‡	<100	100-120	>120	Bradycardia
Pulsus paradoxus	Absent <10 mm Hg	May be present 10-25 mm Hg	Often present >25 mm Hg (adult) 20-40 mm Hg (child)	Absence suggests respiratory muscle fatigue
FUNCTIONAL ASSESSMENT				
Peak expiratory flow (value predicted or personal best)	≥70%	Approx. 40–69% or response lasts <2 hr	<40%	<25%§
Pa_{O_2} (breathing air)	Normal (test not usually necessary)	≥60 mm Hg (test not usually necessary)	<60 mm Hg; possible cyanosis	
and/or				
P_{CO_2}	<42 mm Hg (test not usually necessary)	<42 mm Hg (test not usually necessary)	≥42 mm Hg; possible respiratory failure	
Sa_{O_2} (breathing air) at sea level	>95% (test not usually necessary)	90–95% (test not usually necessary)	<90%	Hypoxia despite oxygen therapy
	Hypercapnia (hypoventilation) develops more readily in young children than in adults and adolescents.			

*Notes:
- The presence of several parameters, but not necessarily all, indicates the general classification of the exacerbation.
- Many of these parameters have not been systematically studied, especially as they correlate with each other; thus they serve only as general guides.
- The emotional impact of asthma symptoms on the patient and family is variable but must be recognized and addressed and can affect approaches to treatment and follow-up.

A toddler with recurrent unilateral wheeze after a choking episode has been labeled as having asthma. Which masquerader listed in the differential should be investigated?

A. Foreign body aspiration
B. Seasonal allergic rhinitis alone
C. Exercise-induced bronchospasm
D. Atopic dermatitis
E. Food-protein enterocolitis

ORIGINAL SOURCE TABLE | Table 185.5 | PDF p. 1405 | printed p. 1390

Table 185.5 Differential Diagnosis of Childhood Asthma

UPPER RESPIRATORY TRACT CONDITIONS

Allergic rhinitis*
Chronic rhinitis*
Sinusitis*
Adenoidal or tonsillar hypertrophy
Nasal foreign body

LARGE/CENTRAL AIRWAYS CONDITIONS

Laryngotracheobronchomalacia*
Laryngotracheobronchitis (e.g., pertussis)*
Laryngeal web, cyst, or stenosis
Exercise-induced laryngeal obstruction
Vocal cord dysfunction*
Vocal cord paralysis
Tracheoesophageal fistula
Vascular ring, sling, or external mass compressing on the airway (e.g., tumor)
Endobronchial or mediastinal tumor
Foreign body aspiration*
Chronic bronchitis from environmental tobacco smoke exposure*
Repaired tracheoesophageal fistula
Toxic inhalations

PERIPHERAL AIRWAYS CONDITIONS

Bronchopulmonary dysplasia (chronic lung disease of preterm infants)
Viral bronchiolitis*
Gastroesophageal reflux*
Causes of bronchiectasis
- Cystic fibrosis
- Immunodeficiency
- Allergic bronchopulmonary mycoses (e.g., aspergillosis)
- Chronic aspiration

Primary ciliary dyskinesia
Bronchiolitis obliterans
Interstitial lung diseases
Hypersensitivity pneumonitis (acute or chronic)
Loeffler syndrome
Eosinophilic granulomatosis with angiitis
Eosinophilic pneumonia
Tropical pulmonary eosinophilia
Pulmonary hemosiderosis
Tuberculosis
Pneumonia
Pulmonary edema (e.g., congestive heart failure)
Pulmonary vascular congestions (congenital or acquired heart disease)
Vasculitis
Sarcoidosis
Visceral larva migrans
Medications associated with chronic cough
- Acetylcholinesterase inhibitors
- β-Adrenergic antagonists
- Angiotensin-converting enzyme inhibitors
- Daptomycin

*More common asthma masqueraders.

An adolescent athlete develops throat tightness and inspiratory stridor during exercise with little bronchodilator response. Which diagnosis fits the distinguishing-features table?

A. Bacterial pneumonia
B. Chronic bronchiectasis
C. Pulmonary embolism
D. Paradoxical vocal cord motion
E. Classic lower-airway asthma

ORIGINAL SOURCE TABLE | Table 185.6 | PDF p. 1405 | printed p. 1390

Table 185.6 Features Distinguishing Paradoxical Vocal Cord Motion Disorder from Asthma

FEATURE	PVCM	ASTHMA
Incidence	Less common	More common
Age and sex	Young, female	Any
Triggers	Usually exercise or emotional stress	Many triggers
History of allergy	Usually absent	May be present
Family history	Usually absent	May be present
Sensation of tightness	Throat	Chest
Inspiratory stridor	More common, heard loudly over larynx	Rare
Sputum production	Rare	Common
Nocturnal awakening with symptoms	Rare	Common
Response to bronchodilators and steroids	No response	Good response
Hypoxemia	Rare	Common
Eosinophilia	Rare	Common in allergic asthma
Chest radiograph	Usually normal	May show hyperinflation and peribronchial thickening
Residual volume and total lung capacity	Normal	May be increased
Flow volume loop	Flattening of inspiratory loop	Obstructive pattern
Bronchial provocation test	May be positive	Usually positive
Laryngoscopy	Inspiratory adduction of the anterior two thirds of vocal folds with posterior chink	Usually normal

PVCM, Paradoxical vocal cord motion.
Modified from Ibrahim WH, Gheriani HA, Almohamed AA, Raza T. Paradoxical vocal cord motion disorder: Past present and future. *Post Grad Med J*. 2007;83:164–172. Table 2, p 168.

A child with episodic wheeze has spirometry below the age-specific lower limit for FEV1/FVC, followed by improvement after a short-acting bronchodilator. What does this pattern support?

A. An isolated oxygen-diffusion defect
B. Variable airflow limitation consistent with asthma
C. Fixed upper-airway obstruction as the only diagnosis
D. Normal lung function by definition
E. Primary restrictive lung disease alone

ORIGINAL SOURCE TABLE | Table 185.7 | PDF p. 1406 | printed p. 1391

Table 185.7 Lung Function Abnormalities in Asthma

Spirometry (in clinic)*†:

Airflow limitation

- Low FEV_1 (relative to percentage of predicted norms), although many children with asthma have normal FEV_1
- FEV_1/FVC ratio below the lower limit of normal for age

Bronchodilator response (to inhaled β-agonist) assesses *reversibility* of airflow limitation

Reversibility is determined by an increase in either FEV_1 >9–12% or predicted FEV_1 >10% after inhalation of a short-acting β-agonist (SABA)‡

Exercise challenge

- Worsening in FEV_1 ≥15%‡

Daily peak expiratory flow (PEF)* or FEV_1 monitoring: day-to-day and/or am-to-pm variation ≥20%‡

*PEF variability is insensitive, while being highly specific for asthma.
†Of note, >50% of children with mild to moderate asthma will have a normal FEV_1 and will not have a significant bronchodilator response.
‡Main criteria consistent with asthma.

A 10-year-old with suspected asthma has a low fractional exhaled nitric oxide after recent inhaled corticosteroid use. Which interpretation accords with the table?

A. Low FeNO definitively excludes all asthma phenotypes
B. Low FeNO confirms eosinophilic inflammation
C. FeNO is independent of anti-inflammatory therapy
D. Low FeNO proves bacterial infection
E. Recent corticosteroid use can lower FeNO

ORIGINAL SOURCE TABLE | Table 185.8 | PDF p. 1407 | printed p. 1392

Table 185.8 Interpretations of FeNO Test Results for Asthma Diagnosis in Nonsmoking Individuals Not Taking Corticosteroids

FeNO LEVEL		
<25 PPB (<20 IN CHILDREN AGE 5-12 YR)	**25-50 PPB (20-35 IN CHILDREN AGE 5-12 YR)**	**>50 PPB (>35 IN CHILDREN AGE 5-12 YR)**
• Recent or current corticosteroid use • Alternative diagnoses • Phenotype less likely to benefit from ICS • Noneosinophilic asthma • COPD • Bronchiectasis • CF • Vocal cord dysfunction • Rhinosinusitis • Smoking • Obesity	• Evaluate in clinical context • Consider other diagnoses • Consider other factors influencing result • Eosinophilic asthma less likely	• Eosinophilic airways inflammation likely • Phenotype more likely to respond to ICS • Allergic asthma • Eosinophilic bronchitis

CF, Cystic fibrosis; COPD, chronic obstructive pulmonary disease; FeNO, fractional exhaled nitric oxide; ICS, inhaled corticosteroid; ppb, parts per billion.
From NAEPPCC Expert Panel Working Group: 2020 Focused Updates to the Asthma Management Guidelines: A Report from the National Asthma Education and Prevention Program Coordinating Committee Expert Panel Working Group. *J Allergy Clin Immunol*. 2020;146(6):1217–1269. Table II.

A child aged 9 years has daytime symptoms twice weekly, no activity limitation, and no frequent steroid-requiring exacerbations. Which impairment category fits the severity table?

A. Severe persistent asthma
B. Status asthmaticus
C. Intermittent asthma
D. Mild persistent asthma
E. Moderate persistent asthma

ORIGINAL SOURCE TABLE | Table 185.9 | PDF p. 1409 | printed p. 1394

Table 185.9 Assessing Asthma Severity*

	CLASSIFICATION OF ASTHMA SEVERITY			
		PERSISTENT		
	INTERMITTENT	MILD	MODERATE	SEVERE
COMPONENTS OF SEVERITY				
Impairment				
Daytime symptoms	≤2 days/wk	>2 days/wk but not daily	Daily	Throughout the day
Nighttime awakenings				
Age 0-4 yr	0	1-2×/mo	3-4×/mo	>1×/wk
Age ≥5 yr	≤2×/mo	3-4×/mo	>1×/wk but not nightly	Often 7×/wk
Short-acting β_2-agonist use for symptoms (not for EIB prevention)	≤2 days/wk	>2 days/wk but not daily, and not more than 1× on any day	Daily	Several times per day
Interference with normal activity	None	Minor limitation	Some limitation	Extreme limitation
Lung function				
FEV_1 % predicted, age ≥5 yr	Normal FEV_1 between exacerbations >80% predicted	≥80% predicted	60–80% predicted	<60% predicted
FEV_1/FVC ratio†:				
Age 5-11 yr	>85%	>80%	75–80%	<75%
Age ≥12 yr	Normal	Normal	Reduced 5%	Reduced >5%
Risk				
Exacerbations requiring systemic corticosteroids				
Age 0-4 yr	0-1/yr (see notes)	≥2 exacerbations in 6 mo requiring systemic CS *or* ≥4 wheezing episodes/yr lasting >1 day *and* risk factors for persistent asthma		
Age ≥5 yr	0-1/yr (see notes)	≥2/yr (see notes)	≥2/yr (see notes)	≥2/yr (see notes)
Consider severity and interval since last exacerbation. Frequency and severity may fluctuate over time for patients in any severity category. Relative annual risk of exacerbations may be related to FEV_1.				

*Notes:
• Level of severity is determined by both impairment and risk. Assess impairment domain by patient's/caregiver's recall of previous 2-4 wk. Symptom assessment for longer periods should reflect a global assessment, such as inquiring whether a patient's asthma is better or worse since the last visit. Assign severity to the most severe category in which any feature occurs.

A child with treated asthma has symptoms four days weekly and frequent nighttime awakening. What does the control table support?

A. Assess as not well controlled and consider treatment adjustment
B. Label as well controlled solely because medication is prescribed
C. Stop controller therapy immediately
D. Ignore symptom frequency if spirometry is normal
E. Use a single IgE value as the only control measure

ORIGINAL SOURCE TABLE | Table 185.10 | PDF p. 1410 | printed p. 1395

Table 185.10 Assessing Asthma Control and Adjusting Therapy in Children*

	CLASSIFICATION OF ASTHMA CONTROL		
	WELL CONTROLLED	**NOT WELL CONTROLLED**	**VERY POORLY CONTROLLE**
COMPONENTS OF CONTROL			
Impairment			
Symptoms	≤2 days/wk but not more than once on each day	>2 days/wk or multiple times on ≤2 days/wk	Throughout the day
Nighttime awakenings:			
Age 0-4 yr	≤1×/mo	>1×/mo	>1×/wk
Age 5-11 yr	≤1×/mo	≥2×/mo	≥2×/wk
Age ≥12 yr	≤2×/mo	1-3×/wk	≥4×/wk
Short-acting β_2-agonist use for symptoms (not for EIB pretreatment)	≤2 days/wk	>2 days/wk	Several times per day
Interference with normal activity	None	Some limitation	Extremely limited
Lung function:			
Age 5-11 yr:			
FEV_1 (% predicted or peak flow)	>80% predicted or personal best	60–80% predicted or personal best	<60% predicted or personal bes
FEV_1/FVC:	>80%	75–80%	<75%
Age ≥12 yr:			
FEV_1 (% predicted or peak flow)	>80% predicted or personal best	60–80% predicted or personal best	<60% predicted or personal bes
Validated questionnaires†:			
Age ≥12 yr:			
ATAQ	0	1-2	3-4
ACQ	≤0.75	≤1.5	N/A
ACT	≥20	16-19	≤15
Risk			
Exacerbations requiring systemic corticosteroids:			
Age 0-4 yr	0-1/yr	2-3/yr	>3/yr
Age ≥5 yr	0-1/yr	≥2/yr (see notes)	
Consider severity and interval since last exacerbation.			
Treatment-related adverse effects	Medication side effects can vary in intensity from none to very troublesome and worrisome; the level of intensity does not correlate to specific levels of control but should be considered in the overall assessment of risk		
Reduction in lung growth *or* progressive loss of lung function	Evaluation requires long-term follow-up care		
RECOMMENDED ACTION FOR TREATMENT			
	Maintain current step Regular follow-up every 1-6 mo to maintain control Consider step down if well controlled for at least 3 mo	Step up† (1 step) and reevaluate in 2-6 wk If no clear benefit in 4-6 wk, consider alternative diagnoses or adjusting therapy For side effects, consider alternative options	Consider short course of oral corticosteroids Step up§ (1-2 steps) and reevaluat in 2 wk If no clear benefit in 4-6 wk, consider alternative diagnoses or adjusting therapy For side effects, consider alternat options

*Notes:
- The stepwise approach is meant to assist, not replace, the clinical decision-making required to meet individual patient needs.
- The level of control is based on the most severe impairment or risk category. Assess impairment domain by caregiver's recall of previous 2-4 wk. Symptom assessr for longer periods should reflect a global assessment, such as inquiring whether the patient's asthma is better or worse since the last visit.

At an asthma follow-up, a child reports taking a controller but continues to have exacerbations. Which clinic action appears among the key elements table?

