

Synopsis of
MEDICINE
IN DENTISTRY

LAWRENCE COHEN

SECOND
EDITION

A Synopsis of MEDICINE in DENTISTRY

LAWRENCE COHEN

Ph.D., M.D., D.D.S., F.D.S.R.C.S. (Eng.), F.A.C.D.

*Chairman, Department of Dentistry, and Attending Physician, Illinois
Masonic Medical Center; Professor, Department of Oral Diagnosis
and Adjunct Professor of Dermatology, University of Illinois,
Chicago, Illinois*

Second Edition



Lea & Febiger

Philadelphia

1977

Library of Congress Cataloging in Publication Data

Cohen, Lawrence.

A synopsis of medicine in dentistry.

Bibliography: p.

Includes index.

1. Internal medicine. 2. Dentistry. I. Title. II. Title: Medicine in dentistry. [DNLM: 1. Dentistry. 2. Internal medicine. WB100 C678] RC46.C58 1977 616'.002'46176 77-24482 ISBN 0-8121-0608-3

Copyright © 1977 by Lea & Febiger. Copyright under the International Copyright Union. All rights reserved. This book is protected by copyright. *No part of it may be reproduced in any manner or by any means without written permission from the publisher.*

Published in Great Britain by Henry Kimpton Publishers, London

PRINTED IN THE UNITED STATES OF AMERICA

A Synopsis of
MEDICINE
in
DENTISTRY

*To my Wife, Gloria, and Sons, Alan, Martin and David
for their patience and forbearance during
the writing of this book*

PREFACE

The response to the first edition of this book was gratifying and prompted an extensive revision for a second edition. My objective in writing both editions is to provide the dental student with the fundamentals of internal medicine in a succinct form.

The importance of general dental practice residencies in hospitals has now been recognized. Many dental residents rotate through anesthesiology and internal medicine and are exposed to a variety of techniques and procedures for the first time. Therefore, I have amplified several topics in this second edition to explain the scientific bases for some important areas of medicine, such as shock, arterial blood gases and acid base balance.

It is routine now in the United States for paramedics to provide Advanced Cardiac Life Support. This involves intubation, interpretation of electrocardiographs and the administration of drugs by the intravenous route. Because electrocardiography plays such an important role in the diagnosis of cardiac disease, I have included an introductory section on some of the common cardiac arrhythmias.

I wish to thank Miss Susan Zimmerman, medical illustrator, Illinois Masonic Medical Center, for her excellent drawings. In addition, I thank my secretary, Celeste Johnson, for typing the manuscript.

Chicago, Illinois

LAWRENCE COHEN

CONTENTS

CHAPTER	PAGE
1 INFECTIOUS DISEASES.	1
2 ALLERGY AND IMMUNE REACTIONS	19
3 DISEASES OF THE RESPIRATORY SYSTEM	31
4 DISEASES OF THE CARDIOVASCULAR SYSTEM	49
5 DISEASES OF THE KIDNEY	79
6 DISEASES OF THE GASTROINTESTINAL TRACT	86
7 DISEASES OF THE BLOOD	109
8 DISEASES OF THE LYMPH NODES	135
9 DISEASES OF THE SALIVARY GLANDS	141
10 DISORDERS OF NUTRITION	146
11 ENDOCRINE DISORDERS	159
12 DISEASES OF THE BONES AND JOINTS	185
13 DISEASES OF THE NERVOUS SYSTEM	199
14 THE MEDICAL ASPECTS OF HEAD INJURIES	223
15 SOME DRUGS USED IN THERAPY	228
APPENDIX	237
INDEX	241

CHAPTER 1

INFECTIOUS DISEASES

Infectious diseases may be classified according to the etiologic agent: bacterial, viral, spirochetal and fungal. Some of the infectious diseases are discussed in other sections of this book: for example, the common cold, influenza and tuberculosis are reviewed under *Respiratory Diseases*.

Various terms will be used in the discussion of infectious diseases, a few of which follow. The incubation period, for example, is the time which elapses between the invasion of the tissues by the infecting organism and the onset of the clinical symptoms.

During the course of some of these infections a rash appears on the skin (exanthem) and in some of them an eruption appears on the mucous membranes (enanthem). In describing a skin rash the expressions below are used:

Erythema. A diffuse reddening of the skin.

