

TERRY DEAN JR. | LOUIS M. BELL



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PEDIATRICS BOARD REVIEW

CERTIFICATION AND RECERTIFICATION

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Contents

- 1** *Maximizing Test Performance: Effective Study and Test-Taking Strategies,* 1

TERRY DEAN JR.

SECTION 1

Allergy, 5

- 2** *Atopic Diseases,* 6
KIRAN P. PATEL and LAUREN A. SANCHEZ
- 3** *Allergies, Anaphylaxis, and Drug Reactions,* 8
KIRAN P. PATEL and LAUREN A. SANCHEZ
- 4** *Selected Topics in Allergy,* 13
KIRAN P. PATEL and LAUREN A. SANCHEZ

SECTION 2

Cardiology, 17

- 5** *Clinical Approach to Common Cardiac Complaints,* 18
DESIRÉE R. CONRAD, ALAINA K. KIPPS, CHARITHA D. REDDY, and NEHA J. PURKEY
- 6** *Structural Heart Disease,* 24
ANNA M. DEITCH, NEHA J. PURKEY, ALAINA K. KIPPS, and CHARITHA D. REDDY
- 7** *Acquired Heart Disease,* 41
LYNDSY HUNTER-ADAMSON, NEHA J. PURKEY, CHARITHA D. REDDY, and ALAINA K. KIPPS
- 8** *Selected Topics in Cardiology,* 45
BRITTANY M. NAVARRE, NEHA J. PURKEY, ALAINA KIPPS, and CHARITHA D. REDDY

SECTION 3

Dermatology, 55

- 9** *Neonatal Skin Disorders,* 56
VICTORIA MITRE and RAEGAN HUNT
- 10** *Dermatologic Emergencies,* 62
KELLY K. BARRY, ELENA B. HAWRYLUK, and DIANA BARTENSTEIN REUSCH
- 11** *Infectious Dermatology,* 73
ASHLEY TORKAN ZILBERSTEIN and LACEY KRUSE
- 12** *Selected Topics in Dermatology,* 83
NONYE OGBUEFI, DURI YUN, and BRANDI KENNER-BELL

SECTION 4

Emergency Medicine and Trauma, 97

- 13** *Shock,* 98
GAYATHRI PRABHAKAR and MEKELA WHYTE-NESFIELD
- 14** *BRUE, SIDS, and Cardiopulmonary Arrest,* 103
JONATHAN BRYAN COOPER-SOOD
- 15** *Pediatric Trauma,* 112
MALEK MAZZAWI and CAMILO E. GUTIERREZ
- 16** *Bites, Stings, and Wounds,* 122
MAMATA V. SENTHIL
- 17** *Heat-Related Illnesses and Pediatric Drowning,* 128
COURTNEY ELLEN NELSON
- 18** *Toxicology,* 134
MEGHAN MEGHPARA and COURTNEY ELLEN NELSON
- 19** *Selected Topics in Emergency Medicine,* 142
PETER AIDAN SLOANE

SECTION 5

Endocrinology, 149

- 20** *Hyperglycemia and Hypoglycemia,* 150
SARAH JIANG and WINNIE SIGAL
- 21** *Differences of Sexual Development and Pubertal Development,* 159
CAMILIA KAMOUN and MARIA G. VOGIATZI
- 22** *Growth Disorders,* 171
EINAS ALKHATIB and ROOPA KANAKATTI SHANKAR
- 23** *Thyroid Disorders,* 176
ANDREW J. BAUER
- 24** *Disorders of Calcium Homeostasis,* 185
JANET YI MAN LEE
- 25** *Adrenal Disorders,* 192
MARISSA JOELLE KILBERG and ALLIE DAYNO
- 26** *Endocrine Dysnatremias,* 198
MARISSA JOELLE KILBERG
- 27** *Selected Topics in Endocrinology,* 201
MARIA ZHADINA