A. Limit the visit to total serum IgE
B. Avoid a written action plan
C. Defer assessment of environmental exposures indefinitely
D. Demonstrate inhaler technique and have the child show it back
E. Assume inhaler technique is correct without assessment

ORIGINAL SOURCE TABLE | Table 185.11 | PDF p. 1411 | printed p. 1396

Table 185.11 Key Elements of Productive Clinic Visits for Asthma

Standardize assessment of asthma control (e.g., Asthma Control Test, exacerbations in past 12 mo)

Specify goals of asthma management

Explain basic facts about asthma

- Contrast normal vs asthmatic airways
- Link airways inflammation, "twitchiness," and bronchoconstriction
- Long-term-control and quick-relief medications
- Address concerns about potential adverse effects of asthma pharmacotherapy

Teach, demonstrate, and have patient show proper technique

- Inhaled medication use (spacer use with metered-dose inhaler)

Investigate and manage factors that contribute to asthma severity

- Environmental exposures
- Comorbid conditions

Create written two-part Asthma Action Plan (see Fig. 185.5)

- Daily management
- Action plan for asthma exacerbations

Regular follow-up visits

- Twice yearly (more often if asthma not well controlled)
- Monitor lung function at least annually

A child with sensitized asthma has persistent symptoms and tobacco smoke exposure in the home. Which contributing factor does the table prioritize?

A. Ignore indoor environmental history
B. Reduce environmental tobacco smoke exposure
C. Discontinue all physical activity indefinitely
D. Eliminate every food regardless of allergy assessment
E. Rely only on increasing rescue bronchodilator use

ORIGINAL SOURCE TABLE | Table 185.12 | PDF p. 1413 | printed p. 1398

Table 185.12 Control of Factors Contributing to Asthma Severity

ELIMINATE OR REDUCE PROBLEMATIC ENVIRONMENTAL EXPOSURES
Environmental tobacco smoke elimination or reduction in home and automobiles
Allergen exposure elimination or reduction in sensitized asthmatic patients
• Pets (cats, dogs, rodents, birds)
• Pests (mice, rats)
• Dust mites
• Cockroaches
• Molds
Other airway irritants
• Wood- or coal-burning smoke
• Strong chemical odors and perfumes (e.g., household cleaners)
• Dusts
TREAT COMORBID CONDITIONS
Rhinitis
Sinusitis
Gastroesophageal reflux

particulates as tobacco smoke, and should also be avoided (see Chapter

An adolescent with asthma has required chronic systemic corticosteroids for more than a month. Which adverse-effect surveillance category does the risk table assign?

A. Low risk
B. No risk
C. Only medication-free follow-up
D. Mild transient risk unrelated to cumulative exposure
E. High risk

ORIGINAL SOURCE TABLE | Table 185.16 | PDF p. 1418 | printed p. 1403

Table 185.16 Risk Assessment for Corticosteroid Adverse Effects

	CONDITIONS	RECOMMENDATIONS
Low risk	(≤1 risk factor*) Low- to medium-dose ICS (see Table 185.13)	Monitor blood pressure and weight with each physician visit Measure height annually (stadiometry); monitor periodically for declining growth rate and pubertal developmental delay Encourage regular physical exercise Ensure adequate dietary calcium and vitamin D with additional supplemer for daily calcium if needed. Avoid smoking and alcohol Ensure TSH status if patient has history of thyroid abnormality
Medium risk	(If >1 risk factor,* consider evaluating as high risk) High-dose ICS (see Table 185.13) At least four courses of OCS per year	As above, *plus*: Yearly ophthalmologic evaluations to monitor for cataracts or glaucoma Baseline bone densitometry (DEXA scan) Consider patient at increased risk for adrenal insufficiency, especially with physiologic stressors (e.g., surgery, accident, significant illness)
High risk	Chronic systemic corticosteroids (>7.5 mg daily or equivalent for >1 mo) ≥7 OCS burst treatments per year Very-high-dose ICS (e.g., fluticasone propionate ≥800 μg/day)	As above, *plus*: DEXA scan: if DEXA *z* score ≤1.0, recommend close monitoring (every 12 mo) Consider referral to a bone or endocrine specialist Bone age assessment Complete blood count Serum calcium, phosphorus, and alkaline phosphatase determinations Urine calcium and creatinine measurements Measurements of testosterone in males, estradiol in amenorrheic premenopausal women, vitamin D (25-OH and 1,25-OH vitamin D), parathyroid hormone, and osteocalcin Urine telopeptides for those receiving long-term systemic or frequent OC treatment Assume adrenal insufficiency for physiologic stressors (e.g., surgery, accident, significant illness)

*Risk factors for osteoporosis: presence of other chronic illness(es), medications (corticosteroids, anticonvulsants, heparin, diuretics), low body weight, family history of osteoporo significant fracture history disproportionate to trauma, recurrent falls, impaired vision, low dietary calcium and vitamin D intake, and lifestyle factors (decreased physical activity, smoking, alcohol intake).

DEXA, Dual-energy x-ray absorptiometry; ICS, inhaled corticosteroid; OCS, oral corticosteroid; TSH, thyroid-stimulating hormone.

A teenager with asthma previously required intensive care and intubation. Which future-risk category in the morbidity table is relevant?

A. Only a minor allergic-rhinitis symptom
B. A diagnostic criterion for food allergy
C. Biologic risk for severe morbidity and mortality
D. A reassuring marker of low future risk
E. Only an economic risk factor

ORIGINAL SOURCE TABLE | Table 185.18 | PDF p. 1423 | printed p. 1408

Table 185.18 Risk Factors for Asthma Morbidity and Mortality

BIOLOGIC
Previous severe asthma exacerbation (intensive care unit admission, intubation for asthma)
Sudden asphyxia episodes (respiratory failure, arrest)
Two or more hospitalizations for asthma in past year
Three or more emergency department visits for asthma in past year
Increasing and large diurnal variation in peak flows
Use of >2 canisters of short-acting β-agonists per month
Poor response to systemic corticosteroid therapy
Male sex
Low birthweight
Non-White
Sensitivity to *Alternaria*
ENVIRONMENTAL
Allergen exposure
Environmental tobacco smoke exposure
Air pollution exposure
Urban environment
ECONOMIC AND PSYCHOSOCIAL
Poverty
Crowding
Mother <20 yr old
Mother with less than high school education
Inadequate medical care
Inaccessible
Unaffordable
No regular medical care (only emergency)
Lack of written Asthma Action Plan
No care sought for chronic asthma symptoms
Delay in care of asthma exacerbations
Inadequate hospital care for asthma exacerbation
Psychopathology in the parent or child
Poor perception of asthma symptoms or severity
Alcohol or substance abuse

An infant has itchy, relapsing eczema on the face and extensor surfaces. Which diagnosis is supported by the major-feature table?

A. Atopic dermatitis
B. Urticarial vasculitis
C. Hereditary angioedema
D. Acute anaphylaxis
E. Isolated allergic rhinitis

ORIGINAL SOURCE TABLE | Table 186.1 | PDF p. 1429 | printed p. 1413

Table 186.1 Clinical Features of Atopic Dermatitis

MAJOR FEATURES
Pruritus
Facial and extensor eczema in infants and children
Flexural eczema in adolescents
Chronic or relapsing dermatitis
Personal or family history of atopic disease
ASSOCIATED FEATURES
Xerosis
Cutaneous infections (*Staphylococcus aureus*, group A streptococcus, herpes simplex, coxsackievirus, vaccinia, molluscum, warts)
Nonspecific dermatitis of the hands or feet
Ichthyosis, palmar hyperlinearity, keratosis pilaris
Nipple eczema
White dermatographism and delayed blanch response
Anterior subcapsular cataracts, keratoconus
Elevated serum IgE levels
Positive results of immediate-type allergy skin tests
Early age at onset
Dennie lines (Dennie-Morgan infraorbital folds)
Facial erythema or pallor
Course influenced by environmental and/or emotional factors
Lichenification
Perioral, periauricular sites
Prurigo

therapy. Wet dressing therapy can be complicated by maceration and

A toddler with a presumed eczema flare has intense nocturnal itch and household contacts with similar symptoms. Which table-listed alternative diagnosis should be evaluated?

A. Hereditary angioedema
B. Asthma
C. Vasovagal syncope
D. Scabies
E. Seasonal allergic conjunctivitis

ORIGINAL SOURCE TABLE | Table 186.2 | PDF p. 1430 | printed p. 1414

Table 186.2 Differential Diagnosis of Atopic Dermatitis

	MAIN AGE GROUP AFFECTED	FREQUENCY*	CHARACTERISTICS AND CLINICAL FEATURES
OTHER TYPES OF DERMATITIS			
Seborrheic dermatitis	Infants	Common	Salmon-red greasy scaly lesions, often on the scalp (cradle cap) and napkin area; generally presents in the first 6 wk of life; typically clears within weeks
Seborrheic dermatitis	Adults	Common	Erythematous patches with yellow, white, or grayish scales in seborrheic areas, particularly the scalp, central face, and anterior chest
Nummular dermatitis	Children and adults	Common	Coin-shaped scaly patches, mostly on legs and buttocks; usually no itch
Irritant contact dermatitis	Children and adults	Common	Acute to chronic eczematous lesions, mostly confined to the site of exposure; history of locally applied irritants is a risk factor; might coexist with AD
Allergic contact dermatitis	Children and adults	Common	Eczematous rash with maximum expression at sites of direct exposure but might spread; history of locally applied irritants is a risk factor; might coexist with AD
Lichen simplex chronicus	Adults	Uncommon	One or more localized, circumscribed, lichenified plaques that result from repetitive scratching or rubbing because of intense itch
Asteatotic eczema	Adults	Common	Scaly, fissured patches of dermatitis overlying dry skin, most often on lower legs
INFECTIOUS SKIN DISEASES			
Dermatophyte infection	Children and adults	Common	One or more demarcated scaly plaques with central clearing and slightly raised reddened edge; variable itch
Impetigo	Children	Common	Demarcated erythematous patches with blisters or honey-yellow crusting
Scabies	Children	Common†	Itchy superficial burrows and pustules on palms and soles, between fingers, and on genitalia; might produce secondary eczematous changes
HIV	Children and adults	Uncommon	Seborrhea-like rash
CONGENITAL IMMUNODEFICIENCIES (SEE TABLE 186.3)			
KERATINIZATION DISORDERS			
Ichthyosis vulgaris	Infants and adults	Uncommon	Dry skin with fine scaling, particularly on the lower abdomen and extensor areas; perifollicular skin roughening; palmar hyperlinearity; full form (i.e., 2 *FLG* pathogenic variants) is uncommon; often coexists with AD
NUTRITIONAL DEFICIENCY–METABOLIC DISORDERS			
Zinc deficiency (acrodermatitis enteropathica)	Children	Uncommon	Erythematous scaly patches and plaques, most often around the mouth and anus; rare congenital form accompanied by diarrhea and alopecia
Biotin deficiency (nutritional or biotinidase deficiency)	Infants	Uncommon	Scaly periorofacial dermatitis, alopecia, conjunctivitis, lethargy, hypotonia
Pellagra (niacin deficiency)	All ages	Uncommon	Scaly crusted epidermis, desquamation, sun-exposed areas, diarrhea
Kwashiorkor	Infants and children	Geographic dependent	Flaky scaly dermatitis, swollen limbs with cracked peeling patches
Phenylketonuria	Infants	Uncommon	Eczematous rash, hypopigmentation, blonde hair, developmental delay
NEOPLASTIC DISEASE			
Cutaneous T-cell lymphoma	Adults	Uncommon	Erythematous pink-brown macules and plaques with a fine scale; poorly responsive to topical corticosteroids; variable itch (in early stages)
Langerhans cell histiocytosis	Infants	Uncommon	Scaly and purpuric dermatosis, hepatosplenomegaly, cytopenias

*Common = approximately 1 in 10 to 1 in 100; uncommon = 1 in 100 to 1 in 1,000; rare = 1 in 1,000 to 1 in 10,000; very rare = <1 in 10,000.
†Especially in developing countries.
AD, Atopic dermatitis; FLG, filaggrin gene.

An infant with severe eczematous dermatitis also has thrombocytopenia and recurrent infections. Which associated immune deficiency is table-listed?

A. Familial cold autoinflammatory syndrome
B. Wiskott–Aldrich syndrome
C. Isolated terminal complement deficiency
D. C1-inhibitor deficiency
E. Selective lactase deficiency

ORIGINAL SOURCE TABLE | Table 186.3 | PDF p. 1431 | printed p. 1415

Table 186.3 Features of Primary Immunodeficiencies Associated with Eczematous Dermatitis

DISEASE	GENE	INHERITANCE	CLINICAL FEATURES	LAB ABNORMALITIES
AD-HIES	*STAT3*	AD, less commonly sporadic	Cold abscesses Recurrent sinopulmonary infections Mucocutaneous candidiasis Coarse facies Minimal trauma fractures Scoliosis Joint hyperextensibility Retained primary teeth Coronary artery tortuosity or dilation Lymphoma	High IgE (>2,000 IU/μL) Eosinophilia
DOCK8 deficiency	*DOCK8*	AR	Severe mucocutaneous viral infections Mucocutaneous candidiasis Atopic features (asthma, allergies) Squamous cell carcinoma Lymphoma	High IgE Eosinophilia With or without decreased IgM
PGM3 deficiency	*PGM3*	AR	Neurologic abnormalities Leukocytoclastic vasculitis Atopic features (asthma, allergies) Sinopulmonary infections Mucocutaneous viral infections	High IgE Eosinophilia
WAS	*WASP*	XLR	Hepatosplenomegaly Lymphadenopathy Atopic diathesis Autoimmune conditions (especially hemolytic anemia) Lymphoreticular malignancies	Thrombocytopenia (<80,000/μL) Low mean platelet volume Eosinophilia is common Lymphopenia Low IgM, variable IgG
SCID	Variable, depends on type	XLR and AR most common	Recurrent, severe infections Failure to thrive Persistent diarrhea Recalcitrant oral candidiasis Omenn syndrome: lymphadenopathy, hepatosplenomegaly, erythroderma	Lymphopenia common Variable patterns of reduced lymphocyte subsets (T, B, natur killer cells) Omenn syndrome: high lymphocytes, eosinophilia, high I
IPEX	*FOXP3*	XLR	Severe diarrhea (autoimmune enteropathy) Various autoimmune endocrinopathies (especially diabetes mellitus, thyroiditis) Food allergies	High IgE Eosinophilia Various autoantibodies
Netherton syndrome	*SPINK5*	AR	Hair shaft abnormalities Erythroderma Ichthyosis linearis circumflexa Food allergies Recurrent gastroenteritis Neonatal hypernatremic dehydration Upper and lower respiratory infections	High IgE Eosinophilia

AD, Autosomal dominant; AD-HIES, autosomal-dominant hyper-IgE syndrome; AR, autosomal recessive; *DOCK8*, dedicator of cytokinesis 8 gene; IPEX, immune dysregulation, polyendocrinopathy, enteropathy, X-linked syndrome; PGM3, phosphoglucomutase 3; SCID, severe combined immunodeficiency; WAS, Wiskott-Aldrich syndrome; XLR, X-linked reces
From Kliegman RM, Bordini BJ, eds. Undiagnosed and rare diseases in children. *Pediatr Clin N Amer*. 2017;64(1):41–42.