Punctate erythema. Small points of redness.

Macule. A circumscribed discoloration of the skin which is not raised above the surface of the surrounding skin.

Papule. A small raised area which can be felt with the fingers.

Vesicle. A small blister occurring on the skin or mucous membranes.

Bulla. A large blister occurring on the skin or mucous membranes.

Pustule. A small elevation of the skin containing pus.

BACTERIAL INFECTIONS

Streptococcal Infections

Infections with Group A β -hemolytic streptococci include streptococcal tonsillitis or pharyngitis, scarlet fever, impetigo and erysipelas. The

incubation period is two to four days. The sequelae of streptococcal infections are rheumatic fever and acute glomerulonephritis.

Group A hemolytic streptococci contain group A carbohydrate, type-specific M protein, streptokinase, deoxyribonuclease, streptolysin S and O, hyaluronidase and erythrogenic toxin. Antistreptolysin O is present in high titer in the serum of patients recovering from a recent streptococcal infection.

DICK TEST. This is performed with an intracutaneous inoculation of 0.1 ml. of a standardized dilution of erythrogenic toxin. The reaction is read in 24 hours and is positive if the local erythema measures 1 cm. or more in diameter. A positive reaction indicates absence of antitoxin or susceptibility to scarlet fever; a negative reaction indicates neutralization of toxin by antitoxin, or immunity to scarlet fever.

STREPTOCOCCAL TONSILLITIS

Streptococcal tonsillitis differs from scarlet fever only in that the latter is due to infection with an erythrogenic strain of the organism in a nonimmune person. The erythrogenic toxin is responsible for the punctate erythematous rash and the characteristic enanthem.

SCARLET FEVER

The disease is ushered in abruptly by fever, vomiting, sore throat, and such constitutional symptoms as headache, chills, and malaise. Within 12 to 48 hours after onset, the typical rash appears.

The significant objective findings are the fever, enanthem and exanthem.

The typical enanthem consists of markedly red and edematous tonsils and stippling of the palate. The tongue is also coated with white fur through which the enlarged papillae stand out—the so-called “white strawberry tongue.” When the white fur peels off, the tongue has the appearance of a red strawberry. The cheeks are flushed and there is circumoral pallor. The exanthem follows within 12 to 48 hours and consists of a fine punctate erythema.

LABORATORY DIAGNOSIS. A sterile swab is rubbed over each tonsillar area and the posterior pharynx. To avoid contaminating the swab, the tongue or lips are not touched. The organism is grown on blood agar, and typically clear hemolysis occurs around the colonies after 18 to 24 hours' incubation.

IMPETIGO

Impetigo is characterized by superficial purulent crusting lesions of the skin and may be caused by either hemolytic streptococci or staphylococci.

The vesicular stage of impetigo may be transient. The typical lesion is a thick, adherent amber-colored crust.

ERYSIPELAS

Erysipelas is characterized by a red indurated thickening of the skin with a raised, firm margin. The skin lesion is usually associated with fever and constitutional symptoms.

TREATMENT. Penicillin is the drug of choice for the Group A β -hemolytic streptococcal infections and is usually given for 10 days to prevent acute glomerulonephritis. In penicillin-sensitive patients erythromycin is given.

Diphtheria

Diphtheria is caused by *Corynebacterium diphtheriae* and has an incubation period of two to four days. The organism has no invasive power and the threat to life results entirely from the effect of the toxin on the heart, nerves and adrenals.

CLINICAL FEATURES. The local lesion of diphtheria on the mucosa of the pharynx, larynx or trachea is characterized by the formation of a false membrane. The organism causes surface necrosis of the mucosa which combines with the fibrinous exudate to form a "membrane" which is strongly adherent to squamous epithelium but is loosely attached to ciliated epithelium. The exudate on the tonsil may resemble a streptococcal or Vincent's infection. Most diphtheritic lesions have a characteristically offensive pungent odor. There is congestion and edema of the surrounding structures. Periadentitis involving the connective tissue around the lymph nodes gives rise to the classical "bull-neck" of diphtheria.