SECTION 6**Gastroenterology, 205**

- 28** *Clinical Approach to Emesis and Diarrhea,* 206
NINA N. SAINATH
- 29** *Clinical Approach to Gastrointestinal Bleed,* 211
LISA FAHEY and ALEXANDER COE
- 30** *Esophageal and Gastric Disorders,* 214
BRIDGET C. GODWIN and JENNIFER B. WEBSTER
- 31** *Intestinal Disorders,* 221
MÁIRE A. CONRAD and ELIZABETH CLABBY MAXWELL
- 32** *Hepatobiliary Disorders and Liver Failure,* 235
MICHAL REBEKA ROZENFELD BAR LEV, Yael MOZER-GLASSBERG,
and ORITH WAISBOURD-ZINMAN
- 33** *Pancreatic Disorders,* 252
JEFFERSON NAYLOR BROWNELL
- 34** *Functional Gastrointestinal Disorders,* 257
ALICE CHAR-MEI HUANG and JENNIFER B. WEBSTER

SECTION 7**Genetics and Dysmorphology, 261**

- 35** *Selected Topics in Genetics and Dysmorphology,* 262
LEAH KE'ALA'AUMOE DOWSETT

SECTION 8**Hematology, 281**

- 36** *Anemia and Erythrocyte Disorders,* 282
ARUN GURUNATHAN
- 37** *Platelet Disorders and Coagulopathies,* 293
TIFFANY L. LUCAS
- 38** *Selected Topics in Hematology,* 299
ALEKSANDRA S. DAIN

SECTION 9**Immunology, 305**

- 39** *Clinical Approach to Suspected Immune Deficiency,* 306
ALICE CHAN and LAUREN A. SANCHEZ
- 40** *Phagocyte Disorders,* 308
ALICE CHAN and LAUREN A. SANCHEZ

- 41** *Humoral and Combined Immunodeficiencies,* 311
ALICE CHAN and LAUREN A. SANCHEZ

- 42** *Selected Topics in Immunology,* 314
ALICE CHAN and LAUREN A. SANCHEZ

SECTION 10**Infectious Diseases, 317**

- 43** *Clinical Approach to Common Infectious Disease Complaints,* 318
NICOLE HAMES
- 44** *Bacteria,* 325
VIRGINIA LONG, SHREYA DOSHI, and MARIA SUSANA RUEDA-ALTEZ
- 45** *Viruses,* 345
LORI KESTENBAUM HANDY
- 46** *Fungi, Worms, and Parasites,* 356
MUAYAD ALALI and LESLIE A. ENANE
- 47** *Therapeutic Agents in Infectious Diseases,* 365
BEATRIZ LARRU MARTINEZ and ANDREW IAN TAYLOR

SECTION 11**Metabolism, 373**

- 48** *Clinical Approach to Suspected Metabolic Disorders,* 374
ALANNA STRONG and REBECCA D. GANETZKY
- 49** *Overview of Metabolic Disorders,* 378
ALANNA STRONG and REBECCA GANETZKY

SECTION 12**Nephrology, 401**

- 50** *Management of Fluids and Electrolytes,* 402
SHEENA SHARMA
- 51** *Clinical Approach to Acid-Base Disturbances,* 408
LINDA WANG and AADIL KAKAJIWALA
- 52** *Clinical Approach to Common Complaints in Nephrology,* 414
RADHA GAJJAR
- 53** *Nephritic and Nephrotic Syndromes,* 423
ANITA TAMBAY PEREZ and MELISSA REBA MEYERS
- 54** *Selected Topics in Nephrology,* 430
LINDA WANG and AADIL KAKAJIWALA

SECTION 13**Neurology, 439**

- 55** *Pediatric Neurologic Assessment,* 440
NAN LIN and DAVID R. GARNER
- 56** *Altered Mental Status and Headache,* 444
DAVID R. GARNER and NAN LIN
- 57** *Seizures,* 456
DOUGLAS M. SMITH
- 58** *Weakness and Ataxia,* 460
RACHEL GOTTLIEB-SMITH
- 59** *Congenital Malformations of the Central Nervous System,* 468
DOUGLAS M. SMITH
- 60** *Selected Topics in Neurology,* 471
JULIE ZIOBRO and PATRICK DONALD LEE MABRAY

SECTION 14**Oncology, 479**

- 61** *Hematologic Malignancies,* 480
DAVID S. ANDERSON
- 62** *Neuroblastoma and Brain Tumors,* 486
CHRISTINA SOPHIA TURN and ANDREA WEBSTER CARRION
- 63** *Solid Tumors,* 490
CHRISTINA SOPHIA TURN and RACHEL M. HURLEY
- 64** *Selected Topics in Oncology,* 495
EMILY ABBEY and KERI TONER