Parents of a child with atopic dermatitis ask whether to exclude multiple foods empirically to prevent flares. Which counseling principle does the table favor?

A. Eliminate all common allergens routinely
B. Stop breastfeeding whenever eczema occurs
C. Avoid all routine vaccines regardless of immune status
D. Permanently prohibit sports and exercise
E. Maintain a normal diet unless a specific food allergy is established

ORIGINAL SOURCE TABLE | Table 186.4 | PDF p. 1432 | printed p. 1416

Table 186.4 Counseling and Aggravating Factors for Patients with Atopic Dermatitis

Maintain cool temperature in bedroom, and avoid too many bed covers.
Increase emollient use with cold weather.
Avoid exposure to herpes sores; urgent visit if flare of unusual aspect.
Clothing: Avoid skin contact with irritating fibers (wool, large-fiber textiles).
Do not use tight and too-warm clothing to avoid excessive sweating.
New, nonirritating clothing designed for AD children is being evaluated.
Tobacco: Avoid exposure.
Vaccines: Normal schedule in noninvolved skin, including egg-allergic patients (see text).
Sun exposure: No specific restriction.
Usually helpful because of improvement of epidermal barrier.
Encourage summer holidays in altitude or at beach resorts.
Physical exercise, sports: No restriction.
If sweating induces flares of AD, progressive adaptation to exercise.
Shower and emollients after swimming pool.
Food allergens:
Maintain breastfeeding exclusively to 4-6 mo if possible.
Consider evaluation for early introduction of allergens (see Chapter 192).
Otherwise normal diet, unless an allergy workup has proved the need to exclude a specific food.
Indoor aeroallergens: House dust mites.
Use adequate ventilation of housing; keep the rooms well aerated even in winter.
Avoid wall-to-wall carpeting.
Remove dust with a wet sponge.
Vacuum floors and upholstery with an adequately filtered cleaner once a week.
Avoid soft toys in bed (cradle), except washable ones.
Wash bedsheets at a temperature higher than 55°C (131°F) every 10 days.
Use bed and pillow encasings made of Gore-Tex or similar material.
Furred pets: Advise to avoid. If allergy is demonstrated, be firm on avoidance measures, such as pet removal.
Pollen: Close windows during peak pollen season on warm and dry weather days and restrict, if possible, time outdoors.
Windows may be open at night and early in the morning or during rainy weather.
Avoid exposure to risk situations (lawn mowing).
Use pollen filters in motor vehicles.
Clothes and pets can vectorize aeroallergens, including pollen.

Adapted from Darsow U, Wollenberg A, Simon D, et al. ETFAD/EADV Eczema Task Force 2009 position paper on diagnosis and treatment of atopic dermatitis. *J Eur Acad Dermatol Venereol*. 2010;24:321.

A child has widespread dry skin, incessant itching, excoriation, oozing, and skin thickening. Which physical severity category applies?

A. Moderate eczema without extensive changes
B. No atopic dermatitis by definition
C. Severe eczema
D. Clear eczema
E. Mild eczema

ORIGINAL SOURCE TABLE | Table 186.5 | PDF p. 1433 | printed p. 1417

Table 186.5 Categorization of Physical Severity of Atopic Eczema

Clear: Normal skin, with no evidence of atopic eczema
Mild: Areas of dry skin, infrequent itching (with or without small areas of redness)
Moderate: Areas of dry skin, frequent itching, redness (with or without excoriation and localized skin thickening)
Severe: Widespread areas of dry skin, incessant itching, redness (with or without excoriation, extensive skin thickening, bleeding, oozing, cracking, and alteration of pigmentation)

From Lewis-Jones S, Mugglestone MA, Guideline Development Group. Management of atopic eczema in children aged up to 12 years: summary of NICE guidance. *BMJ.* 2007;335:1263–1264.

A child needs a low-potency topical corticosteroid for a sensitive area. Which agent is listed in the least-potent group?

A. Hydrocortisone
B. Clobetasol propionate
C. Fluocinonide 0.1%
D. Betamethasone dipropionate 0.05% ointment
E. Mometasone furoate 0.1% ointment

ORIGINAL SOURCE TABLE | Table 186.6 | PDF p. 1433 | printed p. 1417

Table 186.6 Selected Topical Corticosteroid Preparations*

GROUP 1
Clobetasol propionate (Temovate) 0.05% ointment/cream
Betamethasone dipropionate (Diprolene) 0.05% ointment/lotion/gel
Fluocinonide (Vanos) 0.1% cream

GROUP 2
Mometasone furoate (Elocon) 0.1% ointment
Halcinonide (Halog) 0.1% cream
Fluocinonide (Lidex) 0.05% ointment/cream
Desoximetasone (Topicort) 0.25% ointment/cream
Betamethasone dipropionate (Diprolene) 0.05% cream

GROUP 3
Fluticasone propionate (Cutivate) 0.005% ointment
Halcinonide (Halog) 0.1% ointment
Betamethasone valerate (Valisone) 0.1% ointment

GROUP 4
Mometasone furoate (Elocon) 0.1% cream
Triamcinolone acetonide (Kenalog) 0.1% ointment/cream
Fluocinolone acetonide (Synalar) 0.025% ointment

GROUP 5
Fluocinolone acetonide (Synalar) 0.025% cream
Hydrocortisone valerate (Westcort) 0.2% ointment

GROUP 6
Desonide (DesOwen) 05% ointment/cream/lotion
Alclometasone dipropionate (Aclovate) 0.05% ointment/cream

GROUP 7
Hydrocortisone (Hytone) 2.5%, 1%, 0.5% ointment/cream/lotion

*Representative corticosteroids are listed by group from 1 (superpotent) through 7 (least potent).

Adapted from Stoughton RB. Vasoconstrictor assay-specific applications. In: Malbach HI, Surber C, eds. *Topical Corticosteroids*. Basel, Switzerland: Karger; 1992. p 42–53.

A school-aged boy has chronic bilateral itchy red eyes with giant cobblestone papillae in a warm, dry climate. Which allergic eye condition in the comparison table is most likely?

A. Giant papillary conjunctivitis from a prosthesis
B. Isolated bacterial conjunctivitis
C. Hereditary angioedema
D. Vernal keratoconjunctivitis
E. Seasonal allergic conjunctivitis alone

ORIGINAL SOURCE TABLE | Table 188.1 | PDF p. 1438 | printed p. 1422

Table 188.1 Allergic Diseases of the Eye

DISEASE	CLINICAL PARAMETERS	SIGNS/SYMPTOMS	DIFFERENTIAL DIAGNOSIS
Seasonal allergic conjunctivitis (SAC)	• Sensitized individuals • Both females and males • Bilateral involvement • Seasonal allergens • Self-limiting	• Ocular itching • Tearing (watery discharge) • Chemosis, redness • Often associated with rhinitis • Not sight threatening	• Infective conjunctivitis • Preservative toxicity • Medicamentosa • Dry eye • PAC/AKC/VKC
Perennial allergic conjunctivitis (PAC)	• Sensitized individuals • Both females and males • Bilateral involvement • Year-round allergens • Self-limiting	• Ocular itching • Tearing (watery discharge) • Chemosis, redness • Often associated with rhinitis • Not sight threatening	• Infective conjunctivitis • Preservative toxicity • Medicamentosa • Dry eye • SAC/AKC/VKC
Atopic keratoconjunctivitis (AKC)	• Sensitized individuals • Peak incidence 20-50 years of age • Both females and males • Bilateral involvement • Seasonal/perennial allergens • Atopic dermatitis • Chronic symptoms	• Severe ocular itching • Red flaking periocular skin • Mucoid discharge, photophobia • Corneal erosions • Scarring of conjunctiva • Cataract (anterior subcapsular) • Sight threatening	• Contact dermatitis • Infective conjunctivitis • Blepharitis • Pemphigoid • VKC/SAC/PAC/GPC
Vernal keratoconjunctivitis (VKC)	• Some sensitized individuals • Peak incidence 3-20 years of age • Males predominate 3:1 • Bilateral involvement • Warm, dry climate • Seasonal/perennial allergens • Chronic symptoms	• Severe ocular itching • Severe photophobia • Thick, ropy discharge • Cobblestone papillae • Corneal ulceration and scarring • Sight threatening	• Infective conjunctivitis • Blepharitis • AKC/SAC/PAC/GPC
Giant papillary conjunctivitis (GPC)	• Sensitization not necessary • Both females and males • Bilateral involvement • Prosthetic exposure • Occurs anytime • Chronic symptoms	• Mild ocular itching • Mild mucoid discharge • Giant papillae • Contact lens intolerance • Foreign body sensation • Protein buildup on contact lens • Not sight threatening	• Infective conjunctivitis • Preservative toxicity • SAC/PAC/AKC/VKC

From Barney NP. Allergic and immunologic diseases of the eye. In: Burks AW, Holgate ST, O'Hehir RE, et al., eds. *Middleton's Allergy: Principles and Practice*. 9th ed. Philadelphia: Elsevier, 2020; Table 38.1, p. 607.

A child develops acute urticaria during a viral illness without a clear food exposure. Which etiologic group appears in the acute-urticaria table?

A. Only physical urticaria
B. Infections
C. Only inherited complement deficiency
D. Only food allergy
E. Only systemic autoimmune disease

ORIGINAL SOURCE TABLE | Table 189.1 | PDF p. 1442 | printed p. 1426

Table 189.1 Etiology of Acute Urticaria

Foods	Egg, milk, wheat, peanuts, tree nuts, soy, shellfish, fish, direct mast cell degranulation
Medications	Suspect all medications, even nonprescription or homeopathic
Insect stings	Hymenoptera (honeybee, yellow jacket, hornets, wasp, fire ants), biting insects (papular urticaria)
Infections	**Bacterial** (streptococcal pharyngitis, *Mycoplasma*, sinusitis); **Viral** (hepatitis, mononucleosis [Epstein-Barr virus], coxsackieviruses A and B); **Parasitic** (*Ascaris, Ancylostoma, Echinococcus, Fasciola, Filaria, Schistosoma, Strongyloides, Toxocara, Trichinella*); **Fungal** (dermatophytes, *Candida*)
Contact allergy	Latex, pollen, animal saliva, nettle plants, caterpillars
Transfusion reactions	Blood, blood products, or intravenous immune globulin administration

From Lasley MV, Kennedy MS, Altman LC. Urticaria and angioedema. In: Altman LC, Becker JW, Williams PV, eds. *Allergy in Primary Care*. Philadelphia: Saunders; 2000: p. 232.

An adolescent has recurring wheals for several months, reproducibly provoked by scratching the skin. Which chronic-urticaria category fits?

A. Acute food-triggered anaphylaxis
B. Hereditary angioedema without wheals
C. Serum sickness as the required diagnosis
D. Fixed drug eruption
E. Inducible dermographism

ORIGINAL SOURCE TABLE | Table 189.2 | PDF p. 1442 | printed p. 1426

Table 189.2 Etiology of Chronic Urticaria

Spontaneous/autoimmune	Approximately 30% of chronic urticaria cases are inducible urticaria, and 60–70% are spontaneous; of the spontaneous cases approximately 20–60% have autoantibodies (see text)
Inducible	Dermographism Cholinergic urticaria Cold urticaria (see Table 189.6) Delayed pressure urticaria Solar urticaria Vibratory urticaria Aquagenic urticaria
Autoimmune diseases	Systemic lupus erythematosus Juvenile idiopathic arthritis Thyroid disease (Graves, Hashimoto) Celiac disease Inflammatory bowel disease Urticarial vasculitis
Autoinflammatory/periodic fever syndromes	See Tables 189.3 and 189.6.
Neoplastic	Lymphoma Mastocytosis Leukemia
Angioedema	Hereditary angioedema Acquired angioedema Angiotensin-converting enzyme inhibitors

Modified from Lasley MV, Kennedy MS, Altman LC. Urticaria and angioedema. In: Altman LC, Becker JW, Williams PV, eds. *Allergy in Primary Care*. Philadelphia: Saunders; 2000: p. 234.

A child has recurrent cold-triggered urticarial rash, fever, and arthralgia. Which gene is listed for familial cold autoinflammatory syndrome?

A. FOXP3
B. C1-INH
C. NLRP3
D. BTK
E. ELANE

ORIGINAL SOURCE TABLE | Table 189.3 | PDF p. 1443 | printed p. 1427

Table 189.3 Febrile Autoinflammatory Diseases Causing Urticaria in Children

DISEASE	GENE (PROTEIN)	INHERITANCE	ATTACK LENGTH	TIMING OF ONSET	CUTANEOUS FEATURES	EXTRACUTANEOUS CLINICAL FEATURES
FCAS	*NLRP3* (cryopyrin)	AD	Brief; minutes to 3 days	Neonatal or infantile	Cold-induced pseudourticaria	Arthralgia Conjunctivitis Headache
Muckle-Wells syndrome	*NLRP3* (cryopyrin)	AD	1-3 days	Neonatal, infantile, childhood (can be later)	Widespread pseudourticaria	Arthralgia/arthritis Sensorineural hearing loss Conjunctivitis/episcleritis Headache Amyloidosis
Neonatal-onset multisystem inflammatory disease (aka chronic infantile neurologic cutaneous articular syndrome)	*NLRP3* (cryopyrin)	AD	Continuous flares	Neonatal or infantile	Widespread pseudo-urticaria	Deforming osteoarthropath epiphyseal overgrowth Sensorineural hearing loss Dysmorphic facies Chronic aseptic meningitis, headaches, papilledema, seizures Conjunctivitis/uveitis, optic atrophy Growth retardation Developmental delay Amyloidosis
HIDS	*MVK* (mevalonate kinase)	AR	3-7 days	Infancy (<2 yr)	Intermittent morbilliform or urticarial rash Aphthous mucosal ulcers Erythema nodosum	Arthralgia/arthritis Cervical lymphadenopathy Severe abdominal pain Diarrhea/vomiting Headache Elevated IgD and IgA antibo levels Elevated urine mevalonic a during attacks
Tumor necrosis factor receptor–associated periodic syndrome	*TNFRSF1A* (TNFR1)	AD	>7 days	Childhood	Intermittent migratory erythematous macules and edematous plaques overlying areas of myalgia, often on limbs Periorbital edema	Migratory myalgia Conjunctivitis Serositis Amyloidosis
Systemic-onset juvenile idiopathic arthritis (SoJIA)	Polygenic	Varies	Daily (quotidian)	Peak onset at 1-6 yr	Nonfixed erythematous rash; may be urticarial With or without dermographism With or without periorbital edema	Polyarthritis Myalgia Hepatosplenomegaly Lymphadenopathy Serositis
PLAID	*PLCG2*	AD	N/A	Infancy	Urticaria induced by evaporative cooling Ulcers in cold-exposed areas	Allergies Autoimmune disease Recurrent sinopulmonary infections Elevated IgE antibody level Decreased IgA and IgM antibody levels Often elevated antinuclear antibody titers

AD, Autosomal dominant; AR, autosomal recessive; HIDS, hyperimmunoglobulinemia D syndrome; FCAS, familial cold-induced autoinflammatory syndrome; N/A, not available; PLAID, PLCG2-associated antibody deficiency and immune dysregulation.
From Youseff MJ, Chiu YE. Eczema and urticaria as manifestations of undiagnosed and rare diseases. *Pediatr Clin North Am*. 2017;64:39–56. Table 2, pp. 49–50.

