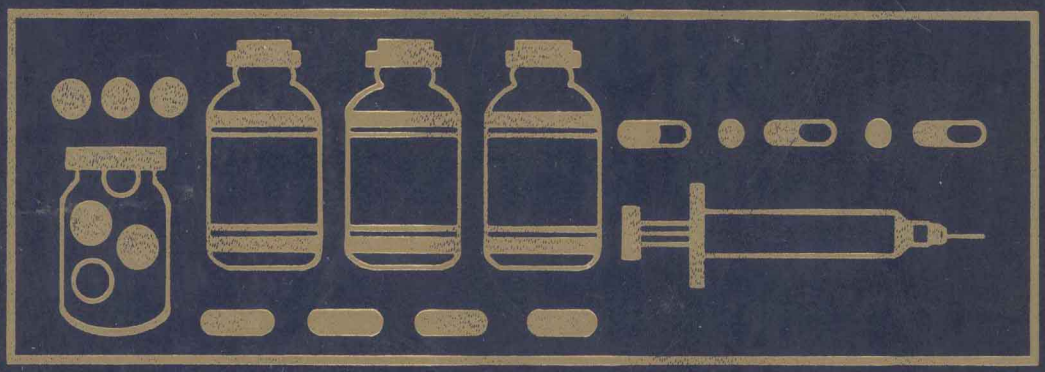
Drugs and Nursing Implications

Edition 4



GOVONI & HAYES

Edition
4

Drugs and Nursing Implications

LAURA E. GOVONI Ph.D., R.N.

Lecturer and Consultant, Medical-Surgical Nursing;
Formerly Professor, Graduate Division, University
Connecticut School of Nursing

JANICE E. HAYES Ph.D., R.N.

Professor, Graduate Division, University of Connecticut
School of Nursing

ACC Appleton-Century-Crofts/Norwalk, Connecticut

NOTICE. Our knowledge in the clinical sciences is constantly changing. As new information becomes available, changes in treatment and in the use of drugs become necessary. The authors and the publisher of this volume have, as far as it is possible to do so, taken care to make certain that the doses of drugs and schedules of treatment are correct and compatible with the standards generally accepted at the time of publication. The reader is advised to consult carefully the instruction and information material included in the package insert of each drug or therapeutic agent before administration in order to make certain that the recommended dosage is correct and that there have been no changes in the recommended dose of the drug or in the indications or contraindications in its utilization. This advice is especially important when using new or infrequently used drugs.

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Drugs and Nursing Implications

PREFACE

Since the publication of the last edition, knowledge about pharmacology and the dynamics of drug use has undergone a quantum increase. Lay literature focusing on drug therapy has proliferated in response to an alerted and concerned public. During the same period, the scope of nursing functions and responsibilities has continued to expand, placing great demands on the practitioner to be well informed about current therapies and capable of translating this knowledge into safe, effective nursing action. The natural outgrowth of increased available information about drugs is the more comprehensive discussion of nursing implications in pharmacotherapy recorded in this edition.

The following areas of information (from which many nursing implications are derived) have also been expanded: 1) pharmacokinetics: drug half-life, protein binding, dynamics of absorption and excretion, drug-receptor relationships; 2) variations in drug response imposed by both immaturity and aging; 3) expected and potential drug-drug and drug-food interactions; and 4) drug interferences with diagnostic tests and procedures.

Additional features of this edition include: 1) expansion of the sections on absorption and fate which provide significant information to planners of the dose schedules; 2) increased numbers of trade names for each drug (in response to regional differences in drug availability); 3) arrangement of adverse reactions for most drugs by functional or systemic constellations to facilitate observation and interpretation; 4) the listing of fixed combination drugs that incorporate the agent under discussion; 5) an increased number of specifically designed nursing interventions (see Index) related to particular pharmacotherapeutic regimens, and 6) expanded data bases for informed observations, analysis, and prediction of drug response (e.g., RDA, clinical signs and symptoms, diagnostic tests, normal laboratory values).

The reader is reminded that clinical studies of patient response to drugs and new laboratory evidence often result in dose changes. The authors have made every effort to provide accurate information about dose regimens; however, we advise constant checking of product information supplied with the drug when dispensed and/or other up-to-date authoritative sources of drug information before administering a drug, particularly if it is infrequently used or is a new product.