SECTION 15**Ophthalmology, 505**

- 65** *Clinical Approach to Common Ophthalmologic Complaints,* 506
MARINA A. EISENBERG
- 66** *Disorders of Eye Alignment and Movement,* 512
CATHERINE S. CHOI
- 67** *Disorders of the External Eye and Anterior Segment,* 515
CATHERINE S. CHOI
- 68** *Disorders of the Posterior Segment,* 521
MARINA A. EISENBERG

SECTION 16**Otorhinolaryngology, 527**

- 69** *Disorders of the Ear and Audition,* 528
DAVID R. LEE and BRIAN KIP REILLY
- 70** *Disorders of the Nose and Sinuses,* 538
ASHLEY MARIE LLOYD and HABIB GEORGE ZALZAL
- 71** *Disorders of the Oropharynx and Neck,* 548
EVIE C. LANDRY, COLIN BOHR, and LYUBA GITMAN

SECTION 17**Orthopedics, 559**

- 72** *Common Orthopedic Injuries,* 560
JOSEPH L. YELLIN and JOHN T. LAWRENCE
- 73** *Selected Topics in Orthopedics,* 576
JOSEPH L. YELLIN and JOHN TODD R. LAWRENCE

SECTION 18**Pulmonology, 595**

- 74** *Clinical Approach to Common Pulmonologic Complaints,* 596
CASANDRA AREVALO MARCANO and PI CHUN CHENG
- 75** *Neonatal Lung Disorders,* 604
JULIE L. FIERRO
- 76** *Disorders of the Upper Airway,* 613
JEANETTE TRAVER VAN STEYN and NICHOLAS LEE FRIEDMAN
- 77** *Disorders of the Lower Airway,* 620
JEANETTE TRAVER VAN STEYN, NICHOLAS LEE FRIEDMAN, and JASON Z. BRONSTEIN
- 78** *Parenchymal Lung Disease,* 631
ELIZABETH GIBB and KENSHO IWANAGA
- 79** *Systemic Diseases With Pulmonary Involvement,* 638
AGNES SIBILSKI MONTGOMERY and IMAN SAMI-ZAKHARI

SECTION 19**Psychiatry, 649**

- 80** *Selected Topics in Psychiatry,* 650
KATRINA A. FLETCHER, GWENDOLYN J. MESSER, CATHARYN A. TURNER II, and AMY KIM

SECTION 20**Rheumatology, 665**

- 81** *Clinical Approach to Arthritis,* 666
SARAH D. BAYEFSKY and JOYCE CHUN-LING CHANG
- 82** *Inflammatory Arthritis,* 670
SARAH D. BAYEFSKY and JOYCE CHUN-LING CHANG
- 83** *Systemic Autoimmune Diseases,* 674
SARAH D. BAYEFSKY and JOYCE CHUN-LING CHANG
- 84** *Selected Topics in Rheumatology,* 680
SARAH D. BAYEFSKY and JOYCE CHUN-LING CHANG

SECTION 21**Urology, 685**

- 85** *Disorders of the Kidney, Collecting System/Ureters, Bladder, and Urethra,* 686
HANS G. POHL
- 86** *Disorders of the Male Genital System,* 696
HANS G. POHL

SECTION 22**Ambulatory Pediatrics, 703**

- 87** *Nutrition,* 704
KAYLA BERGMAN and NICOLE POTOLICCHIO
- 88** *Immunizations,* 715
GRACE KIM
- 89** *Preventive Pediatrics,* 724
JACQUELINE STEELE and KAITLYN M. MURPHY
- 90** *Normal Development and Disorders of Cognition, Language, and Learning,* 729
JACQUELINE STEELE and KAITLYN M. MURPHY
- 91** *Child Abuse and Neglect,* 735
RICKI STEPHANIE CARROLL and STEPHANIE ANNE DEUTSCH
- 92** *Social Determinants of Health,* 744
GWYNNE LATIMER

SECTION 23**Adolescent Medicine, 749**

- 93** *Menstrual Disorders and Hormonal Contraception,* 750
BROCK D. LIBBY and JENNIFER H. CHUANG
- 94** *Infectious Diseases of the Genital Tract,* 757
ROSHEEN GRADY and JENNIFER H. CHUANG
- 95** *Selected Topics in Gynecology,* 770
JENNIFER H. CHUANG
- 96** *Selected Topics in Adolescent Medicine,* 775
JENNIFER H. CHUANG
- 97** *Selected Topics in Prenatal Medicine and Obstetrics,* 783
ROXANNE RENGIFO, DINEASHA POTTER-MCQUILKIN, and JENNIFER H. CHUANG