A patient has painful urticarial plaques lasting more than 36 hours and leaving bruising. Which interpretation is favored by the distinguishing-features table?

A. Evaluate for an urticarial syndrome such as vasculitis
B. Classify as ordinary transient urticaria without further assessment
C. Diagnose isolated dermographism from the duration alone
D. Assume uncomplicated acute food allergy
E. Conclude the lesions are normal skin variants

ORIGINAL SOURCE TABLE | Table 189.4 | PDF p. 1444 | printed p. 1428

Table 189.4 Distinguishing Features Between Urticaria and Systemic Urticarial Syndromes

COMMON URTICARIA	URTICARIAL SYNDROMES (≥1 OF FOLLOWING)
Only typical wheals:	Atypical "wheals":
Erythematous edematous lesions	Infiltrated plaques
Transient (<24-36 hr)	Persistent (>24-36 hr)
Asymmetric distribution	Symmetric distribution
Resolution without signs	Resolution with signs (hypo/hyperpigmentation, bruising, or scarring)
No associated different elementary lesions (papules, vesicles, purpura, crustae)	Associated different elementary lesions (papules, vesicles, purpura, scaling, crustae)
Pruritic (rarely stinging/burning)	Not pruritic; rather painful or burning
Possibly associated with angioedema	Usually no associated angioedema
No associated systemic symptoms	Often associated with systemic symptoms (fever, malaise, arthralgia, abdominal pain, weight loss, acral circulatory abnormalities, neurologic signs

From Peroni A, Colato C, Zanoni G, Girolomoni G. Urticarial lesions: if not urticaria, what else? The differential diagnosis of urticaria. *J Am Acad Dermatol*. 2009;62(4):559.

A child develops raised itchy wheals minutes after a clinician lightly strokes the skin. Which physical urticaria is being demonstrated?

A. Solar urticaria
B. Aquagenic urticaria
C. Cold urticaria
D. Dermographism
E. Delayed pressure urticaria

ORIGINAL SOURCE TABLE | Table 189.5 | PDF p. 1445 | printed p. 1429

Table 189.5 Comparison of the Physical Urticarias

URTICARIA	RELATIVE FREQUENCY	PRECIPITANT	TIME OF ONSET	DURATION	LOCAL SYMPTOMS	SYSTEMIC SYMPTOMS	TESTS	MECHANISM	TREATMENT
Symptomatic dermographism	Most frequent	Stroking skin	Minutes	2-3 hr	Irregular pruritic attacks	None	Stroke skin	Passive transfer, IgE, histamine, possible role of adenosine triphosphate, substance P, possible direct pharmacologic mechanism	Continual antihistamines
Delayed dermographism	Rare	Stroking skin	30 min to 8 hr	<48 hr	Burning, deep swelling	None	Stroke skin, observe early and late	Unknown	Avoidance of precipitants
Pressure urticaria	Frequent	Pressure	3-12 hr	8-24 hr	Diffuse, tender swelling	Flulike symptoms	Apply weight	Unknown	Avoidance of precipitants; if severe, omalizumab
Solar urticaria	Frequent	Various wavelengths of light	2-5 min	15 min to 3 hr	Pruritic wheals	Wheezing, dizziness, syncope	Phototest	Passive transfer, reverse passive transfer, IgE, possible histamine	Avoidance of precipitants; antihistamines, sunscreens, antimalarials
Familial cold urticaria	Rare	Change in skin temperature	30 min to 3 hr	<48 hr	Burning wheals	Tremor; headache; arthralgia; fever	Expose skin to cold air	Unknown	Avoidance of precipitants
Essential acquired cold urticaria	Frequent	Cold contact	2-5 min	1-2 hr	Pruritic wheals	Wheezing, syncope, drowning	Apply ice-filled copper beaker to arm, immerse	Passive transfer, reverse passive transfer, IgE (IgM), histamine; vasculitis can be induced	Cyproheptadine hydrochloride, other antihistamines; desensitization; avoidance of precipitants
Heat urticaria	Rare	Heat contact	2-5 min (rarely delayed)	1 hr	Pruritic wheals	None	Apply hot water–filled cylinder to arm	Possibly histamine; possibly complement	Antihistamines, desensitization; avoidance of precipitants
Cholinergic urticaria	Very frequent	General overheating of body	2-20 min	30 min to 1 hr	Papular, pruritic wheals	Syncope; diarrhea; vomiting, salivation; headaches	Bathe in hot water; exercise until perspiring, inject methacholine chloride	Passive transfer; possible immunoglobulin; product of sweat gland stimulation; histamine, reduced protease	Application of cold water or ice to skin; antihistamines; omalizumab
Aquagenic urticaria	Rare	Water contact	Several minutes	30-45 min	Papular, pruritic wheals	None reported	Apply water compresses to skin	Unknown	Avoidance of precipitants; antihistamine; application of inert oil
Vibratory angioedema	Very rare	Vibrating against skin	2-5 min	1 hr	Angioedema	None reported	Apply vibration to forearm	Unknown	Avoidance of precipitants

Ig, Immunoglobulin.

From Lee AD, Ierizzo JL. Urticaria. In: Callen JP, Ierizzo JL, Bolognia JL, et al., eds. Dermatological Signs of Internal Disease. 4th ed. Philadelphia: Elsevier; 2009: p. 59, Table 6.4.

An adolescent reports hives after evaporative cooling and recurrent sinopulmonary infections. Which hereditary cold-urticaria disorder is listed?

A. Uncomplicated allergic rhinitis
B. PLCG2-associated antibody deficiency and immune dysregulation (PLAID)
C. Classic isolated seasonal pollen allergy
D. X-linked agammaglobulinemia
E. Isolated hereditary angioedema

ORIGINAL SOURCE TABLE | Table 189.6 | PDF p. 1446 | printed p. 1430

Table 189.6 Hereditary Diseases with Cold-Induced Urticaria

		EPISODIC SYMPTOMS	SUSTAINED/PROGRESSIVE SYMPTOMS
CAPS	FCAS	Urticarial rash, arthralgia, myalgia, chills, fever, swelling of extremities	Renal amyloidosis
	MWS	Urticarial rash, arthralgia, chills, fever	Sensorineural deafness, renal amyloidosis
	CINCA	Fever	Rash, arthritis, chronic meningitis, visual defect, deafness, growth retardation, renal amyloidosis
NAPS12 (FCAS2)		Fever, arthralgia, myalgia, urticaria, abdominal pain, aphthous ulcers, lymphadenopathy	Sensorineural deafness
PLAID (FCAS3)		Urticaria induced by evaporative cooling, sinopulmonary infections	Serum low IgM and IgA levels; high IgE levels; decreased B and NK cells; granulomata; antinuclear antibodies

CAPS, Cryopyrin-associated periodic syndromes; FCAS, familial cold-induced autoinflammatory syndrome; MWS, Muckle-Wells syndrome; CINCA, chronic infantile neurologic cutaneous articular syndrome; NAPS, NLRP-12–associated periodic syndrome; PLAID, PLCG2-associated antibody deficiency and immune dysregulation; NK, natural killer; Ig, immunoglobulin.

From Kanazawa N. Hereditary disorders presenting with urticaria. *Immunol Allergy Clin N Amer*. 2014;34:169–179. Table 4, p. 176.

A child has episodic swelling without hives and a family history of similar attacks. Which laboratory evaluation appears for hereditary angioedema?

A. Total IgE alone
B. A single skin prick test to peanut
C. An isolated complete blood count alone
D. A bronchodilator response test
E. C4 and C1-inhibitor protein and function

ORIGINAL SOURCE TABLE | Table 189.7 | PDF p. 1447 | printed p. 1431

Table 189.7 Diagnostic Testing for Urticaria and Angioedema

DIAGNOSIS	DIAGNOSTIC TESTING
Chronic spontaneous urticaria	No testing required for diagnosis
Food and drug reactions	Elimination of offending agent, skin testing, and challenge with suspected foods
Autoimmune urticaria	Autologous serum skin test; antithyroid antibodies
Thyroiditis	Thyroid-stimulating hormone; antithyroid antibodies
Infections	Appropriate cultures or serology
Collagen vascular diseases and cutaneous vasculitis	Skin biopsy, CH_{50}, C1q, C4, immunofluorescence of tissues, antinuclear antibodies, cryoglobulins
Cold urticaria	Ice cube test usually positive but may be negative in familial autoinflammatory disorders
Solar urticaria	Exposure to defined wavelengths of light, red blood cell protoporphyrin, fecal protoporphyrin, and coproporphyrin
Dermographism	Stroking with narrow object (e.g., tongue blade)
Pressure urticaria	Application of pressure for defined time and intensity
Vibratory urticaria	Vibration for 4 min
Aquagenic urticaria	Challenge with tap water at various temperatures
Urticaria pigmentosa	Skin biopsy, test for dermographism
Hereditary angioedema	C4, C1-INH testing by protein and function
Familial cold autoinflammatory syndrome	Genetic testing for *NALP3* pathogenic variants

of anaphylactic shock. The combination of ingestion of various food allergens and postprandial exercise has been associated with urticaria/

A child has recurrent nonurticarial angioedema with low C4, low C1-inhibitor protein, and reduced function. Which table pattern fits?

A. Idiopathic angioedema with normal complement
B. Simple dermographism
C. Type 1 hereditary angioedema
D. Type 2 hereditary angioedema
E. Hereditary angioedema with normal C1 inhibitor

ORIGINAL SOURCE TABLE | Table 189.9 | PDF p. 1449 | printed p. 1433

Table 189.9 Complement Evaluation of Patients with Recurrent Angioedema

	C4	C1-INH LEVEL	C1-INH FUNCTION	C1q
Idiopathic angioedema	Normal	Normal	Normal	Normal
Type 1 HAE	Low	Low	Low	Normal
Type 2 HAE	Low	Normal	Low	Normal
HAE-nlC1-INH	Normal	Normal	Normal	Normal
Acquired C1-INH deficiency	Low	Low	Low	Low
Urticarial vasculitis	Low or normal	Normal	Normal	Low or normal

C1-INH, C1 inhibitor; HAE-nlC1-INH, hereditary angioedema with normal C1-INH; nl, normal.
From Joshi SR, Khan DA. Urticaria and angioedema. In: Leung DYM, Akdis CA, Bacharier LB, et al., eds. *Pediatric Allergy Principles and Practice*. 4th ed. Philadelphia: Elsevier; 20
Table 39.3, p. 338.

Minutes after eating a known allergen, a child develops generalized hives and wheezing. Which diagnosis meets the listed clinical criteria?

A. Anaphylaxis
B. Isolated urticaria without systemic involvement
C. A vasovagal episode
D. Chronic spontaneous urticaria
E. Serum sickness

ORIGINAL SOURCE TABLE | Table 190.1 | PDF p. 1450 | printed p. 1434

Table 190.1 Clinical Criteria for Diagnosing Anaphylaxis

Anaphylaxis is highly likely when any one of the following three 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 urticaria, itching or flushing, swollen lips-tongue-uvula)

And at least one of the following:

   A. Respiratory compromise (e.g., dyspnea, wheeze-bronchospasm, stridor, hypoxemia)
   B. Reduced blood pressure or associated symptoms of end-organ dysfunction (e.g., hypotonia [collapse], syncope, incontinence)

OR

2. Two or more of the following that occur rapidly after exposure to a **likely allergen** (or other trigger) for that patient (minutes to several hours)
   A. Involvement of the skin/mucosal tissue (e.g., generalized urticaria, itch-flush, swollen lips-tongue-uvula)
   B. Respiratory compromise (e.g., dyspnea, wheeze or bronchospasm, stridor, reduced peak expiratory flow, hypoxemia)
   C. Reduced blood pressure or associated symptoms (e.g., hypotonia [collapse], syncope, incontinence)
   D. Persistent gastrointestinal symptoms (e.g., crampy abdominal pain, vomiting)

OR

3. Reduced blood pressure after exposure to a **known allergen** or other trigger for that patient (minutes to hours).
   A. Infants and children: low systolic blood pressure (age-specific) or greater than 30% decrease in systolic blood pressure
   B. Adults: systolic blood pressure of less than 90 mm Hg or greater than 30% decrease from that person's baseline

From Sampson HA, Muñoz-Furlong A, Campbell RL, et al. Second symposium on the definition and management of anaphylaxis: summary report–Second National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network symposium. *J Allergy Clin Immunol*. 2006;117(2):391-7.

A child develops anaphylaxis after a yellow-jacket sting. Which community trigger category is listed?

A. Latex as the only possible cause
B. Food additive exposure as the required cause
C. An exercise trigger alone
D. Hymenoptera venom
E. Aeroallergen exposure as the necessary cause

ORIGINAL SOURCE TABLE | Table 190.2 | PDF p. 1451 | printed p. 1435

Table 190.2 Anaphylaxis Triggers in the Community*

ALLERGEN TRIGGERS (IgE-DEPENDENT IMMUNOLOGIC MECHANISM)*	OTHER IMMUNE MECHANISMS (IgE INDEPENDENT)
Foods (e.g., peanut, tree nuts, shellfish, fish, milk, egg, wheat, soy, sesame, meat [galactose-α-1,3-galactose])	IgG mediated (infliximab, high molecular weight dextrans)
Food additives (e.g., spices, colorants, vegetable gums, contaminants)	Immune aggregates (IVIG)
Stinging insects: Hymenoptera species (e.g., bees, yellow jackets, wasps, hornets, fire ants)	Drugs (aspirin, NSAID, opiates, contrast material, ethylene oxide/dialysis tubing)
Medications (e.g., β-lactam antibiotics, ibuprofen)	Complement activation
Biologic agents (e.g., monoclonal antibodies [infliximab, omalizumab] and allergens [challenge tests, specific immunotherapy])	Physical factors (e.g., exercise,† cold, heat, sunlight/ultraviolet radiation)
Natural rubber latex	Ethanol
Vaccines	Idiopathic*
Inhalants (rare) (e.g., horse or hamster dander, grass pollen)	

*In the pediatric population, some anaphylaxis triggers, such as hormones (progesterone), seminal fluid, and occupational allergens, are uncommon, as is idiopathic anaphylaxis.
†Exercise with or without a co-trigger, such as a food or medication, cold air, or cold water.
IVIG, Intravenous immunoglobulin; NSAID, nonsteroidal antiinflammatory drug.
Adapted from Leung DYM, Sampson HA, Geha RS, et al. *Pediatric Allergy Principles and Practice*. Philadelphia: Elsevier; 2010. p. 652.