Diphtheria toxin may affect the myocardium causing toxic myocarditis between the eighth and tenth day; the first signs are tachycardia and an irregular pulse. The motor nerves may be affected producing palatal, pharyngeal, laryngeal and ocular paralysis. Kidney involvement may cause albuminuria and in severe cases renal failure.

TREATMENT. Diphtheria antitoxin is given as early in the disease as possible.

PROPHYLAXIS. Active immunization will prevent diphtheria and is normally effected in children by three injections of alum precipitated toxoid at one-month intervals. It is usual to give a combined triple vaccine (DPT) against diphtheria, pertussis (whooping cough) and tetanus.

The Schick test is used to detect whether a person has immunity against diphtheria. The test is performed by injecting into the skin of the forearm 0.1 ml. of diluted highly purified diphtheria toxin. The development of a variable area of redness at the site of inoculation over a period of 72 to 120 hours signifies a positive reaction and indicates that the patient is susceptible to the disease. A negative Schick test signifies that the subject is unlikely to contract clinical diphtheria.

Whooping Cough (Pertussis)

Whooping cough is caused by *Bordetella pertussis* and has an incubation period of seven to ten days. It is one of the most serious of the acute specific fevers of childhood.

CLINICAL FEATURES. After what appears to be a common cold (catarrhal stage), a harsh cough develops and becomes progressively more severe. Paroxysms of coughing (during which a series of explosive coughs occurs) are followed by a prolonged inspiration through a partly closed glottis. This gives rise to a characteristic "whoop." Following a series of paroxysms the child often vomits or coughs up thick mucus. During a spasm of coughing the child may abrade the lingual frenum against the lower incisors thus causing a frenal ulcer, or he may rupture a blood vessel in the conjunctiva giving rise to a subconjunctival hemorrhage. In the respiratory tract bronchopneumonia and areas of collapse of the lung (atelectasis) may occur which in later life may result in bronchiectasis.

LABORATORY DIAGNOSIS. At the end of the catarrhal stage, white blood cell counts of 20,000 to 30,000 per cubic millimeter, with 60 percent or more lymphocytes, are suggestive of the disease. *B. pertussis* is best isolated from the nasopharynx by means of the nasopharyngeal swab. The typical colonies can be cultured on Bordet-Gengou medium containing penicillin.

TREATMENT. Available evidence suggests that pertussis immune globulin may exert a beneficial effect on infants with severe whooping cough. Tetracycline, chloramphenicol, streptomycin and erythromycin have been used independently or in conjunction with pertussis immune globulin. The child should be fed immediately after he has vomited to prevent weight loss.

PROPHYLAXIS. Active immunization with triple (DPT) vaccine will prevent an attack.

Tetanus

The etiologic agent of tetanus is an anaerobic spore-forming rod, *Clostridium tetani*. The clinical manifestations of the disease result from the activity of a soluble exotoxin, tetanospasmin, which is elaborated at the site of injury by vegetative forms of the organism.

The incubation period of tetanus in human beings is usually 3 to 21 days.

CLINICAL FEATURES. There are three clinical forms of tetanus: generalized, cephalic and local. *Local tetanus* is characterized by persistent, unyielding rigidity of the group of muscles in close proximity to the site of injury. This form of the disease has a fatality rate of about 1 percent.

Generalized tetanus is the most common form of the disease. Trismus is the presenting symptom and sign in over 50 percent of cases. Tonic contractions of the muscles of jaws, face, neck, back and abdomen are common. The typical tetanic seizure is characterized by a sudden burst of tonic contraction of muscle groups causing opisthotonos, flexion and adduction of the arms, clenching of the fists on the thorax and extension of

the lower extremities. The patient is completely conscious during such episodes and experiences intense pain.

Cephalic tetanus is the rarest form and has an incubation period of only one or two days. It follows injuries of the head or otitis media and the prognosis is extremely poor. Dysfunction of the cranial nerves occurs and the seventh nerve is affected most often.

The average death rate of tetanus is 45 to 55 percent.

TREATMENT. Human immune globulin in intramuscular doses of 3000 to 6000 U. is the antitoxin now used.

Penicillin kills the vegetative forms of *Cl. tetani*. The administration of 1,200,000 U. of procaine penicillin once daily, or 1,000,000 U. of penicillin G intramuscularly every six hours for ten days, is recommended in all cases of tetanus. Tetracycline, 2 gm. per day, may also be used.