Several points should be made about the use of this edition:

1. Referral to a prototype drug for more detailed discussion includes the assumption that listed drug-drug interactions and diagnostic test interferences apply to both prototype and referred drug(s), unless specified otherwise.
2. Although alphabetical sequence prevents arrangement of drugs by class, the reader may locate the drugs within a given group or class by using the Index.
3. The reader is reminded that the drug may be listed in the Index in four ways: by generic name, by trade name, by pharmacologic action, and by chemical classification.

The user of this volume is provided with concise, scholarly, and professional suggestions meant to assist in the design and implementation of interventions in pharmacotherapy, reinforce the development of nurse-patient relationships based on mutual understanding about the drug regimen, and permit the adaptation of drug therapy to meet the particular needs of the patient.

In conclusion, we have found that the writing of the fourth edition has mandated not only constant study and analysis of reported clinical findings in nursing and in pharmacotherapy but also the acquisition of new scientific background in pharmacology and related disciplines in order to identify the critical elements basic to nursing interventions in drug therapy.

Comments, queries, and suggestions from our readers are always welcomed to help provide accuracy and relevance in future editions.

ACKNOWLEDGMENTS

As with previous editions, many individuals, including students, professional colleagues, and friends from the lay public have influenced the preparation of this edition. Again, we are most grateful and professionally proud to have had the expert assistance of Paul Pierpaoli and Dr. Patricia Mullins. Their critical reading of drug descriptions and resultant comments and suggestions were invaluable. We are also grateful to Alex Cardoni, Dr. Henry Palmer, and Sheldon Sones for their generous help and willingness to provide documented answers to our questions at a moments notice.

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R.N., M.S.; Assistant Professor, School of Nursing, University of Southern Maine; *Abraham Kurien*, M.D., Chief of The Cardiology Service, Manchester Memorial Hospital, Ct.; *Diane LaRochelle*, R.N., Ph.D., Assistant Professor and Associate Dean, School of Nursing, University of Connecticut; *Barbara Lindberg*, R.N., Assistant Head Nurse, Coronary Care Unit, St. Francis Hospital and Medical Center Foundation, Hartford, Ct.; *Edmund Lowrie*, M.D., Senior Vice President, National Medical Care, Inc., Boston, Mass.; *Ernestine Lowrie*, R.N., Vice President of Clinical Services, National Medical Care, Inc., Boston, Ma.; *Elizabeth Luginbuhl*, R.N., M.S., Pediatric Nurse Coordinator, Hartford Hospital, Ct.; *Alberta R. Macione*, R.N., Ph.D., Assistant Professor, Department of Nursing, University of Massachusetts; *Donna Marrero*, R.N., Assistant Head Nurse, Hartford Hospital, Ct.; *Cynthia Meinsen* R.N., B.S.N., Nurse Clinician, Department of Cardiac Rehabilitation, St. Francis Hospital and Medical Center Foundation, Hartford, Ct.; *Howard Meridy*, M.D., Associate in Anesthesiology, Hartford Hospital, Ct.; *Patricia Mullins*, Pharm.D, Pharmacist, Drug Information Center, Medical College of Virginia; *Henry A. Palmer*, Ph.D., Clinical Professor, Assistant Dean for Clinical Affairs, School of Pharmacy, University of Connecticut; *Paul Pierpaoli*, R.Ph., Director, Department of Pharmacy Services, Associate Professor Pharmacy, Medical College of Virginia; *Tina Puia*, Certified Ophthalmic Assistant, Eye Physician Associates of Hartford, Ct.; *Marie Roberto*, R.N., M.S.N., Director, Division of Community Nursing and Home Health, State Department of Health Services, Ct.; *Susan Safrino*, R.N., B.S.N., Cardiac Nurse Specialist, Cardiology Department, The Mount Sinai Hospital, Hartford, Ct.; *Claudia Schmalenberg*, R.N., M.S.N., Assistant Vice President for Clinical Practice, The Mount Sinai Hospital, Hartford, Ct.; *Ijaz Shafi*, M.D., Assistant Clinical Professor in Ophthalmology, University of Connecticut School of Medicine; *John R. Shepherd*, D.D.S., Periodontist, West Hartford, Ct.; *Sheldon Sones*, R.Ph., Director of Pharmacy, Meriden Memorial Hospital, Ct.; *Anne Symecko*, R.N., Supervisor Nursing Service, St. Francis Hospital and Medical Center Foundation, Hartford, Ct.; *Helen Valentine*, R.N., M.S., Vernon, Ct.; *William G. Wilcox*, D.M.D., Elmwood, Ct.