SECTION 24**Research and Statistics, 791**

- 98** *Clinical Epidemiology,* 792
ANITA K. PATEL

SECTION 25**Ethics, 799**

- 99** *Ethics,* 800
SARA TAUB and MATTHEW DRAGO

SECTION 26**Patient Safety and Quality Improvement, 807**

- 100** *Patient Safety and Quality Improvement,* 808
EVAN DALTON and JESSICA K. HART

Index, 813

SECTION

1

Allergy

2

Atopic Diseases

KIRAN P. PATEL, MD, MS and LAUREN A. SANCHEZ, MD

Basic Information

- The atopic diseases include atopic dermatitis (eczema), food allergies, allergic rhinitis, and asthma
- “Atopic march” or “allergic march” refers to the sequential presentation of atopic diseases, typically in the order listed above, from infancy through childhood
- Causes
 - Complex interaction between genetic risk factors and environmental influence
 - Early exposure to endotoxins, pets, farm animals, and daycare may be protective against the development of atopic dermatitis (“hygiene hypothesis”)
 - Maternal diet and breastfeeding do not play a role in the development of atopy
 - Delaying introduction of solid foods past 4 to 6 months does *not* prevent development of food allergies; however, early introduction of peanuts in infants with moderate to severe eczema lowers the risk of development of peanut allergy

Atopic Dermatitis

Basic Information

- Chronic, inflammatory, and pruritic skin disease
- Prevalence: up to 20% prevalence in children and rising, especially in developed nations
- Etiology is multifactorial (epidermal barrier dysfunction, altered skin flora, immune dysregulation)
- Known risk factors include parental history of atopic disease, genetic variants in filaggrin (FLG), environmental exposures
- Distinct from idiopathic nummular eczema, allergic contact dermatitis, chronic hand eczema
- See [Chapter 12](#) for additional information

Clinical Presentation

- Can present as early as 2 to 6 months of age; 85% to 90% present by age 5 years
- Chronic or relapsing course of red macules and papules with intense pruritus and lichenification
 - <2 years: distribution is face and extensor aspects of extremities
 - >2 years: distribution is flexural areas of extremities
- Exacerbated by infections (e.g., *Staphylococcus aureus*), heat, low humidity, chemical irritants, and environmental allergens in some

- Only one-third of patients with severe eczema have foods that consistently exacerbate atopic dermatitis; thus empiric food elimination is not recommended in the treatment of eczema
 - Food elimination for eczema is not recommended due to negative impact on nutrition and increased overall risk of food allergy development

Diagnosis and Evaluation

- Most cases of eczema are diagnosed by history and examination of skin
 - Distribution of eczema usually differs by age (see above)
 - Appearance of eczema can vary depending on skin color
- Biopsy of skin is generally not needed unless eczema is refractory to conservative therapies or other systemic disorder is suspected
- Differential diagnosis:
 - Allergic or irritant contact dermatitis from plants (i.e., poison ivy), metals (nickel-plated jewelry), and chemical products (e.g., perfumes, cleaning products)
 - Immunodeficiency (HIV, Wiskott-Aldrich syndrome, severe combined immunodeficiency, hyper-IgE syndrome)
 - Metabolic disorders (zinc, vitamin B6, or niacin deficiency)

Treatment

- Mainstays of therapy are emollients/moisturizers and topical corticosteroids; choose strength of steroid based on location and severity of eczema
- Daily baths or showers with unscented or mild soap, followed by application of emollient; wet wrap therapy for severe eczema
- Control of itching with oral antihistamines (cetirizine, hydroxyzine)
- Other topical therapies: topical phosphodiesterase-4 inhibitors (crisaborole), calcineurin inhibitors (tacrolimus, pimecrolimus). Refer to Dermatology for cyclosporin, methotrexate, anti-IL-4/anti-IL-13 therapy for severe eczema
- Management of superinfection:
 - *S. aureus* infections of eczema are common
 - Eczema herpeticum: herpes simplex virus infection; treatment with acyclovir

Food Allergies

- See [Chapter 3](#) for more information

Table 4.1 Disorders Associated With Elevated Serum Immunoglobulin E

Allergic disease
 Atopic dermatitis (eczema)
 Tissue-invasive helminthic infections
 Hyperimmunoglobulin E syndromes
 Allergic bronchopulmonary aspergillosis
 Wiskott-Aldrich syndrome
 Bone marrow transplantation
 Hodgkin disease
 Bullous pemphigoid
 Idiopathic nephrotic syndrome

From Table 142-1: Environmental Control of Allergen Exposure. *Nelson's Textbook of Pediatrics*. 20th ed. Elsevier; 2016.



