

儿科学实习手册

盛德乔 王敏 主编



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留学生临床实习指导丛书

Internship
Handbook
of Pediatrics

儿科学实习手册

主 编 盛德乔 王 敏

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留学生临床实习指导丛书

总 主 编 赵 云

编 委 会 盛德乔 龙 兵 简道林

李俊明 王 敏 李志英

李新志 叶 红 查运红

李志芳

前言

随着我国经济社会发展，综合国力提升，国际化已经成为我国高等教育发展的重要战略路径。在高等教育向国际化的迈进中，临床医学来华留学生教育规模不断扩大，质量稳步提高，是发展最快的专业之一。在医学专业人才培养过程中，临床实习教学是十分重要的环节，受到国家教育主管部门和医学院校的广泛重视。为了进一步做实临床实习教学过程，保障教学质量，我们根据国家教育主管部门要求，参照国内外相关专业标准，结合本校临床专业来华留学生教育十多年的经验积累，组织编写了《留学生临床实习指导丛书》。丛书包括内科学、外科学、妇产科学、儿科学四个分册。从便于携带、方便查阅的角度出发，本着内容准确、重点突出、精炼实用的原则，各分册主要涵盖临床上的常见病和多发病，重点描述疾病的诊断、鉴别诊断和治疗要点。

初次进行留学生指导教材编写工作，编著者和编辑们认真细致，尽了最大努力，但因水平有限，时间仓促，书中难免有疏漏和不足之处，敬请读者及同道指正。

赵云
2015.3.29

With China's economic and social development, the comprehensive national strength increases, internationalization has become an important strategic path for the development of China's higher education. Since internationalization of higher education provides constant expansion and steady enhancement for international students, clinical medicine now has ranked one of the fastest-developed majors. As a significant step in the training of international medical students, clinical practice teaching deserves special attention from both Chinese education authorities and medical colleges. In order to enhance the teaching process and ensure the teaching quality, our teaching team compiles this *Clinical Internship Guidance Collection for International Students*. Based on the supervision of the state education authorities, this Collection explores decades' experience of international students' clinical study in our university by reference to professional standards at home and abroad. Easy-to-carry and reader-friendly, this Collection consist of four parts, i.e. internal medicine, surgery, obstetrics & gynecology and pediatrics, with the consideration of content precision, keys highlight and practical use. Each book mainly covers clinical common, frequently encountered diseases, and focuses on disease diagnosis, differential diagnosis and treatment.

Sincere efforts have been made to verify the correctness in the compilation of this Collection for international students. However, some mistakes and errors in this Collection may be unavoidable due to our time and knowledge limit. Thus, we appreciate all your kind criticism and corrections.

Zhao Yun
March 29, 2015

Internship Handbook of Pediatrics

儿科学实习手册

内容简介:

《儿科学实习手册》为来华留学生临床实习指导系列丛书之一，本书由一批具有丰富的教学和临床工作经验的教师队伍完成。本书从实习医师临床工作的角度，介绍了儿科常见病的诊断要点、治疗措施及常用临床操作的方法和规则，重点阐述了基本理论、基本技能和基本操作，并提出了可供医学生进行临床实习参考的指导原则和工作技巧。

本书力求全面、简洁、易于理解。希望本书有利于儿科实习医师职业技能的提高，为将来的临床工作打下坚实基础。同时，也便于临床课学习期的医学生、带教医师及住院医师参考。

Introduction:

The Internship Handbook of Pediatrics is one of *Clinical Internship Guidance Collection for International Students*. It is contributed by clinicians with extensive experience in teaching medical students and residents and in clinical work. It introduces essentials of diagnosis of common diseases, therapeutic measures and clinical operational approaches and in rules of pediatrics from the perspective of clinical practice of interns, and emphasizes on elaboration of basic theories, skills and operations and suggests referable guiding principles and practical skills for clinical practice.

Our goal is to be comprehensive, concise, and reader friendly. We hope this book is helpful for enhancing occupational skills of pediatrics interns and therefore lays a solid foundation for their future clinical work. Meanwhile it is a good reference for medical students who are studying clinical courses, and for clinical teachers and resident doctors.