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LAURA E. GOVONI
JANICE E. HAYES
1982

PREFACE TO THE THIRD EDITION

In this completely new edition of *Drugs and Nursing Implications*, over 200 new drugs have been added, and the number of prototypes has been increased and more fully described. This edition also contains several new sections. Both format and style that students and teachers found useful in former editions have been retained, i.e., alphabetical sequence according to generic names, and use of prototypes as major sources of information about drug groups. Prototypal organization places emphasis on the fact that drugs related by chemical and structural similarities often share mechanisms of action and other pharmacologic properties. When a drug is referred to the prototype, it shares Nursing Implications, Laboratory Test Interferences, and Drug Interactions, unless otherwise noted. Differences in the congeneric and second order compounds are described briefly and include particularized nursing implications.

With each edition, our conviction grows that involvement in the total pharmacotherapeutic regimen is one of the most challenging responsibilities of the nurse. Each activity related to drug therapy is important; all aspects of safe, effective treatment beginning with the scheduling of doses and continuing through the analysis of response to the drug regimen as a base for patient teaching require the ability to integrate knowledge about pharmacodynamics into all phases of the nursing process.

Interdigitation of drug therapy with the coexisting nursing regimen requires a data bank of relevant drug information; such a resource is presented in the sections preceding nursing implications. Data about drug effects reflect increased information about what happens at the cellular and molecular levels as well as at the organ level. Information about drug entry, transport, biotransformation, and excretion has been expanded. The reader also is alerted to time factors (i.e., peak action periods, duration of effect, drug half-life) that suggest administration schedules and indicate periods of potential danger from overdose or toxicity. Sections related to limitations of drug use incorporate new knowledge about drug passage across cell membranes (passage into breast milk, across the blood-brain barrier, and the placenta), the effect of age on drug response, and the problems of multiple drug therapies. All of these sections provide clinically oriented information useful in the design of nursing-pharmacotherapeutic regimens.

When feasible, adverse reactions (allergy, sensitivity, toxicity, untoward effects) have been grouped according to body system or to clusters of reactions. All have been reported in clinical research literature and their multiplicity places a very real burden on the designer of patient care who cannot chance ignorance if drug-induced diseases or conditions are to be prevented.

New features of the third edition deal with drug interactions, drug-induced interferences with clinical tests, and pediatric dosage regimens. Information about drug interactions (already a subspecialty in pharmacology) is accumulating at a rate that creates and maintains a gap between report and validation. The full extent of patient problems directly related to drug interactions is still not clear; however, this section of the drug monograph alerts the nurse to the importance of continuous and sophisticated observation of the patient whose expected response to one drug may be altered or cancelled by another. Since interactions, usually reported by drug class

and by prototypes, have been described as "almost always preventable," the significance of this new section cannot be overemphasized.*

Another new feature, the explanation of interferences with laboratory tests by selected drugs further illustrates the complexities of drug therapy. Although most changes in test results are important to the physician or nurse, some also must be made known to the patient, e.g., drug-induced color changes in urine, feces, or skin, or false positive tests for glycosuria.

Since the last edition, professional nursing responsibility and functions have continued to expand, particularly in those states where Nurse Practice Acts have been revised. These acts have either introduced or strengthened a legally protected movement toward broader decision-making activities and more independence in nursing practice. Another significant development in recent years is the increased availability of information about drugs and drug treatment to patients via lay press and visual media. Both of these forces have influenced the rationale for inclusions in the section on nursing implications in drug therapy.

Nursing implications of pharmacotherapeutics represent a synthesis of scientific information from many disciplines (medicine, nutrition, pharmacology, pathophysiology, clinical laboratory science as well as from nursing). Our experience in teaching courses in pharmacodynamics, the experiential knowledge and suggestions of colleagues in nursing and allied health sciences and of our students provided much of the data base from which nursing implications were extrapolated and made explicit. Beliefs that eventual self-care within one's own environment is an ultimate goal for all patients and that each patient has unique qualities and needs guided the design of nursing implications.

A wide variety of consequences of drug therapy and implications for nurses have been included: normal laboratory values and their alterations, new methods for drug administration, expected plasma levels of drugs, differences in drug response due to different age groups, particularities about the pathophysiological condition being treated, nutritional factors important to treatment success, and teaching plans. The latter will help the patient acquire essential information needed to provide effective safe use of the drug and to assist in the decision related to when total self-care may be assumed.

In conclusion, we have found the writing of the third edition to be a renewed education in pharmacotherapy and patient care. We can only hope that utilization of this book will help others to add to their knowledge and to appreciate the significant role of the nurse in successful drug therapy.