Fig. 4.1 Clinical presentation of urticaria. (Photograph courtesy of Pete Smith, MD, Griffith University, Brisbane, Queensland, Australia.)

immunosuppression (cyclosporin, tacrolimus), biologics (omalizumab)

- Prognosis is good with resolution in up to 50% within 1 year of onset

Hereditary Angioedema (HAE)

Basic Information

- Most often an autosomal dominant disease due to a deficiency in C1-esterase inhibitor, leading to dysregulation of the complement pathway and intermittent episodes of swelling of various body parts
 - Type 1: 85% of patients, quantitative defect in C1-esterase inhibitor
 - Type 2: 15% of patients, qualitative defect in C1-esterase inhibitor

Clinical Presentation

- Tissue swelling (most commonly skin, upper respiratory tract, GI tract) *without* urticaria or pruritus
 - Majority of patients experience abdominal pain
 - Laryngeal swelling can be fatal
- Episodes may be preceded by trauma
- Untreated, symptoms can last for several days

Diagnosis and Evaluation

- Diagnosis based on clinical history and physical exam during episodes

- Labs: C4 as initial screening test (generally decreased when asymptomatic, absent during attacks), C1 esterase inhibitor protein level and function

Treatment

- First-line therapy: replacement of C1 esterase inhibitor is needed
- Second-line therapy (if above not available): fresh frozen plasma
- Symptoms DO NOT improve with antihistamines, steroids, or epinephrine
- In the acute setting of an attack: consult allergist on-call and protect airway, with supportive management for dehydration and pain if present

Mastocytosis

Basic Information

- Two forms:
 - Cutaneous mastocytosis (predominantly children), three subcategories:
 - Urticaria pigmentosa (also maculopapular cutaneous mastocytosis, MPCM), most common form of cutaneous mastocytosis in children
 - Diffuse cutaneous mastocytosis (DCM)
 - Mastocytoma of the skin
 - Systemic mastocytosis (predominantly adults)
 - Majority of patients will have cutaneous involvement

Clinical Presentation

- Cutaneous mastocytosis
 - Eighty percent of patients will have brown or red skin lesions. See Fig. 4.2
 - Darier's sign (wheal or reddening of the skin with mechanical stroking or rubbing). See Fig. 4.3
 - Less than 10% of patients develop systemic symptoms
- Systemic mastocytosis
 - Previously mentioned cutaneous symptoms
 - Systemic symptoms: idiopathic anaphylaxis, flushing, hives, angioedema, diarrhea, fatigue, bone pain, wheezing

Diagnosis and Evaluation

- Cutaneous mastocytosis
 - Clinical diagnosis: skin biopsy if diagnosis is unclear
 - No bone marrow biopsy needed in children
- Systemic mastocytosis
 - Bone marrow biopsy and biopsy of the affected organs
 - Serum tryptase level, KIT D816V mutation analysis

Treatment

- Cutaneous mastocytosis
 - Topical corticosteroids for pruritic lesions
 - Oral second-generation antihistamines for pruritus
- Systemic mastocytosis
 - Management per hematologist

Hypereosinophilia

Basic Information

- Hypereosinophilia: absolute eosinophil counts (AEC) >500 cells/ μ L



Fig. 4.2 Subforms of CM. Cutaneous manifestations in mastocytosis are categorized into MPCM, presenting with disseminated brown lesions (A); DCM, presenting with generalized erythema and thickened skin (B); and mastocytoma, presenting with a brown or red elevated lesion (C). (From Hartmann K, Escribano L, Grattan C, et al. Cutaneous manifestations in patients with mastocytosis: consensus report of the European Competence Network on Mastocytosis; the American Academy of Allergy, Asthma & Immunology; and the European Academy of Allergy and Clinical Immunology. *J Allergy Clin Immunol.* 2016;137(1):35–45. <https://doi.org/10.1016/j.jaci.2015.08.034>.)