Contents

Chapter 1: Neonatal Diseases

- Section 1: Infectious Pneumonia of Newborns / 001
- Section 2: Neonatal Hyperbilirubinemia
(Unconjugated Hyperbilirubinemia) / 005
- Section 3: Hemolytic Disease of Newborn (HDN) / 014
- Section 4: Neonatal Sepsis / 020
- Section 5: Neonatal Cold Injury Syndrome / 025
- Section 6: Intracranial Haemorrhage of Newborns / 029
- Section 7: Hypoxic–ischemic Encephalopathy / 037
- Section 8: Respiratory Distress Syndrome / 042
- Section 9: Asphyxia of Newborns / 052
- Section 10: Neonatal Necrotizing Enterocolitis / 060

Chapter 2: Children's Nutrition and Nutritional Diseases

- Section 1: Rickets of Vitamin D Deficiency / 067
- Section 2: Tetany of Vitamin D deficiency / 071
- Section 3: Obesity / 073

Chapter 3: Respiratory System

- Section 1: Acute Upper Respiratory Tract Infections / 075
- Section 2: Bronchitis / 077
- Section 3: Pneumonia / 080
- Section 4: Laryngitis / 082

Chapter 4: Digestive System

Section 1: Acute Gastritis / 083

Section 2: Diarrhea / 085

Section 3: Peptic Ulcer Disease / 087

Chapter 5: Circulation System Diseases

Section 1: Rheumatic Heart Disease (RHD) / 089

Section 2: Congenital Heart Disease / 092

Section 3: Viral Myocarditis / 102

Section 4: Cardiac Arrhythmias / 105

Chapter 6: Urinary System

Section 1: Urinary Tract Infection / 113

Section 2: Acute Glomerulonephritis / 119

Section 3: Nephrotic Syndrome / 124

Section 4: Acute Renal Failure / 129

Chapter 7: Nervous System Diseases

Section 1: Bacterial Meningitis / 135

Section 2: Viral Meningoencephalitis / 141

Section 3: Cerebral Palsy / 146

Section 4: Guillain–Barr é Syndrome / 149

Section 5: Seizures in Childhood / 151

Chapter 8: Blood System Diseases

Section 1: Iron Deficiency Anemia / 162

Section 2: Vitamin B₁₂ Deficiency Anemia / 166

Section 3: Aplastic Anemia (AA) / 169

Section 4: Glucose-6-phosphate

Dehydrogenase (G6PD) Deficiency / 171

Section 5: Idiopathic Thrombocytopenic Purpura / 174

Section 6: Childhood Leukemia / 178

Chapter 9: Endocrine System

Section 1: Precocious Puberty / 184

Section 2: Congenital Hypothyroidism / 189

Section 3: Diabetes Mellitus / 194

Chapter 10: Infectious Diseases

Section 1: Measles / 202

Section 2: Chickenpox/Varicella / 206

Section 3: Roseola Infantum / 208

Section 4: Primary Pulmonary Tuberculosis
and Tuberculous Meningitis / 210

Section 5: Infectious Mononucleosis / 215

Chapter 11: Immunopathy

Section 1: Asthma / 217

Section 2: Anaphylactoid Purpura / 221

Section 3: Juvenile Rheumatoid Arthritis / 225

Section 4: Kawasaki Disease / 230

Chapter 12: Inborn Errors of Metabolism

Phenylketonuria / 234

CHAPTER 1 NEONATAL DISEASES

SECTION 1: INFECTIOUS PNEUMONIA OF NEWBORNS

1. Definition

Infectious pneumonia can be divided into intrauterine infectious pneumonia, intrapartum infectious pneumonia and postnatal infectious pneumonia.

Intrauterine infectious pneumonia: This is due to aspiration of contaminated amniotic fluid or hematogenous spread. It is often associated with obstetric factors, such as amniotic membrane rupture as early as 24 hours or chorioamnionitis contaminated amniotic fluid, or mother is infected during pregnancy.

Intrapartum infectious pneumonia: The disease is caused due to aspiration of contaminated secretion in mother's birth canal, and hematogenous spread due to infected and unclean umbilical.

Postnatal infectious pneumonia: The disease develops due to contact transmission, hematogenous transmission or iatrogenic transmission.

Intrauterine or intrapartum infectious pneumonia: Gram

negative bacilli are the most common infectious bacteria.