An adolescent with food allergy frequently leaves home without an epinephrine autoinjector. Which patient risk factor in the table does this illustrate?

A. Evidence of hereditary angioedema
B. Adolescent risk-taking and inconsistent autoinjector carriage
C. A protective effect of older age
D. Proof that all reactions will be mild
E. A reason to discontinue avoidance counseling

ORIGINAL SOURCE TABLE | Table 190.3 | PDF p. 1453 | printed p. 1437

Table 190.3 Patient Risk Factors for Anaphylaxis

AGE-RELATED FACTORS
Infants: anaphylaxis can be difficult to recognize, especially if the first episode; patients cannot describe symptoms
Adolescents and young adults: increased risk-taking behaviors, such as failure to avoid known triggers and to carry an epinephrine autoinjector consistently
Pregnancy: risk of iatrogenic anaphylaxis, as from β-lactam antibiotics to prevent neonatal group B streptococcal infection, agents used perioperatively during caesarean sections, and natural rubber latex
Older people: increased risk of death because of concomitant disease and drugs
CONCOMITANT DISEASES
Asthma and other chronic respiratory diseases
Cardiovascular diseases
Systemic mastocytosis or monoclonal mast cell–activating syndrome
Allergic rhinitis and eczema*
Depression, cognitive dysfunction, substance misuse
DRUGS
NSAIDs
β-Adrenergic blockers†
Mast cell destabilizers
ACE inhibitors†
Sedatives, antidepressants, narcotics, recreational drugs, and alcohol may decrease the patient's ability to recognize triggers and symptoms.
Caffeine
FACTORS THAT MAY INCREASE RISK FOR ANAPHYLAXIS OR MAKE IT MORE DIFFICULT TO TREAT
Age
Asthma
Eczema
Drugs
Alcohol
Other cofactors such as exercise, infection, menses

*Atopic diseases are a risk factor for anaphylaxis triggered by food, latex, and exercise, but not for anaphylaxis triggered by most drugs or by insect stings.
†Those taking β-blockers may not respond optimally to epinephrine treatment and may need glucagon, a polypeptide with non–catecholamine-dependent inotropic and

A child becomes pale and faints during a blood draw, without hives, wheeze, or allergen exposure. Which anaphylaxis mimic is listed?

A. IgE-mediated food anaphylaxis
B. Hereditary angioedema
C. Serum sickness
D. Mast-cell leukemia
E. Vasovagal response

ORIGINAL SOURCE TABLE | Table 190.4 | PDF p. 1453 | printed p. 1437

Table 190.4 Differential Diagnosis of Anaphylaxis

Anaphylaxis to exogenously administered agents
Physical factors
Exercise
Cold, heat, sunlight
Idiopathic

VASODEPRESSOR (VASOVAGAL) RESPONSES
Flushing syndromes
Carcinoid, pheochromocytoma, medullary carcinoma of the thyroid
Menopause
Side effects of chlorpropamide, alcohol, calcium channel blockers
Autonomic epilepsy

FOOD-ASSOCIATED SYNDROMES
Scombroidosis
Sulfites
Monosodium glutamate (MSG)

OTHER FORMS OF SHOCK
Cardiogenic
Septic
Vascular

EXCESS ENDOGENOUS PRODUCTION OF HISTAMINE SYNDROMES
Mast cell activation syndrome
Systemic mastocytosis
Cutaneous mastocytosis
Mast cell leukemia
Acute promyelocytic leukemia

NONORGANIC DISEASE
Panic attacks
Munchausen stridor
Vocal cord dysfunction
Undifferentiated somatoform anaphylaxis

MISCELLANEOUS
Acute urticaria with or without angioedema
Hereditary angioedema
Idiopathic angioedema
Neurologic (seizure, stroke)
Red man syndrome (vancomycin)
Capillary leak syndrome

From Dreskin SC, Stitt JM. Anaphylaxis. In: Burks AW, Holgate ST, O'Hehir RE, et al., eds. *Middleton's Allergy: Principles and Practice*. 9th ed. Philadelphia: Elsevier; 2020: Box 75.6, p. 1237.

A child has hives, wheezing, and hypotension immediately after an allergen exposure. Which first-line emergency treatment in the management table should be given promptly?

A. Observation without treatment
B. An inhaled bronchodilator alone
C. Intramuscular epinephrine
D. Oral cetirizine alone
E. A topical corticosteroid alone

ORIGINAL SOURCE TABLE | Table 190.5 | PDF p. 1454 | printed p. 1438

Table 190.5 Management of a Patient with Anaphylaxis

TREATMENT	MECHANISM(S) OF EFFECT	DOSAGE(S)	COMMENTS; ADVERSE REACTIONS
PATIENT EMERGENCY MANAGEMENT (DEPENDENT ON SEVERITY OF SYMPTOMS)			
Epinephrine (adrenaline)	α_1-, β_1-, β_2-Adrenergic effects	0.01 mg/kg, up to 0.3 mg IM in lateral thigh (0.5 mg autoinjectors are not available in the United States) Epinephrine autoinjector: 0.1 mg for 7.5-13 kg 0.15 mg for <25 kg 0.3 mg for 25 kg or more A second dose may be given in 5 min if symptoms worsen or do not improve	Tachycardia, hypertension, nervousness, headache, nausea, irritability, tremor
Cetirizine (liquid)	Antihistamine (competitive of H_1 receptor)	Cetirizine liquid: 5 mg/5 mL 0.25 mg/kg, up to 10 mg PO	Hypotension, tachycardia, somnolence
Alternative: Diphenhydramine	Antihistamine (competitive of H_1 receptor)	1.25 mg/kg up to 50 mg PO or IM	Hypotension, tachycardia, somnolence, paradoxical excitement
Transport to an emergency facility			
EMERGENCY PERSONNEL MANAGEMENT (DEPENDENT ON SEVERITY OF SYMPTOMS)			
Epinephrine (adrenaline)	α_1-, β_1-, β_2-Adrenergic effects	0.01 mg/kg, up to 0.5 mg IM in lateral thigh Epinephrine autoinjector: 0.1 mg for 7.5-13 kg 0.15 mg for <25 kg 0.3 mg for 25 kg or more 0.01 mL/kg/dose of 1:1,000 (vial) solution, up to 0.5 mL IM May repeat every 10-15 min For severe hypotension: 0.01 mL/kg/dose of 1:10,000 slow IV push	Tachycardia, hypertension, nervousness, headache, nausea, irritability, tremor
Supplemental oxygen and airway management			
Volume Expanders			
Crystalloids (normal saline or Ringer lactate)		30 mL/kg in first hour	Rate titrated against BP response If tolerated, place patient supine with legs raised
Colloids (hydroxyethyl starch)		10 mL/kg rapidly followed by slow infusion	Rate titrated against BP response If tolerated, place patient supine with legs raised
Antihistamines			
Cetirizine (liquid)	Antihistamine (competitive of H_1 receptor)	Cetirizine liquid: 5 mg/5 mL 0.25 mg/kg, up to 10 mg PO	Hypotension, tachycardia, somnolence
Alternative: Diphenhydramine	Antihistamine (competitive of H_1 receptor)	1.25 mg/kg, up to 50 mg PO, IM, or IV	Hypotension, tachycardia, somnolence, paradoxical excitement
Ranitidine	Antihistamine (competitive of H_2 receptor)	1 mg/kg, up to 50 mg IV Should be administered slowly	Headache, mental confusion
Alternative: Cimetidine	Antihistamine (competitive of H_2 receptor)	4 mg/kg, up to 200 mg IV Should be administered slowly	Headache, mental confusion
Corticosteroids			
Methylprednisolone	Antiinflammatory	Solu-Medrol (IV): 1-2 mg/kg, up to 125 mg IV Depo-Medrol (IM): 1 mg/kg, up to 80 mg IM	Hypertension, edema, nervousness, agitation
Prednisone	Antiinflammatory	1 mg/kg up, to 75 mg PO	Hypertension, edema, nervousness, agitation
Nebulized albuterol	β-Agonist	0.83 mg/mL (3 mL) via mask with O_2	Palpitations, nervousness, CNS stimulation, tachycardia; use to supplement epinephrine when bronchospasm appears unresponsive; may repeat
Preventive Treatment Prescription for epinephrine autoinjector and antihistamine Provide written plan outlining patient emergency management (may download form from http://www.aap.org or http://www.foodallergy.org; English and Spanish versions available) Follow-up evaluation to determine/confirm etiology Immunotherapy for insect sting allergy *Patient Education* Instruction on avoidance of causative agent Information on recognizing early signs of anaphylaxis Stress early treatment of allergic symptoms to avoid systemic anaphylaxis Encourage wearing medical identification jewelry			

BP, Blood pressure; CNS, central nervous system; IM, Intramuscularly; IV, intravenously; PO, orally; qd, every day.

A caregiver delays epinephrine for a child with anaphylaxis and gives an oral antihistamine for itching instead. Which problem does the consideration table highlight?

A. Symptom relief can mask warning signs and delay epinephrine
B. Antihistamines reverse hypotension as reliably as epinephrine
C. A previous mild reaction guarantees safety
D. Epinephrine must always be withheld when hives are present
E. Autoinjectors are intended only for adults

ORIGINAL SOURCE TABLE | Table 190.6 | PDF p. 1456 | printed p. 1440

Table 190.6 Considerations with Epinephrine Injection for Anaphylaxis

WHY HEALTHCARE PROFESSIONALS FAIL TO INJECT EPINEPHRINE PROMPTLY

- Lack of recognition of anaphylaxis symptoms; failure to diagnose anaphylaxis
- Episode appears mild, or there is a history of previous mild episode(s)*
- Inappropriate concern about transient mild pharmacologic effects of epinephrine (e.g., tremor)
- Lack of awareness that serious adverse effects are almost always attributable to epinephrine overdose or IV administration, especially IV bolus, rapid IV infusion, or IV infusion of a 1:1,000 epinephrine solution instead of an appropriately diluted solution (1:10,000 concentration)

WHY PATIENTS AND CAREGIVERS FAIL TO INJECT EPINEPHRINE PROMPTLY

- Lack of recognition of anaphylaxis symptoms; failure to diagnose anaphylaxis
- Episode appears mild, or there is a history of previous mild episode(s)*
- H_1 antihistamine or asthma puffer is used initially instead, relieving early warning signs such as itch or cough, respectively
- Prescription for epinephrine autoinjectors (EAIs) is not provided by physician
- Prescription for EAIs is provided but not filled at pharmacy (e.g., not affordable)
- Patients do not carry EAIs consistently (due to size and bulk, or "don't think they'll need it")
- Patients and caregivers are afraid to use EAIs (concern about making an error when giving the injection or about a bad outcome)
- Patients and caregivers are concerned about injury from EAIs
- Competence in using EAIs is associated with regular allergy clinic visits; it decreases as time elapses from first EAI instruction; regular retraining is needed
- Difficulty in understanding how to use EAIs (15% of mothers with no EAI experience could not fire an EAI immediately after a one-on-one demonstration)
- Errors in EAI use can occur despite education, possibly related to the design of some EAIs

WHY PATIENTS OCCASIONALLY FAIL TO RESPOND TO EPINEPHRINE INJECTION

- Delayed recognition of anaphylaxis symptoms; delayed diagnosis
- Error in diagnosis: problem being treated (e.g., foreign body inhalation) is not anaphylaxis
- Rapid progression of anaphylaxis†

Epinephrine†:

- Injected too late; dose too low on mg/kg basis; dose too low because epinephrine solution has degraded (e.g., past the expiration date, stored in a hot place)
- Injection route or site not optimal; dose took too long to be absorbed
- Patient suddenly sits up or walks or runs, leading to the empty ventricle syndrome
- Concurrent use of certain medications (e.g., β-adrenergic blockers)

*Subsequent anaphylaxis episodes can be more severe, less severe, or similar in severity.
†Median times to respiratory or cardiac arrest are 5 min in iatrogenic anaphylaxis, 15 min in stinging-insect venom anaphylaxis, and 30 min in food anaphylaxis; however, regardless of the trigger, respiratory or cardiac arrest can occur within 1 min in anaphylaxis.
Adapted from Leung DYM, Szefler SJ, Bonilla FA Akdis CA, Sampson HA, eds. *Pediatric Allergy Principles and Practice*. Philadelphia: Elsevier; 2016: p. 531.

A child develops fever, rash, and arthralgia days after treatment with cefaclor. Which table-listed drug is associated with serum-sickness reactions?

A. Inhaled albuterol
B. Topical hydrocortisone
C. Intranasal saline
D. Cefaclor
E. Ipratropium nasal spray

ORIGINAL SOURCE TABLE | Table 191.1 | PDF p. 1457 | printed p. 1441

Table 191.1 Proteins and Medications That Cause Serum Sickness*

PROTEINS FROM OTHER SPECIES
Antibotulinum globulin
Antithymocyte globulin
Antitetanus toxoid
Antivenin (Crotalidae) polyvalent (horse serum based)
Crotalidae polyvalent immune Fab (ovine serum based)
Antirabies globulin
Infliximab
Rituximab
Etanercept
Omalizumab
Adalimumab
Natalizumab
Anti-HIV antibodies ([PE]HRG214)
Hymenoptera stings
Streptokinase
H1N1 influenza vaccine
Rabies vaccine
DRUGS
Antibiotics
Cefaclor
Penicillins
Trimethoprim sulfamethoxazole
Minocycline
Meropenem
Neurologic
Bupropion
Carbamazepine
Phenytoin
Sulfonamides
Barbiturates

*Based on review of the most current literature. Other medications that are not listed might also cause serum sickness.

Adapted from Aceves SS. Serum sickness. In: Burg FD, Ingelfinger JR, Polin RA, Gershon AA, eds. *Current Pediatric Therapy*. 18th ed. Philadelphia: Elsevier; 2006: p. 1138.

A child develops bloating and diarrhea after milk but has no hives, wheeze, or immune-mediated findings; lactase deficiency is documented. Which category fits?