Fig. 4.3 Darier's sign. (A–C) A wheal-and-flare reaction develops upon stroking of a CM lesion with a tongue spatula. Darier's sign is a highly specific diagnostic feature of CM. (From Hartmann K, Escribano L, Grattan C, et al. Cutaneous manifestations in patients with mastocytosis: consensus report of the European Competence Network on Mastocytosis; the American Academy of Allergy, Asthma & Immunology; and the European Academy of Allergy and Clinical Immunology. *J Allergy Clin Immunol.* 2016;137(1):35–45. <https://doi.org/10.1016/j.jaci.2015.08.034>.)

- Hypereosinophilic syndrome: hypereosinophilia plus organ dysfunction due to eosinophilia
- Mild, transient eosinophilia common in pediatric population
 - Severity
 - Mild: AEC 500–1 500 cells/ μ L
 - Moderate: AEC 1 500–5000 cells/ μ L
 - Severe: AEC >5000 cells/ μ L
- Primary causes:
 - Hypereosinophilic syndrome (HES): types include myeloproliferative, lymphocytic, overlap (hypereosinophilia with eosinophilic disease of one organ system), familial (autosomal dominant), idiopathic
- Secondary causes (more common in children)
 - Infections: parasites (e.g., strongyloides, toxocariasis, hookworm, scabies), fungal infections (e.g., coccidiomycosis), mycobacterial infection, HIV
 - Allergic disorders:
 - Atopic disease (asthma, atopic dermatitis, allergic rhinitis)
 - Drug-induced, DRESS
 - Allergic bronchopulmonary aspergillosis
 - Eosinophilic disorders
 - Eosinophilic gastrointestinal disease
 - Eosinophilic granulomatosis with polyangiitis
 - Primary immunodeficiencies
 - Autosomal dominant hyper-IgE syndrome (Job syndrome), Omenn syndrome
 - Malignancy-related (leukemia or lymphoma, some solid tumors)
 - Other: autoimmune disorders (e.g., sarcoidosis), adrenal insufficiency, graft-versus-host disease

Clinical Presentation

- Primary HES: usually insidious with skin rash, respiratory (cough, shortness of breath), GI, cardiac and/or neurologic symptoms
- Secondary HES: eosinophilia may be incidental finding during workup for underlying condition

Diagnosis and Evaluation

- Repeat CBC to determine whether eosinophilia is transient, persistent, or rising
- Physical examination and lab testing (e.g., CBC and smear, electrolytes, liver function tests) to evaluate for end-organ involvement
- Primary HES: bone marrow biopsy may be necessary for diagnosis
- Secondary HES: testing for secondary causes is disease specific, will depend on history and physical examination
- IgE may be elevated for certain diseases associated with eosinophilia (see [Table 4.1](#))

Treatment

- Primary HES is treated only if there is end-organ involvement
- Secondary causes are treated for the underlying disorder

Vaginal ring, 755
 Valacyclovir, 758
 Validity, of diagnostic and screening tests, 792
 Value equation, 810
 Variceal bleeding, 213
 Varicella, 77
 Varicella-zoster virus (VZV), 70, 346
 vaccine, 717f–718f, 720
 Varicocele, 700
 Vascular anomalies, 473
 Vascular lesions, 90
 Vascular rings, 616
 Vascular slings, 616
 Vasculitic-appearing rash, 682
 Vasculitides, 682
 Vasculitis, 471
 Vasodilatory shock, 98
 Vasomotor rhinitis, 7
 Vasoocclusive crisis (pain crisis), 288
 Vasopressin, 198
 Vasopressor infusion, 12
 Venooclusive disease (VOD). *See*
 Sinusoidal obstruction syndrome
 Venous hum, 20t
 Ventricular fibrillation, 50
 Ventricular preexcitation, 45, 49f
 Ventricular septal defect, 27
 clinical presentation, 27
 diagnosis and evaluation, 28
 treatment, 28
 Ventricular tachycardia, 45, 46f
 Ventriculoperitoneal (VP) shunts, 489
 Vertical misalignment, 513
 Very-long-chain acyl-CoA (VLCAD)
 deficiency, 385, 386t
 Vesicoureteral reflux, 689, 690f
 antibiotic prophylaxis, 690
 associated anomalies, 689
 grades of, 690f
 male circumcision for, 690
 nuclear renal scan, 689
 physical examination, 689
 Randomized Intervention for
 Children with Vesicoureteral
 Reflux trial, 690
 renal US, 689
 risk factors, 689
 serum laboratory tests, 689
 surgical correction, 690
 urinalysis and urine culture, 689
 voiding cystourethrogram scan, 689–690
 Vestibular migraine, 537
 Vestibular neuritis, 537
 Viral cervical adenitis, 551
 Viral infections, 76
 Viral sialadenitis, 552
 Viral suppression, 481
 Virchow's triad, 298
 acquired causes, 298