Postnatal infectious pneumonia: *Staphylococcus aureus* and *Escherichia coli* are the most common. Many opportunistic pathogenic bacteria such as *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Citrobacter*, anaerobic bacteria and fungi can also cause pneumonia. Pneumonia due to virus infection mostly occurs in late newborns.

2. Diagnosis

2.1 Clinical manifestation:

2.1.1 Intrauterine infectious pneumonia: The clinical manifestations vary greatly. It often outbreaks in 24 hours after the birth. Asphyxia often occur at birth. Shortness of breath, groan and poor response etc. occurs after anabiosis. Moist rales often can be heard in lungs. Respiratory failure, cyanosis and etc. occur in severe cases. Hematogenous infection often lacks the signs of lung, but manifests jaundice, hepatosplenomegaly, feature of meningitis and other multi system involvement.

2.1.2 Intrapartum infectious pneumonia: The pulmonary manifestations such as dyspnea, apnea and pulmonary rales, which can lead to respiratory failure. The onset varies according to the different pathogens. Bacterial infection occurs in 3–5 days after the birth, and may be associated with septicemia. Type II herpesvirus infection may occur in 5–10 days after the birth. The incubation period of chlamydia infection is 3 to 12 weeks. The gastric juice, tracheal secretions and blood etc. should be extracted immediately after the birth and the smear and culture examination are helpful for the diagnosis.

2.1.3 Postnatal infectious pneumonia: Cough, cyanosis, dyspnea and infection toxic symptoms will appear, accompanied by low body temperature, poor response, drowsiness, coma, convulsions, respiratory failure and circulatory failure. Early pulmonary signs are not obvious. Moist rales can appear in the course. Respiratory syncytial virus pneumonia can manifest gasp and pulmonary wheezing etc. Staphylococcus aureus pneumonia may lead to empyema.

2.2 Auxiliary examinations

2.2.1 Chest X-ray: The lung markings increase and thicken. There are focal, segmental or diffuse inflammatory infiltrates. There is no change of X-ray film at the first day of the birth in intrauterine and intrapartum infectious pneumonia. The focus can appear in the late course of disease by dynamic checking; Lung bullae may appear in staphylococcus aureus pneumonia. The lung field opacity reduces combined with air bronchogram in early-onset GBS pneumonia, which is not easy to be distinguished from RDS.

2.2.2 Laboratory examinations: The serum IgM and IgA elevate after the birth, suggesting intrauterine infection; the specific increase of IgM and IgG, which is of more diagnostic value. The positive rate of blood culture is not high. The gastric juice within 1 hour after the birth and tracheal secretions within 8 hours are extracted for smear and culture, which is helpful for etiological diagnosis. The blood gas analysis should be made for the patients with expiratory dyspnea. The blood biochemical examination should be done for the severer patients to understand liver and kidney function, myocardial

enzymes and electrolytes etc.

3. Treatment

3.1 Treatment for respiratory tract: Nebulizer inhalation, postural drainage, periodical turn over, regularly buckle back, sputum suction, aiming to maintain airway patency.

3.2 Oxygen therapy: The nasal catheter, mask, hood, rhinobyon and CPAP can be selected to provide oxygen according to patient's condition. Mechanical ventilation can be used for respiratory failure. The arterial blood PaO_2 should be maintained at 50–80mmHg.

3.3 Antibiotic treatment

3.3.1 Bacterial pneumonia: The antibiotics should be used as soon as possible. After that the antibiotics should be adjusted according to culture results.

3.3.2 Erythromycin 30–40mg/ (kg.d) and Azithromycin 5–10 mg/ (kg.d) are used for Chlamydia and mycoplasma infection. The duration of treatment will last for 2–3 weeks.

3.3.3 α -interferon, acyclovir and Ganciclovir etc. can be used for virus infection.

3.4 Supportive therapy: Balance of water and electrolyte and circulation disorder should be corrected. The intake of liquid should be appropriately limited. The infusion speed should be slow. Plasma, albumin and immunoglobulin should be intravenously infused if necessary.