A. Drug allergy
B. Nonimmune food intolerance
C. IgE-mediated cow-milk allergy
D. Food-protein-induced enterocolitis by definition
E. Anaphylaxis

ORIGINAL SOURCE TABLE | Table 192.1 | PDF p. 1458 | printed p. 1442

Table 192.1 Adverse Food Reactions

FOOD INTOLERANCE (NON–IMMUNE SYSTEM MEDIATED, NONTOXIC, NONINFECTIOUS)

Host Factors

Enzyme deficiencies—lactase (primary or secondary), sucrase/isomaltase, hereditary fructose intolerance, galactosemia, alcohol dehydrogenase deficiency
Gastrointestinal disorders—inflammatory bowel disease, irritable bowel syndrome, pseudoobstruction, colic
Idiosyncratic reactions—caffeine in soft drinks ("hyperactivity")
Psychologic—food phobias, obsessive/compulsive disorder
Migraines (rare)

Food Factors (Toxic or Infectious or Pharmacologic)

Infectious organisms—*Escherichia coli*, *Staphylococcus aureus*, *Clostridium perfringens*, *Shigella*, botulism, *Salmonella*, *Yersinia*, *Campylobacter*
Toxins—histamine (scombroid poisoning), saxitoxin (shellfish)
Pharmacologic agents—caffeine, theobromine (chocolate, tea), tryptamine (tomatoes), tyramine (cheese), benzoic acid in citrus fruits (perioral flare)
Contaminants—heavy metals, pesticides, antibiotics

FOOD ALLERGY

IgE Mediated

Cutaneous—urticaria, angioedema, morbilliform rashes, flushing, contact urticarial
Gastrointestinal—oral allergy syndrome, gastrointestinal anaphylaxis
Respiratory—acute rhinoconjunctivitis, bronchospasm
Generalized—anaphylactic shock, exercise-induced anaphylaxis

Mixed IgE Mediated and Non–IgE Mediated

Cutaneous—atopic dermatitis, contact dermatitis
Gastrointestinal—allergic eosinophilic esophagitis and gastroenteritis
Respiratory—asthma

Non–IgE Mediated

Cutaneous—contact dermatitis, dermatitis herpetiformis (celiac disease)
Gastrointestinal—food protein–induced enterocolitis, proctocolitis, and enteropathy syndromes, celiac disease
Respiratory—food-induced pulmonary hemosiderosis (Heiner syndrome)
Unclassified

IgE, Immunoglobulin E.

An infant has recurrent vomiting after feeds and poor growth. Which nonallergic gastrointestinal disorder in the food-reaction differential should be considered?

A. Seasonal allergic conjunctivitis
B. Vernal keratoconjunctivitis
C. Dermographism
D. Hereditary angioedema
E. Pyloric stenosis

ORIGINAL SOURCE TABLE | Table 192.2 | PDF p. 1459 | printed p. 1443

Table 192.2 Differential Diagnosis of Adverse Food Reactions

GASTROINTESTINAL DISORDERS (WITH VOMITING AND/OR DIARRHEA)

Structural abnormalities (pyloric stenosis, Hirschsprung disease, reflux)
Enzyme deficiencies (primary or secondary)
  Disaccharidase deficiency—lactase, fructase, sucrase-isomaltase
  Galactosemia
Malignancy with obstruction
Other: pancreatic insufficiency (cystic fibrosis), peptic disease

CONTAMINANTS AND ADDITIVES

Flavorings and preservatives—rarely cause symptoms
  Sodium metabisulfite, monosodium glutamate, nitrites
Dyes and colorings—very rarely cause symptoms (urticaria, eczema)
  Tartrazine
Toxins
  Bacterial, fungal (aflatoxin), fish related (scombroid, ciguatera)
Infectious organisms
  Bacteria (*Salmonella, Escherichia coli, Shigella*)
  Virus (rotavirus, enterovirus)
  Parasites (*Giardia*, Akis simplex [in fish])
Accidental contaminants
  Heavy metals, pesticides
Pharmacologic agents
  Caffeine, glycosidal alkaloid solanine (potato spuds), histamine (fish), serotonin (banana, tomato), tryptamine (tomato), tyramine (cheese)

PSYCHOLOGIC REACTIONS

Food phobias

A toddler with confirmed egg allergy is reassessed over time. Which food in the natural-history table commonly has onset during the first year of life?

A. Tree nut only after adulthood
B. Apple only in infancy
C. Hen egg white
D. Shellfish in most infants
E. Fish only in the neonatal period

ORIGINAL SOURCE TABLE | Table 192.3 | PDF p. 1459 | printed p. 1443

Table 192.3 Natural History of Food Allergy

FOOD	USUAL AGE AT ONSET OF ALLERGY	USUAL AGE AT RESOLUTION
Hen's egg white	0-1 yr	7 yr (75% of cases resolve)*
Cow's milk	0-1 yr	5 yr (76% of cases resolve)*
Peanut	1-2 yr	Persistent (20% of cases resolve)
Tree nuts	1-2 yr; in adults, onset occurs after cross reactivity to birch pollen	Persistent (9% of cases resolve)
Fish	Late childhood and adulthood	Persistent†
Shellfish (crustacean)	Adulthood (in 60% of patients with this allergy)	Persistent
Wheat*	6-24 mo	5 yr (80% of cases resolve)
Soybean*	6-24 mo	2 yr (67% of cases resolve)
Kiwi	Any age	Unknown
Apple, carrot, and peach‡	Late childhood and adulthood	Unknown

*Studies suggest that resolution may occur at a later age, especially in children with multiple food allergies and lifetime peak food-specific IgE >50 kU_A/L.
†Fish allergy that is acquired in childhood can resolve.
‡Allergy to fresh apples, carrots, and peaches (**oral allergy syndrome**) is typically caused by heat-labile proteins. Fresh fruit causes oral pruritus, but cooked fruit is tolerated. Ther
generally no risk of anaphylaxis, although in rare cases, allergies to cross-reactive lipid transfer protein can cause anaphylaxis after ingestion of fruits (e.g., peach) and vegetable

Minutes after a food exposure, a child has lip swelling, wheezing, and vomiting. Which table category best describes the timing and organ involvement?

A. Immediate multisystem food-allergic reaction
B. Delayed-only eczematous reaction
C. Isolated nonimmune lactose intolerance
D. A reaction restricted to the skin
E. A chronic food-protein proctocolitis pattern

ORIGINAL SOURCE TABLE | Table 192.4 | PDF p. 1460 | printed p. 1444

Table 192.4 Symptoms of Food-Induced Allergic Reactions

TARGET ORGAN	IMMEDIATE SYMPTOMS	DELAYED SYMPTOMS
Cutaneous	Erythema Pruritus Urticaria Morbilliform eruption Angioedema	Erythema Flushing Pruritus Morbilliform eruption Angioedema Eczematous rash
Ocular	Pruritus Conjunctival erythema Tearing Periorbital edema	Pruritus Conjunctival erythema Tearing Periorbital edema
Upper respiratory	Nasal congestion Pruritus Rhinorrhea Sneezing Laryngeal edema Hoarseness Dry staccato cough	
Lower respiratory	Cough Chest tightness Dyspnea Wheezing Intercostal retractions Accessory muscle use	Cough Dyspnea Wheezing
Gastrointestinal (oral)	Angioedema of the lips, tongue, or palate Oral pruritus Tongue swelling	
Gastrointestinal (lower)	Nausea Colicky abdominal pain Reflux Vomiting Diarrhea	Nausea Abdominal pain Reflux Vomiting Diarrhea Hematochezia Irritability and food refusal with weight loss (young children)
Cardiovascular	Tachycardia (occasionally bradycardia in anaphylaxis) Hypotension Dizziness Fainting Loss of consciousness	
Other	Uterine contractions Sense of "impending doom"	

From Boyce JA, Assa'ad A, Burks AW, et al. Guideline for the diagnosis and management of food allergy in the United States: report of the NIAID-sponsored expert panel. *J Allergy Clin Immunol*. 2010;126(6):S1–S58. Table IV, p. S19.

Parents ask whether deliberately delaying introduction of allergenic foods prevents allergy in their otherwise healthy infant. Which prevention approach appears in the table?

A. Avoid allergens during maternal pregnancy as routine prevention
B. Use hydrolyzed formula in every infant regardless of feeding choice
C. Require routine testing before any food introduction
D. Introduce infant-safe peanut and egg around 6 months, not before 4 months
E. Delay every allergenic food until after the second birthday

ORIGINAL SOURCE TABLE | Table 192.6 | PDF p. 1463 | printed p. 1447

Table 192.6 Approaches to Prevention of Food Allergy

RECOMMENDED
Infant-safe forms of peanut, egg introduced around age 6 mo, not before 4 mo
Other allergens may be introduced around this time as well
Allergy testing before introduction not usually needed (see text)
Infants with severe eczema or egg allergy may benefit from evaluation for early peanut introduction at 4-6 mo
Diverse infant diet
UNPROVEN/NOT RECOMMENDED
Hydrolyzed formulas
Maternal allergen avoidance during pregnancy or lactation
Purposeful delay in introducing allergens to infants

* https://www.niaid.nih.gov/diseases-conditions/guidelines-clinicians-and-patients-food-allergy..

An infant repeatedly has profuse vomiting two hours after oats, with pallor and lethargy but no hives or wheeze. Which diagnosis is most consistent with the FPIES criteria table?

A. Chronic spontaneous urticaria
B. Acute food protein-induced enterocolitis syndrome
C. IgE-mediated anaphylaxis with immediate hives
D. Lactase deficiency
E. Seasonal allergic rhinitis

ORIGINAL SOURCE TABLE | Table 192.8 | PDF p. 1467 | printed p. 1451

Table 192.8 FPIES Diagnostic Criteria

ACUTE FPIES*	
MAJOR CRITERIA (BOTH MUST BE MET), PLUS	MINOR CRITERIA (≥3 OCCURRING WITH EPISODE)
• Vomiting 1-4 hr after suspect food ingestion • Absence of immediate, IgE-mediated allergic symptoms (hives, itching, swelling, wheezing, cough)	• ≥2 episodes with same food • One episode with a different food • Lethargy • Pallor • Need for ER visit • Need for IV fluid support • Diarrhea within 24 hr (usually 5-10 hr) • Hypotension • Hypothermia
CHRONIC FPIES†	
SYMPTOMS AND SEVERITY	CRITERIA
Milder (lower doses with intermittent ingestion): • Intermittent vomiting and/or diarrhea • Growth faltering • No dehydration or metabolic acidosis **Severe** (higher doses with chronic ingestion): • Intermittent but progressive vomiting and watery diarrhea (occasionally with blood) • Poor weight gain or failure to thrive • Possible dehydration and metabolic acidosis, anemia, hypoproteinemia, neutrophilia, thrombocytosis	• Resolution of symptoms within days to weeks after elimination of offending food(s) • Acute recurrence of symptoms (vomiting in 1-4 hr, diarrhea in <24 hr, usually 5-10 hr) when the food is reintroduced, following a period of elimination • Confirmatory OFC required for conclusive diagnosis; if OFC not performed diagnosis remains presumptive

*Major criterion must be met (both) plus at least three minor criteria.
†General criteria because of paucity of available data.
FPIES, Food protein–induced enterocolitis syndrome; ER, emergency room; IV, intravenous; OFC, oral food challenge.

Minutes after a penicillin dose, a child develops urticaria and bronchospasm. Which immune mechanism category fits the drug-allergy classification?

A. Type II cytotoxic reaction
B. Type III immune-complex reaction
C. Type IVa delayed Th1 reaction
D. Type IVc cytotoxic T-cell reaction
E. Type I IgE-mediated reaction

ORIGINAL SOURCE TABLE | Table 193.1 | PDF p. 1470 | printed p. 1454

Table 193.1 Classification of Drug Allergies

TYPE	TYPE OF IMMUNE RESPONSE	PATHOPHYSIOLOGY	CLINICAL SYMPTOMS	TYPICAL CHRONOLOGY OF THE REACTION
1	IgE	Mast cell and basophil degranulation	Anaphylactic shock Angioedema Urticaria Bronchospasm	Within 1-6 hr after the last intake of the drug
2	IgG and complement	IgG and complement-dependent cytotoxicity	Cytopenia	5-15 days after the start of the eliciting drug
3	IgM or IgG and complement or FcR	Deposition of immune complexes	Serum sickness Urticaria Vasculitis	7-8 days for serum sickness/urticaria 7-21 days after the start of the eliciting drug for vasculitis
4a	Th1 (IFN-γ)	Monocytic inflammation	Eczema	1-21 days after the start of the eliciting drug
4b	Th2 (IL-4 and IL-5)	Eosinophilic inflammation	Maculopapular exanthem, DRESS	1 to several days after the start of the eliciting drug for MPE 2-6 wk after the start of the eliciting drug for DRESS
4c	Cytotoxic T cells (perforin, granzyme B, FASL)	Keratinocyte death mediated by CD4 or CD8	Maculopapular exanthem, SJS/TEN, pustular exanthem	1-2 days after the start of the eliciting drug for fixed drug eruption 4-28 days after the start of the eliciting drug for SJS/TEN
4d	T cells (IL-8/CXCL8)	Neutrophilic inflammation	Acute generalized exanthematous pustulosis	Typically 1-2 days after the start of the eliciting drug *(but could be longer)*

CXCL8, C-X-C motif chemokine ligand 8; DRESS, drug reaction with eosinophilia and systemic symptoms; FASL, FAS ligand; FcR, Fc receptor; IFN-γ , interferon gamma; IgE, immunoglobulin E; IL, interleukin; MPE, malignant pleural effusion; SJS , Stevens-Johnson syndrome; TEN, toxic epidermal necrolysis; Th1, Th2, T-helper cell type 1 and type 2.
From Demoly P, Adkinson NF, Brockow K, et al. International Consensus on drug allergy. *Allergy*. 2014;69(4):420–437.

A child develops a sharply demarcated hyperpigmented plaque that recurs at the same site after the same medication. Which listed drug reaction is most likely?