A variety of drugs have been used to control seizures including barbiturates, chlorpromazine, meprobamate and diazepam (Valium).

PROPHYLAXIS. Active immunization is achieved by means of either alum-precipitated or fluid toxoid. Three injections of the alum preparation, preferably four weeks apart, are given. Fluid toxoid is used only for booster doses when rapid protection is indicated. Booster doses should be repeated every ten years. If an injury has occurred in which there is a possibility of infection with *Cl. tetani* and no toxoid has been given for more than one year, another dose is necessary. Reactions to toxoid occur occasionally but generally are not serious; anaphylaxis may occur rarely.

To protect a patient who has never been actively immunized against tetanus and has sustained a threatening injury, human immune globulin, 250 to 350 U., is injected. At the same time but at a different site, purified fluid toxoid is also given for active-passive immunization.

VIRAL INFECTIONS

Measles (Rubeola, Morbilli)

Measles is caused by a paramyxovirus and is highly contagious. The disease has a tendency to occur in epidemics every second year in the late winter and early spring.

CLINICAL FEATURES. After an incubation period of 10 to 14 days the disease begins with fever, sneezing, running nose, red eyes and a cough. In over 90 percent of the patients pinhead-sized, grayish spots on an erythematous background appear on the buccal mucous membrane in the region of the molar teeth. Known as Koplik's spots, these areas constitute the enanthem; their number varies from only a few to many such spots.

On the third day the skin rash appears behind the ears at first, and then it spreads to the face and trunk. Usually a mild general lymphadenopathy may be noted. The rash from the onset is maculopapular, with round or oval lesions which tend to become confluent and give the characteristic blotchy appearance of measles. As the rash fades it leaves a brownish stain-

ing of the skin associated with desquamation and the temperature falls to normal. Complications of measles are most often due to secondary bacterial infection and include otitis media and bronchopneumonia.

TREATMENT. There is no specific treatment for measles. The child is kept in bed, and any complications are treated with the appropriate antibiotic.

PROPHYLAXIS. Active immunization is obtained by the injection of measles vaccine at 12 months of age or older. Passive immunization is usually conferred by giving gamma globulin within the first few days after exposure has occurred.

Rubella (German Measles)

Rubella occurs mainly in children and adolescents. The incubation period is 12 to 23 days.

CLINICAL FEATURES. German measles begins with fever, catarrh and conjunctivitis and is followed within 24 hours by a generalized, pinkish macular rash. The latter appears first on the forehead and behind the ears and then spreads over the rest of the body. Characteristically the posterior cervical lymph nodes are enlarged. A few petechiae may occur on the soft palate. The disease is usually mild and self-limiting.

Twenty percent of the children born to mothers who have had rubella in the first trimester of pregnancy have congenital defects such as cataract, congenital heart disease, deafness and mental retardation.

DIAGNOSIS. Leukopenia is frequently found in rubella. The virus may be recovered from throat washings within five days after the onset of the rash.

TREATMENT. Treatment is symptomatic.

PROPHYLAXIS. Living vaccines containing virus attenuated by serial passage in tissue culture are injected subcutaneously and will protect against rubella. In the last few years a single subcutaneous injection of a combined vaccine containing live rubella, measles and mumps viruses has been used to protect against all three diseases.

Herpesvirus Infections

The herpesviruses include the causative agents of herpes simplex and the varicella-zoster virus, the causative agent of chickenpox and herpes zoster.

HERPES SIMPLEX

Two varieties of herpes simplex virus (HSV) exist, type 1 and type 2. Type 1 is associated with oral infections and most commonly causes keratitis and herpetic encephalitis. Type 2 is now the third most common venereal disease in the United States, following gonorrhea and syphilis. There is a close association between type 2 HSV infections of the cervix and cervical cancer.

Genital type 2 and less commonly type 1 infections in pregnant women have been found to be the major sources of virus to the newborn. Types 1 and 2 HSV appear to be transmitted by kissing and sexual intercourse.