Viruses, 345
 coronaviruses, 348
 human papillomavirus, 346
 influenza, 348
 measles, 345
 mumps, 345
 rubella, 346
 varicella-zoster, 346
 Visceral larva migrans, 360
 Vision screening, 724
 Visual perceptual/visual motor deficits,
 731–732
 Visual reinforcement audiometry, 529
 Vitamin B12 deficiency, 291
 diagnosis and evaluation, 291
 treatment, 291
 Vitamin D deficiency, 406
 “dependent” rickets, 188
 hereditary resistance to, 189
 resistance, 406
 rickets, 188
 Vitamin K deficiency, 297–298
 Vitiligo, 88f, 88
 Vitreous hemorrhage, 506
 Vocal cord dysfunction, 600
 clinical presentation, 600
 diagnosis and evaluation, 600
 facts, 600
 treatment, 601
 Vocal cord paralysis, 613
 Vomiting, 206
 pattern, 206
 and regurgitation, 206
 von Gierke disease, 391
 von Willebrand disease, 296
 laboratory results, 296t
 treatment, 296–297
 type 1, 296
 type 2, 296
 type 3, 296
 Vulvar ulcers, 770
 Vulvovaginal candidiasis, 766

W

Waddell's triad, 112
 WAGR syndrome, 277
 Walled-off necrosis (WON), 255
 Warm shock, 98
 Warthin's tumor, 553
 Warts, 79
 Water balance, 402, 403f
 normal physiology of, 198
 Water loss, sources of, 403t
 Water moccasins, bites from, 123
 Water safety, 727
 Weight loss therapy, 619
 Western blot, 76
 Wheezing, 598
 causes, 600t
 facts, 598

White blood cell (WBC) count, 618
 White pupil, 506
 Whole bowel irrigation, 134
 Wide complex tachycardia, 45, 46f
 Williams-Beuren syndrome, 191
 Williams syndrome, 191, 272, 273f
 cardiac defect in, 53t
 Wilms tumor, 490
 heritable conditions associated
 with, 491t
 Wilson disease, 247
 Wiskott-Aldrich syndrome, 6, 295–296,
 306, 312
 Wolffian ducts, 159, 686
 Wolff-Parkinson-White (WPW) pattern,
 45, 49f
 Wolf-Hirschhorn syndrome (WHS),
 279–280
 Worms, 360
 Wounds, 126

X

X-linked adrenoleukodystrophy
 (XALD), 395
 X-linked agammaglobulinemia, 645
 X-linked congenital adrenal
 hypoplasia, 196
 X-linked dominant (XLD) pattern of
 inheritance, 262, 268, 271–272
 X-linked hypophosphatemic rickets, 189
 X-linked ichthyosis, 95t
 X-linked recessive (XLR) pattern of
 inheritance, 262, 263f
 47, XXX syndrome, 267
 47, XXY syndrome, 267
 Xylose testing, 209

Y

Yersinia enterocolitica, 343
Yersinia pestis, 344
 Yuzpe method, 756

Z

Zellweger spectrum, 395
 Zollinger-Ellison syndrome, 218, 252
 Zoonoses
 Bacillus anthracis (anthrax), 344
 Bartonella henselae (cat scratch
 disease), 342
 Brucella, 343
 Chlamydia psittaci (psittacosis), 342
 Coxiella burnetii (Q fever), 344
 Leptospira, 343
 Listeria monocytogenes, 342
 Pasteurella multocida, 343
 Yersinia enterocolitica, 344
 Yersinia pestis (plague), 344
 Z-scores, 705