A. DRESS
B. Anaphylaxis
C. Fixed drug eruption
D. Acute urticaria
E. Serum sickness

ORIGINAL SOURCE TABLE | Table 193.2 | PDF p. 1470 | printed p. 1454

Table 193.2 Heterogeneity of Drug-Induced Allergic Reactions

ORGAN-SPECIFIC REACTIONS	CLINICAL FEATURES	EXAMPLES OF CAUSATIVE AGENTS
CUTANEOUS		
Exanthems	Diffuse fine macules and papules evolve over days after drug initiation Delayed-type hypersensitivity	Allopurinol, aminopenicillins, cephalosporins, antiepileptic agents, antibacterial sulfonamides
Urticaria, angioedema	Onset within minutes of drug initiation Potential for anaphylaxis Usually IgE mediated	β-Lactam antibiotics Platinum agents
Fixed drug eruption	Hyperpigmented plaques Recur at same skin or mucosal site	Tetracycline, sulfonamides, NSAIDs, carbamazepine
Pustules	Acneiform Acute generalized exanthematous pustulosis (AGEP)	Acneiform: corticosteroids, sirolimus AGEP: antibiotics, calcium-channel blockers
Bullous	Tense blisters Flaccid blisters	Furosemide, vancomycin Captopril, penicillamine
SJS	Fever, erosive stomatitis, ocular involvement, purpuric macules on face and trunk with <10% epidermal detachment	Antibacterial sulfonamides, anticonvulsants, oxicam NSAIDs, allopurinol
TEN	Similar features as SJS but >30% epidermal detachment Mortality as high as 50%	Same as SJS
Cutaneous lupus	Erythematous/scaly plaques in photodistribution	Hydrochlorothiazide, calcium channel blockers, ACEIs
Hematologic	Hemolytic anemia, thrombocytopenia, granulocytopenia	Penicillins, quinine, sulfonamides
Hepatic	Hepatitis, cholestatic jaundice	Para-aminosalicylic acid, sulfonamides, phenothiazines
Pulmonary	Pneumonitis, fibrosis	Nitrofurantoin, bleomycin, methotrexate
Renal	Interstitial nephritis, membranous glomerulonephritis	Penicillin, sulfonamides, gold, penicillamine, allopurinol
MULTIORGAN REACTIONS		
Anaphylaxis	Urticaria/angioedema, bronchospasm, gastrointestinal symptoms, hypotension IgE- and non–IgE-dependent reactions	β-Lactam antibiotics, platins
DRESS	Cutaneous eruption, fever, eosinophilia, hepatic dysfunction, lymphadenopathy	Anticonvulsants, sulfonamides, minocycline, allopurinol
Serum sickness	Urticaria, arthralgias, fever	Heterologous antibodies, infliximab
Systemic lupus erythematosus	Arthralgias, myalgias, fever, malaise	Hydralazine, procainamide, isoniazid
Vasculitis	Cutaneous or visceral vasculitis	Hydralazine, penicillamine, propylthiouracil

ACEI, Angiotensin-converting enzyme inhibitor; DRESS, drug reaction with eosinophilia and systemic symptoms; NSAID, nonsteroidal antiinflammatory drug; SJS, Stevens-Johnson syndrome; TEN, toxic epidermal necrolysis.
Adapted from Khan DA, Solensky R. Drug allergy. *J Allergy Clin Immunol*. 2010;125:S126–S137. Table 1, p. S127.

A teenager begins carbamazepine and later develops fever, a widespread eruption, eosinophilia, and systemic symptoms. Which delayed drug-rash category in the table fits?

A. DRESS
B. Simple immediate urticaria
C. Fixed drug eruption alone
D. Vasovagal response
E. Physiologic flushing

ORIGINAL SOURCE TABLE | Table 193.3 | PDF p. 1471 | printed p. 1455

Table 193.3 Delayed Hypersensitivity Drug Rashes by Category

MACULOPAPULAR EXANTHEMS: ANY DRUG CAN PRODUCE A RASH SEVERAL DAYS INTO THE COURSE
Allopurinol
Antibiotics: penicillins, ampicillin, sulfonamides
Antiepileptics: phenytoin, phenobarbital
Antihypertensives: thiazide diuretics, calcium channel blockers
Radiocontrast material
Gold salts
Hypoglycemic drugs
Meprobamate
Phenothiazines
Quinine
DRUG REACTION WITH EOSINOPHILIA AND SYSTEMIC SYMPTOMS (DRESS)
Anticonvulsants: phenytoin, phenobarbital, valproate, lamotrigine, carbamazepine
Antibiotics: sulfonamides, minocycline, dapsone, ampicillin, ethambutol, isoniazid, linezolid, metronidazole, rifampin, streptomycin, vancomycin
Allopurinol
NSAIDs: celecoxib, ibuprofen, diclofenac
Phenothiazines
STEVENS-JOHNSON SYNDROME/TOXIC EPIDERMAL NECROLYSIS
Sulfonamides, phenytoin, barbiturates, carbamazepine, allopurinol, amikacin, phenothiazines, acetazolamide, gold, nitrofurantoin, pentazocine, tetracycline, quinidine
ACUTE GENERALIZED EXANTHEMATOUS PUSTULOSIS
Antibiotics: penicillins, macrolides, cephalosporins, clindamycin, imipenem, fluoroquinolones, isoniazid, vancomycin, minocycline, doxycycline, linezolid, gentamicin, sulfonamides
Antimalarials: chloroquine, hydroxychloroquine
Antifungals: terbinafine, nystatin, amphotericin B, fluconazole, itraconazole
Anticonvulsants: carbamazepine
Calcium-channel blockers
Furosemide, thiazides
Systemic corticosteroids
Protease inhibitors
COLLAGEN VASCULAR OR LUPUS-LIKE REACTIONS
Procainamide, hydralazine, phenytoin, penicillamine, trimethadione, methyldopa, carbamazepine, griseofulvin, nalidixic acid, oral contraceptives, propranolol
ERYTHEMA NODOSUM
Oral contraceptives, penicillins, sulfonamides, diuretics, gold, clonidine, propranolol, opiates
FIXED DRUG REACTIONS
Phenolphthalein, barbiturates, gold, sulfonamides, penicillins, tetracycline, quinolones, carbamazepine, NSAIDs

Note: See Chapter 686 and Table 686.5.
NSAID, Nonsteroidal antiinflammatory drug.
Adapted from Duvic M. Urticaria, drug hypersensitivity rashes, nodules and tumors, and atrophic diseases. In: Goldman L, Schafer AI, eds. *Goldman-Cecil Medicine*. 25th ed.

A child with a convincing immediate amoxicillin allergy is assessed for a related beta-lactam with an identical R1 side chain. Which cephalosporin appears in the shared-side-chain group?

A. Cefotaxime
B. Ceftazidime
C. Cefuroxime
D. Cefadroxil
E. Ceftriaxone

ORIGINAL SOURCE TABLE | Table 193.5 | PDF p. 1473 | printed p. 1457

Table 193.5 Groups of β-Lactam Antibiotics That Share Identical Side Chains*

IDENTICAL R1-GROUP SIDE CHAINS					
Amoxicillin	Ampicillin	Ceftriaxone	Cefoxitin	Cefamandole	Ceftazidime
Cefadroxil	Cefaclor	Cefotaxime	Cephaloridine	Cefonicid	Aztreonam
Cefprozil	Cephalexin	Cefpodoxime	Cephalothin		
Cefatrizine	Cephradine	Cefditoren			
	Cephaloglycin	Ceftizoxime			
	Loracarbef	Cefmenoxime			
IDENTICAL R2-GROUP SIDE CHAINS					
Cephalexin	Cefotaxime	Cefuroxime	Cefotetan	Cefaclor	Ceftibuten
Cefadroxil	Cephalothin	Cefoxitin	Cefamandole	Loracarbef	Ceftizoxime
Cephradine	Cephaloglycin		Cefmetazole		
	Cephapirin		Cefpiramide		

*Each column represents a group with identical side chains.
From Solensky R, Khan DA. Drug allergy: an updated practice parameter. *Ann Allergy Asthma Immunol*. 2010;105:273e1–273e78. Tables 16, 17, p. 273e49.

Answer key and teaching notes

01. D | Table 183.1 | PDF p. 1390, printed p. 1376

Why: The eosinophilia differential includes parasitic and other nonallergic causes.
Closest distractor: An isolated bacterial cystitis is not the more characteristic eosinophilia explanation.

02. B | Table 183.3 | PDF p. 1391, printed p. 1377

Why: The comparison table lists antihistamines as affecting skin tests but not in-vitro specific IgE assays.
Closest distractor: A skin prick test may be suppressed by ongoing antihistamine therapy.

03. E | Table 183.2 | PDF p. 1391, printed p. 1377

Why: DOCK8-associated hyper-IgE immunodeficiency is among nonallergic causes of elevated IgE.
Closest distractor: An elevated total IgE alone cannot establish that rhinitis is the only cause.

04. C | Table 184.1 | PDF p. 1395, printed p. 1380

Why: A nasal foreign body appears under structural/mechanical causes of nonallergic rhinitis.
Closest distractor: Seasonal allergy is less consistent with unilateral foul discharge.

05. A | Table 184.5 | PDF p. 1399, printed p. 1384

Why: The spray table identifies ipratropium as an anticholinergic used for symptomatic rhinorrhea.
Closest distractor: Azelastine is an H1 antagonist rather than the anticholinergic agent requested.

06. D | Table 185.1 | PDF p. 1402, printed p. 1387

Why: Parental asthma is marked as a major risk factor for persistence.
Closest distractor: A single isolated cold is not identified as a major persistence factor.

07. B | Table 185.2 | PDF p. 1403, printed p. 1388

Why: Transient nonatopic wheezing is characterized by early viral-triggered episodes that often resolve.
Closest distractor: Persistent atopy-associated asthma is linked to early eczema or allergen sensitization.

08. E | Table 185.3 | PDF p. 1403, printed p. 1388

Why: Cockroaches appear among indoor aeroallergen triggers in sensitized children.
Closest distractor: Food intolerance does not describe this inhaled allergen trigger.

09. C | Table 185.4 | PDF p. 1404, printed p. 1389

Why: The moderate column includes breathlessness at rest, sitting preference, and speech in phrases.
Closest distractor: The mild column describes breathlessness while walking and speech in sentences.

10. A | Table 185.5 | PDF p. 1405, printed p. 1390

Why: Foreign body aspiration appears among central-airway asthma masqueraders.
Closest distractor: Allergic rhinitis does not explain the abrupt choking and focal wheeze as directly.

11. D | Table 185.6 | PDF p. 1405, printed p. 1390

Why: The table associates paradoxical vocal cord motion with throat tightness, inspiratory stridor, and poor response to bronchodilators.
Closest distractor: Asthma more typically has lower-airway expiratory symptoms and bronchodilator responsiveness.

12. B | Table 185.7 | PDF p. 1406, printed p. 1391

Why: The lung-function table identifies low FEV1/FVC and reversibility after bronchodilator as findings consistent with asthma.
Closest distractor: Fixed upper-airway obstruction would not be best supported by reversible lower-airway airflow limitation.

13. E | Table 185.8 | PDF p. 1407, printed p. 1392

Why: The low-FeNO column explicitly lists recent or current corticosteroid use.
Closest distractor: A low value does not conclusively exclude asthma, especially after treatment.

14. C | Table 185.9 | PDF p. 1409, printed p. 1394

Why: The intermittent column permits symptoms no more than two days per week with no activity interference; the risk domain must also be assessed.
Closest distractor: Mild persistent disease entails more frequent impairment or higher risk.

15. A | Table 185.10 | PDF p. 1410, printed p. 1395

Why: The control table uses symptom and awakening frequency to assess control and guide therapy adjustments.
Closest distractor: Prescribed therapy alone does not demonstrate well-controlled symptoms.

Answer key and teaching notes

16. D | Table 185.11 | PDF p. 1411, printed p. 1396

Why: The productive-visit table explicitly calls for teaching, demonstration, and patient show-back of inhaled medication technique.
Closest distractor: A prescription does not confirm effective use.

17. B | Table 185.12 | PDF p. 1413, printed p. 1398

Why: Tobacco smoke elimination or reduction is listed under control of contributing exposures.
Closest distractor: Increasing rescue use does not address the modifiable exposure.

18. E | Table 185.16 | PDF p. 1418, printed p. 1403

Why: Chronic systemic corticosteroid use is explicitly in the high-risk category of the adverse-effect table.
Closest distractor: Low risk is described for lower-dose inhaled corticosteroids with few risk factors.

19. C | Table 185.18 | PDF p. 1423, printed p. 1408

Why: Prior ICU admission or intubation appears among biologic risk factors for asthma morbidity and mortality.
Closest distractor: It does not imply that later exacerbations are low risk.

20. A | Table 186.1 | PDF p. 1429, printed p. 1413

Why: Pruritus, infant facial/extensor eczema, and chronic or relapsing dermatitis are major atopic dermatitis features.
Closest distractor: Urticarial vasculitis produces a different lesion pattern and persistence.

21. D | Table 186.2 | PDF p. 1430, printed p. 1414

Why: Scabies appears among infectious skin diseases in the atopic dermatitis differential.
Closest distractor: Asthma does not explain a contagious pruritic eruption.

22. B | Table 186.3 | PDF p. 1431, printed p. 1415

Why: Wiskott–Aldrich syndrome is included among immunodeficiencies associated with eczema.
Closest distractor: C1 inhibitor deficiency causes angioedema rather than this eczematous thrombocytopenic phenotype.

23. E | Table 186.4 | PDF p. 1432, printed p. 1416

Why: The counseling table supports a normal diet unless allergy workup establishes a need to avoid a particular food.
Closest distractor: Broad empiric elimination is not the table recommendation.

24. C | Table 186.5 | PDF p. 1433, printed p. 1417

Why: Widespread dryness and incessant itch with oozing or extensive thickening match severe eczema.
Closest distractor: Moderate disease has frequent itch and localized thickening without these extensive findings.

25. A | Table 186.6 | PDF p. 1433, printed p. 1417

Why: Hydrocortisone appears in group 7, the least-potent group of the table.
Closest distractor: Clobetasol is group 1, a superpotent preparation.

26. D | Table 188.1 | PDF p. 1438, printed p. 1422

Why: Vernal keratoconjunctivitis is listed for young patients, male predominance, warm dry climate, and chronic ocular symptoms.
Closest distractor: Giant papillary conjunctivitis is linked to prosthetic exposure.

27. B | Table 189.1 | PDF p. 1442, printed p. 1426

Why: Viral and other infections appear in the acute-urticaria causes table.
Closest distractor: Acute hives need not be attributed exclusively to food allergy.

28. E | Table 189.2 | PDF p. 1442, printed p. 1426

Why: Dermographism is listed under inducible chronic urticaria.
Closest distractor: Hereditary angioedema typically lacks the provoked wheals of dermographism.

29. C | Table 189.3 | PDF p. 1443, printed p. 1427

Why: The autoinflammatory table assigns familial cold autoinflammatory syndrome to NLRP3.
Closest distractor: BTK deficiency is an antibody-development disorder rather than this periodic cold-triggered syndrome.

30. A | Table 189.4 | PDF p. 1444, printed p. 1428

Why: Persistent painful plaques with residual bruising are atypical features suggesting an urticarial syndrome.
Closest distractor: Ordinary wheals are transient and resolve without residual signs.

Answer key and teaching notes

31. D | Table 189.5 | PDF p. 1445, printed p. 1429

Why: The physical-urticaria comparison associates wheals after stroking with dermographism.
Closest distractor: Delayed pressure urticaria appears after sustained pressure rather than immediate light stroking.