Primary Herpetic Infections

These may involve the skin, mucous membranes, conjunctivae or the central nervous system and are usually severe. Eczema herpeticum (Kaposi's varicelliform eruption) is a primary manifestation of herpesvirus infection of the skin of a patient with eczema.

The incubation period for either primary HSV-1 or HSV-2 infection ranges from two to 20 days, with an average of around six days.

Acute Herpetic Stomatitis

Acute herpetic stomatitis usually occurs in children between six months and five years of age but may also be seen in adolescents and adults. Crops of vesicles occur throughout the oral cavity including the gingiva. These vesicles soon rupture, leaving ulcers which become secondarily infected. Typically the child is febrile and cannot eat or drink because of the sore mouth. Healing of the ulcers occurs within 10 to 14 days.

LABORATORY DIAGNOSIS. Provided the lesions have been present for less than two to three days, the typical multinucleated giant cells and giant nuclei are seen; intranuclear inclusions are rare.

TREATMENT. Local applications of 5-iodo-2'-deoxyuridine (IDU) have been used in the treatment of herpetic infections, as IDU interferes with the metabolism of the virus. It has not been as successful as was anticipated. Antibiotics are given to control secondary infection. A soft diet with plenty of liquids is of value. Fruit juices, because of their acidity, cause pain in the early stages of the disease and should be avoided.

Recurrent Herpetic Infections

It has recently been shown that herpes simplex virus lies latent in sensory ganglia and that virus replication can be activated by fevers, exposure to sunlight or menstruation. The recurrent lesions tend to occur around mucocutaneous junctions and are thus found around the lips and nose (herpes labialis or herpes febrilis). The patient experiences a burning sensation in the area, and at the site of irritation a number of thin-walled vesicles appear. These soon rupture and a scab forms which heals without scarring within the next seven days. The lesions tend to reappear at the same site. Recurrent herpetic lesions may also occur intraorally.

TREATMENT. There is no specific treatment. Stoxil ophthalmic ointment applied very early on the lip lesions of herpes labialis frequently aborts the lesions. An antibiotic ointment such as Aureomycin can be applied to reduce secondary infection.

VARICELLA (CHICKENPOX)

Chickenpox is an acute infectious disease caused by a virus which is identical with the virus of herpes zoster. The incubation period is 13 to 17 days. The onset is sudden with fever and the rash appearing together. In children the systemic symptoms are usually mild and complications are rare, but in adults the disease may be more severe.

CLINICAL FEATURES. The eruption begins as small, discrete, irritant red papules which in a few hours turn into thin-walled vesicles containing clear fluid. The vesicles become pustular within 48 hours. During the next two days they dry up to form scabs which soon fall off. The fever usually subsides within two to three days of the onset.

Characteristically the vesicles appear first on the back and chest and then on the lower trunk where they are more numerous than on the face and extremities. The lesions occur in crops so that papules, vesicles and crusts are all present at the same time. This distinguishes chickenpox from smallpox where the lesions are all at the same stage. The oral mucous membrane is frequently affected, and the lesions are rather similar to those of herpes simplex.

LABORATORY DIAGNOSIS. Multinucleated giant cells and inclusion bodies similar to herpes simplex and herpes zoster are found on exfoliative cytologic examination.

TREATMENT. Calamine lotion is used to prevent itching. A phenolic mouthwash is soothing when oral lesions are present.

HERPES ZOSTER (SHINGLES)

In herpes zoster the virus affects a posterior root ganglion, the gasserian ganglion or the geniculate ganglion. The incubation period is short. Within three to seven days after exposure to the virus, the patient experiences a continuous burning pain in the area of the skin supplied by the sensory nerves involved. Two or three days later crops of vesicles appear, and the surrounding skin is inflamed and edematous. The majority of cases occurs in the thoracic area. The rash is almost invariably unilateral.

Herpes Zoster of the Gasserian Ganglion

The maxillary division of the fifth nerve and the mental nerve may be involved by herpes zoster. In both cases, intraoral vesicles are present in the distribution of the involved nerve (Figure 1.1).

Ophthalmic and Geniculate Herpes (p. 210)

TREATMENT. There is no specific treatment. Analgesics are used to control the pain. Antibiotic creams may be used to prevent secondary infection.