(杨波 罗军)

SECTION 2: NEONATAL HYPERBILIRUBINEMIA (UNCONJUGATED HYPERBILIRUBINEMIA)

1. Definition

The rate of bilirubin production exceeds elimination result in the increase of the total serum bilirubin (TSB) , which leads to a clinical condition called hyperbilirubinemia. The accumulation of bilirubin (yellow–orange pigment) in the skin, sclera, and mucosa is called jaundice.

Incidence: Neonatal hyperbilirubinemia is a common problem. Approximately 60~70% of term and about 80% of preterm infants develop jaundice in the first week of life. Incidence is higher in the population living at high altitude. Incidence also varies according to ethnicity. It is lower in African Americans and higher in East Asians, Greeks, and Native Americans.

Physiologic ranges of TSB remain controversial because levels are affected by several factors, such as gestational age, birth weight, disease state, degree of hydration, nutritional status, and ethnic background. Data from recent studies suggest that the upper limits of TSB levels (95th percentile) found in diverse populations of normal newborn infants may be as high as 17~18 mg/dL. Previous studies suggest that a typical peak for TSB is 8~9 mg/dL in predominantly breast–fed infants. Preterm infants have no such established “physiologic” bilirubin guidelines.

Exclusion criteria for diagnosis of physiologic jaundice:

1. Jaundice appearing within the first 24 hours of life.
2. TSB level >95th percentile for age in hours based on a nomogram for hour specific serum bilirubin concentration (Figure 1) .
3. Bilirubin level increasing at a rate >0.2 mg/dL/h or >5 mg/dL/d.
4. Direct serum bilirubin level >1.5~2.0 mg/dL or >20% of the TSB.
5. Jaundice persisting for >2 weeks in full-term infants.

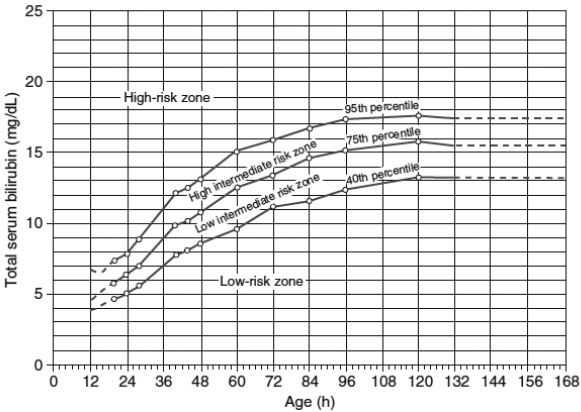


Figure 1 Nomogram for designation of risk in well newborns at ≥ 36 weeks’ gestation with birthweight ≥ 2000 g or ≥ 35 weeks’ gestation with birthweight ≥ 2500 g based on the hour-specific serum bilirubin values. (Reproduced from Bhutani VK, Johnson L, Sivieri EM. Predictive ability of a pre-discharge hour-specific serum bilirubin for subsequent significant hyperbilirubinemia in healthy term and near-term newborns. Pediatrics. 1999; 103:6 - 14.)

2. Clinical Presentation

2.1 Monitor for jaundice: All newborn infants should be monitored routinely for development of jaundice. Each nursery should have an established guideline for the routine assessment of jaundice. Jaundice is visible when the serum bilirubin level approaches 5~7 mg/dL.

2.2 History: Family history of jaundice, anemia, splenectomy, or metabolic disorder is significant and may suggest underlying etiology for jaundice. Maternal history of infection or diabetes may increase the newborn's risk for jaundice. Breast-feeding and factors affecting normal gastrointestinal function in the newborn period increase the tendency for more severe jaundice.

2.3 Physical examination: Signs of bleeding such as cephalhematoma, petechiae, or ecchymoses indicate blood extravasations. Presence of hepatosplenomegaly may signify hemolytic disease, liver disease, or infection. Physical signs of prematurity, plethora with polycythemia, pallor with hemolytic disease, and big infants with maternal diabetes all can be associated with jaundice. Omphalitis, chorioretinitis, microcephaly, petechiae and purpuric lesions suggest infectious causes of increased serum bilirubin.

2.4 Neurologic examination. Severe hyperbilirubinemia can be toxic to the auditory pathways and to the central nervous system, which can result in hearing loss and encephalopathy. The appearance of subtle abnormal neurologic signs heralds the onset of early bilirubin encephalopathy. Clinical signs may include lethargy, poor feeding, vomiting,