32. B | Table 189.6 | PDF p. 1446, printed p. 1430

Why: The hereditary cold-urticaria table lists PLAID with evaporative cooling and sinopulmonary infections.
Closest distractor: Seasonal pollen allergy does not explain cooling-triggered hives with immune deficiency.

33. E | Table 189.7 | PDF p. 1447, printed p. 1431

Why: The diagnostic-testing table calls for C4 and C1-inhibitor testing by protein and function.
Closest distractor: Total IgE does not assess C1-inhibitor deficiency.

34. C | Table 189.9 | PDF p. 1449, printed p. 1433

Why: Type 1 HAE has low C4 and low C1-inhibitor quantity and function in the complement-evaluation table.
Closest distractor: Type 2 HAE generally has normal or elevated C1-inhibitor protein with low function.

35. A | Table 190.1 | PDF p. 1450, printed p. 1434

Why: Acute skin involvement plus respiratory compromise satisfies one clinical criterion for anaphylaxis.
Closest distractor: Isolated urticaria lacks the accompanying respiratory compromise.

36. D | Table 190.2 | PDF p. 1451, printed p. 1435

Why: Yellow jackets are among Hymenoptera insect triggers in the anaphylaxis table.
Closest distractor: An aeroallergen is neither required nor the best match to the immediate sting history.

37. B | Table 190.3 | PDF p. 1453, printed p. 1437

Why: The age-related risk-factor table mentions adolescents failing to avoid known triggers or carry epinephrine consistently.
Closest distractor: Prior behavior does not establish that future reactions are mild.

38. E | Table 190.4 | PDF p. 1453, printed p. 1437

Why: Vasodepressor or vasovagal responses are included in the anaphylaxis differential.
Closest distractor: IgE-mediated anaphylaxis is less supported without an exposure and characteristic multisystem signs.

39. C | Table 190.5 | PDF p. 1454, printed p. 1438

Why: Epinephrine is the primary emergency treatment listed for anaphylaxis.
Closest distractor: An antihistamine does not treat airway or circulatory compromise adequately.

40. A | Table 190.6 | PDF p. 1456, printed p. 1440

Why: The table specifically notes antihistamines used first can relieve early itch while delaying epinephrine.
Closest distractor: Antihistamines do not replace epinephrine for systemic anaphylaxis.

41. D | Table 191.1 | PDF p. 1457, printed p. 1441

Why: Cefaclor is listed among antibiotics associated with serum sickness.
Closest distractor: Intranasal saline is not a serum-sickness agent in the table.

42. B | Table 192.1 | PDF p. 1458, printed p. 1442

Why: Lactase enzyme deficiency is listed as a nonimmune food intolerance.
Closest distractor: IgE-mediated milk allergy has an immune mechanism not established by isolated lactase deficiency.

43. E | Table 192.2 | PDF p. 1459, printed p. 1443

Why: Pyloric stenosis is included among structural gastrointestinal alternatives to food allergy.
Closest distractor: Allergic conjunctivitis does not explain feed-related vomiting and growth concerns.

44. C | Table 192.3 | PDF p. 1459, printed p. 1443

Why: Egg white is listed with usual onset at ages 0–1 year.
Closest distractor: Shellfish often presents later, including adulthood.

45. A | Table 192.4 | PDF p. 1460, printed p. 1444

Why: The immediate column includes oral angioedema, lower-respiratory wheeze, and gastrointestinal vomiting.
Closest distractor: Delayed eczema alone cannot account for this immediate multisystem presentation.

Answer key and teaching notes

46. D | Table 192.6 | PDF p. 1463, printed p. 1447

Why: The prevention table recommends infant-safe peanut and egg around 6 months, not before 4 months, and rejects purposeful delay.
Closest distractor: Routine delayed introduction is listed as unproven or not recommended.

47. B | Table 192.8 | PDF p. 1467, printed p. 1451

Why: Acute FPIES criteria emphasize vomiting 1–4 hours after the food, absence of immediate IgE symptoms, and supporting features such as pallor and lethargy.
Closest distractor: Classic IgE-mediated anaphylaxis is usually immediate and may include hives or wheeze.

48. E | Table 193.1 | PDF p. 1470, printed p. 1454

Why: The table defines type I by IgE-driven mast-cell/basophil degranulation with urticaria and bronchospasm.
Closest distractor: Type III reactions involve immune-complex deposition and a different timing and phenotype.

49. C | Table 193.2 | PDF p. 1470, printed p. 1454

Why: The heterogeneous-reaction table describes fixed drug eruptions as plaques recurring at the same skin or mucosal site.
Closest distractor: Urticaria consists of transient wheals rather than a recurrent fixed plaque.

50. A | Table 193.3 | PDF p. 1471, printed p. 1455

Why: Carbamazepine is listed among anticonvulsants associated with drug reaction with eosinophilia and systemic symptoms.
Closest distractor: Immediate urticaria lacks the delayed fever/eosinophilia/systemic pattern.

51. D | Table 193.5 | PDF p. 1473, printed p. 1457

Why: The R1 side-chain table groups amoxicillin with cefadroxil.
Closest distractor: Ceftriaxone is shown in a different R1 group.

Table selection log

01. Table 183.1: Differential Diagnosis of Childhood Eosinophilia | PDF p. 1390 | printed p. 1376 | Completed

02. Table 183.3: Determination of Allergen--Specific IgE by Skin Testing vs In Vitro Testing | PDF p. 1391 | printed p. 1377 | Completed

03. Table 183.2: Nonallergic Diseases Associated with Increased Serum IgE Concentrations | PDF p. 1391 | printed p. 1377 | Completed

04. Table 184.1: Causes of Rhinitis | PDF p. 1395 | printed p. 1380 | Completed

05. Table 184.5: Miscellaneous Intranasal Sprays | PDF p. 1399 | printed p. 1384 | Completed

06. Table 185.1: Early Childhood Risk Factors for Persistent | PDF p. 1402 | printed p. 1387 | Completed

07. Table 185.2: Asthma Patterns in Childhood, Based on | PDF p. 1403 | printed p. 1388 | Completed

08. Table 185.3: Asthma Triggers | PDF p. 1403 | printed p. 1388 | Completed

09. Table 185.4: Formal Evaluation of Asthma Exacerbation Severity in the Urgent or Emergency Care Setting* | PDF p. 1404 | printed p. 1389 | Completed

10. Table 185.5: Differential Diagnosis of Childhood Asthma | PDF p. 1405 | printed p. 1390 | Completed

11. Table 185.6: Features Distinguishing Paradoxical Vocal Cord Motion Disorder from Asthma | PDF p. 1405 | printed p. 1390 | Completed

12. Table 185.7: Lung Function Abnormalities in Asthma | PDF p. 1406 | printed p. 1391 | Completed

13. Table 185.8: Interpretations of FeNO Test Results for Asthma Diagnosis in Nonsmoking Individuals Not Taking Corticosteroids | PDF p. 1407 | printed p. 1392 | Completed

14. Table 185.9: Assessing Asthma Severity* | PDF p. 1409 | printed p. 1394 | Completed

15. Table 185.10: Assessing Asthma Control and Adjusting Therapy in Children* | PDF p. 1410 | printed p. 1395 | Completed

16. Table 185.11: Key Elements of Productive Clinic Visits for | PDF p. 1411 | printed p. 1396 | Completed

17. Table 185.12: Control of Factors Contributing to Asthma | PDF p. 1413 | printed p. 1398 | Completed

18. Table 185.16: Risk Assessment for Corticosteroid Adverse Effects | PDF p. 1418 | printed p. 1403 | Completed

19. Table 185.18: Risk Factors for Asthma Morbidity and | PDF p. 1423 | printed p. 1408 | Completed

20. Table 186.1: Clinical Features of Atopic Dermatitis | PDF p. 1429 | printed p. 1413 | Completed

21. Table 186.2: Differential Diagnosis of Atopic Dermatitis | PDF p. 1430 | printed p. 1414 | Completed

22. Table 186.3: Features of Primary Immunodeficiencies Associated with Eczematous Dermatitis | PDF p. 1431 | printed p. 1415 | Completed

23. Table 186.4: Counseling and Aggravating Factors for | PDF p. 1432 | printed p. 1416 | Completed

24. Table 186.5: Categorization of Physical Severity of Atopic | PDF p. 1433 | printed p. 1417 | Completed

25. Table 186.6: Selected Topical Corticosteroid Preparations* | PDF p. 1433 | printed p. 1417 | Completed

26. Table 188.1: Allergic Diseases of the Eye | PDF p. 1438 | printed p. 1422 | Completed

27. Table 189.1: Etiology of Acute Urticaria | PDF p. 1442 | printed p. 1426 | Completed

28. Table 189.2: Etiology of Chronic Urticaria | PDF p. 1442 | printed p. 1426 | Completed

29. Table 189.3: Febrile Autoinflammatory Diseases Causing Urticaria in Children | PDF p. 1443 | printed p. 1427 | Completed

30. Table 189.4: Distinguishing Features Between Urticaria | PDF p. 1444 | printed p. 1428 | Completed

31. Table 189.5: Comparison of the Physical Urticarias | PDF p. 1445 | printed p. 1429 | Completed

32. Table 189.6: Hereditary Diseases with Cold--Induced Urticaria | PDF p. 1446 | printed p. 1430 | Completed

33. Table 189.7: Diagnostic Testing for Urticaria and | PDF p. 1447 | printed p. 1431 | Completed

34. Table 189.9: Complement Evaluation of Patients with Recurrent Angioedema | PDF p. 1449 | printed p. 1433 | Completed

35. Table 190.1: Clinical Criteria for Diagnosing Anaphylaxis | PDF p. 1450 | printed p. 1434 | Completed

36. Table 190.2: Anaphylaxis Triggers in the Community* | PDF p. 1451 | printed p. 1435 | Completed

37. Table 190.3: Patient Risk Factors for Anaphylaxis | PDF p. 1453 | printed p. 1437 | Completed

38. Table 190.4: Differential Diagnosis of Anaphylaxis | PDF p. 1453 | printed p. 1437 | Completed

Table selection log

39. Table 190.5: Management of a Patient with Anaphylaxis | PDF p. 1454 | printed p. 1438 | Completed

40. Table 190.6: Considerations with Epinephrine Injection for Anaphylaxis | PDF p. 1456 | printed p. 1440 | Completed

41. Table 191.1: Proteins and Medications That Cause Serum | PDF p. 1457 | printed p. 1441 | Completed

42. Table 192.1: Adverse Food Reactions | PDF p. 1458 | printed p. 1442 | Completed

43. Table 192.2: Differential Diagnosis of Adverse Food | PDF p. 1459 | printed p. 1443 | Completed

44. Table 192.3: Natural History of Food Allergy | PDF p. 1459 | printed p. 1443 | Completed

45. Table 192.4: Symptoms of Food--Induced Allergic | PDF p. 1460 | printed p. 1444 | Completed

46. Table 192.6: Approaches to Prevention of Food Allergy | PDF p. 1463 | printed p. 1447 | Completed

47. Table 192.8: FPIES Diagnostic Criteria | PDF p. 1467 | printed p. 1451 | Completed

48. Table 193.1: Classification of Drug Allergies | PDF p. 1470 | printed p. 1454 | Completed

49. Table 193.2: Heterogeneity of Drug--Induced Allergic Reactions | PDF p. 1470 | printed p. 1454 | Completed

50. Table 193.3: Delayed Hypersensitivity Drug Rashes by | PDF p. 1471 | printed p. 1455 | Completed

51. Table 193.5: Groups of β--Lactam Antibiotics That Share Identical Side Chains* | PDF p. 1473 | printed p. 1457 | Completed

Table 184.2: Oral Allergic Rhinitis Treatments (Prescription, Examples) | PDF p. 1397 | Excluded: Prescription brand/strength list is time sensitive

Table 184.3: Oral Allergic Rhinitis Treatments (Nonprescription, Examples) | PDF p. 1398 | Excluded: Nonprescription brand/strength list is time sensitive

Table 184.4: Combined Antihistamine + Sympathomimetic (Examples) | PDF p. 1398 | Excluded: Combination brand/strength list is time sensitive

Table 184.6: Intranasal Inhaled Corticosteroids | PDF p. 1399 | Excluded: Continues onto PDF p. 1400

Table 185.13: Stepwise Approach for Managing Asthma in Children* | PDF p. 1414 | Excluded: Continues onto PDF p. 1415; step guidance requires current review

Table 185.14: Usual Dosages for Long--Term Control Medications | PDF p. 1416 | Excluded: Long-term drug dosage/formulation list requires current review

Table 185.15: Estimated Comparative Inhaled Corticosteroid Doses | PDF p. 1417 | Excluded: Comparative inhaled steroid dose list requires current review

Table 185.17: Management of Asthma Exacerbation (Status Asthmaticus) | PDF p. 1420 | Excluded: Continues onto PDF p. 1421

Table 187.1: Risk of Systemic Reaction in Untreated Patients with History of Sting Anaphylaxis and/or Positive Venom Skin | PDF p. 1437 | Excluded: Historical recurrence-risk statistics; limited individual decision value

Table 188.2: Topical Ophthalmic Medications for Allergic Conjunctivitis | PDF p. 1440 | Excluded: Continues onto PDF p. 1441

Table 189.8: Treatment of Urticaria and Angioedema | PDF p. 1447 | Excluded: Drug dose list includes older products and requires current review

Table 189.10: Long--Term Prophylactic Treatment Options for Patients with Hereditary Angioedema in the United States | PDF p. 1449 | Excluded: Approval-year and hereditary-angioedema prophylaxis product list

Table 189.11: On--Demand Treatment Options for Patients with Hereditary Angioedema in the United States | PDF p. 1450 | Excluded: Approval-year and on-demand product list

Table 192.5: Clinical Implications of Cross--Reactive Proteins in IgE--Mediated Allergy | PDF p. 1462 | Excluded: Historical cross-reactivity percentage estimates

Table 192.7: Food Protein–Induced Gastrointestinal Syndromes | PDF p. 1465 | Excluded: Continues onto PDF p. 1466

Table 192.9: Empiric Guidelines for Selecting Weaning Foods in Infants with FPIES | PDF p. 1469 | Excluded: Empiric FPIES food-risk recommendations require individual review

Table 193.4: Penicillin Skin Test Reagents | PDF p. 1473 | Excluded: Skin-test reagent concentrations require specialist/current protocol review

Total: 51 completed + 17 excluded = 68 tables in Part XII.

Last completed: Table 193.5, PDF p. 1473. Part XIV begins PDF p. 1476.

Clinical review: Nelson asthma severity/treatment tables should be compared with GINA 2026.

https://ginasthma.org/2026-gina-strategy-report/

Anaphylaxis: current specialist guidance continues to prioritize IM epinephrine.

https://www.worldallergy.org/resources

Early peanut introduction: https://www.niaid.nih.gov/sites/default/files/addendum-peanut-allergy-prevention-guidelines.pdf