Figure 1.1. Herpes zoster. Before the lesions appeared, the patient experienced pain for 24 hours at the site of the skin eruption. The lesions which are now crusted are confined to the mental nerve distribution. Male, aged 29 years.

Smallpox (Variola)

Smallpox is caused by the variola virus, one of the poxviruses. The poxviruses that affect man are the variola, vaccinia, cowpox and molluscum contagiosum viruses.

The variola virus is carried from patient to patient in infected skin secretions or contaminated dust. When inhaled, it reaches the upper respiratory tract where it multiplies in the lymphatic tissues. The incubation period is seven to thirteen days.

CLINICAL FEATURES. Smallpox is ushered in by a high fever (102° – 103°) associated with severe toxemia. On the third or fourth day numerous lesions appear on the oral and pharyngeal mucosa, face and hands and then spread to the trunk and lower limbs. The trunk is involved to a variable extent, but the axillae and groins are spared. This distinguishes the rash from chickenpox which is maximal over the trunk and the lesions of which come out in crops.

The eruption in smallpox begins with macules which change into papules, then into vesicles and finally pustules over a period of four to seven days. In the late vesicular stage of smallpox depressed, “umbilicated” vesicles are seen. The pustulation and the fever which occur during the latter part of the eruptive stage are due to secondary bacterial infection and can be controlled to some extent by the use of antibiotics.

Variola minor (alastrim) is a milder form of smallpox and the mortality rate is much lower than in classical smallpox (variola major).

LABORATORY DIAGNOSIS. Serologic tests are employed in the diagnosis of smallpox but are not as reliable as growing the virus on the chorio-allantoic membrane of a chick embryo where the typical pocks appear within seventy-two hours.

TREATMENT. Treatment is largely symptomatic. Antibiotics are given to control secondary infection.

PROPHYLAXIS. Vaccination with the vaccinia virus will prevent smallpox and is employed in the prevention of epidemics. Methisazone, an antiviral compound, has also been used in the prophylaxis of smallpox. Recent figures released by the World Health Organization indicate that smallpox has been virtually eradicated worldwide.

Mumps (Epidemic Parotitis)

Mumps is caused by the mumps virus, a member of the myxovirus group and is spread by inhalation of droplets. The incubation period is eighteen to twenty-one days. Children and adolescents are most commonly affected.

CLINICAL FEATURES. There is painful swelling of one parotid gland, and within twenty-four hours the other gland enlarges. Mumps is most often bilateral but may be unilateral (Figure 1.2). The submandibular and sublingual salivary glands are occasionally involved. An early sign of mumps is redness of the parotid papilla, but no discharge can be expressed from the parotid duct. The incidence of involvement of testes, pancreas, breast and ovaries increases with age. The fever usually subsides in three to five days and the patient makes a complete recovery in the next seven to ten days.

LABORATORY DIAGNOSIS. Complement fixing antibodies are produced and are of two types: the S antibody, which appears rapidly during the

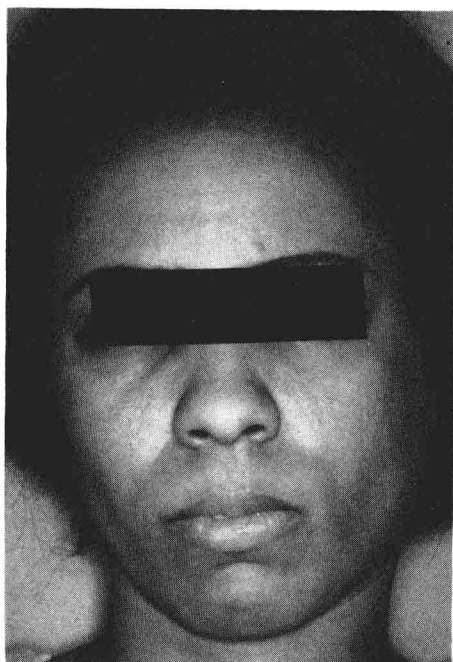


Figure 1.2. Unilateral mumps. The patient complained of pain and swelling in the left parotid region for the previous 24 hours. Clear saliva was expressed from the left parotid duct thus excluding a bacterial infection of the gland. The S antibody was raised, establishing a diagnosis of mumps. The right parotid gland did not enlarge. Female, aged 29 years.