*Hansten, P: Drug Interactions, 3rd ed. Philadelphia, Lea & Febiger, 1975, p. 1.

Drugs and Nursing Implications

CONTENTS

ABSORBABLE GELATIN SPONGE, USP
(Gelfoam)

Hemostatic (local)

ACTIONS AND USES

Sterile, water-insoluble, nonantigenic sponge prepared from purified gelatin solution. Capable of absorbing many times its weight in blood and provides an absorbable matrix into which clot forms and granulation tissue may grow. Since gelatin sponge must remain securely anchored to bleeding site, it is not as effective for brisk arterial bleeding. Entirely absorbed in 4 to 6 weeks when implanted in tissue. When applied to bleeding areas of skin, or nasal, rectal, or vaginal mucosa, completely liquefies in 2 to 5 days.

Used as adjunct to control bleeding and capillary oozing in highly vascular areas that are difficult to suture. Also used as an aid in healing of wounds and decubitus ulcers.

CONTRAINDICATIONS AND PRECAUTIONS

Frank infection; to control postpartum hemorrhage or menorrhagia; use as sole hemostatic agent in patients with blood dyscrasias.

ROUTE AND DOSAGE

Available as sponges, packs, dental packs, prostatectomy cones. *Hemostasis:* use sterile technique. Cut to desired size (minimal amount is applied to cover area). **When applied dry:** piece(s) are compressed before application to bleeding surface, and held in place with moderate pressure for 10 to 15 seconds. **When applied moist:** piece(s) are immersed in either sterile isotonic saline injection or thrombin solution. Remove from solution, squeeze to remove air bubbles, and leave immersed in solution until needed. Sponge should swell to original size and shape. If it does not, remove from solution and knead vigorously until all air is expelled, and immerse again in solution. Wet piece may be blotted with gauze before applying it to bleeding point. Hold in place for 10 to 15 seconds with cotton pledget or gauze; remove carefully with a few drops of sterile water to prevent disturbing gelatin sponge. *Decubitus ulcer:* Following debridement, gelatin sponge is placed aseptically in ulcer and covered with DSD. Dressing may be changed daily, but gelatin sponge should not be disturbed: new sponges may be added as required. If infection develops, sponge should be removed and appropriate therapy given.

NURSING IMPLICATIONS

Since gelatin sponge absorbs fluid and expands, overpacking of cavity or closed tissue space can cause pressure on adjacent structures. Report to physician if patient complains of pain or discomfort.

Be alert to signs of infection: malaise, fever, tenderness, redness, swelling.

ACETAMINOPHEN, USP *(a-seat-a-mee' noe-fen)*

(A'cenol, Acephen, Aceta, Actamin, Aminodyne, Anapap, Anuphen, Apadon, APAP, Dapa, Datril, Dimindol, Dolanex, Dularin, Febridol, Febrigesic, Febrinol, G-1, G-Lixir, Janupap, Liquiprin, Lixagesic, Neopap Supprettes, *N*-acetyl-para-aminophenol, Nilprin, Oraphen-PD, Panex, Paracetamol, Parten, Pedric, Phenaphen, Pirin, Proval, Relenol, SK-APAP, Sonapane, Sudoprin, Tapar, Tempra, Tenol, Tylenol, Valadol, Valorin, and others)

Nonnarcotic analgesic, antipyretic — *Para-aminophenol derivative*

ACTIONS AND USES

p-Aminophenol, coal tar, or aniline derivative and principal active metabolite of acetanilid and phenacetin. Analgesic and antipyretic actions approximately equivalent to those of aspirin. Unlike aspirin, it is less likely to inhibit platelet aggregation; does not produce gastric mucosal erosion and bleeding; has only weak rheumatic, antiinflammatory, and uricosuric properties; and does not antagonize the effects of uricosuric agents. Overdosage appears to be more dangerous than with aspirin because hepatotoxicity may be nonsymptomatic during early stages. Has considerably lower incidence of methemoglobinemia and hemolytic anemia than does phenacetin and the signs and symptoms of acute overdosage are markedly different. Produces analgesia by raising pain threshhold; mechanism of action uncertain, but believed to act primarily on peripheral nervous system. Reduces fever by direct action on hypothalamus heat-regulating center with consequent peripheral vasodilation and dissipation of heat. Analgesic and antipyretic actions believed to be related to inhibition of prostaglandin synthesis. In high doses, induces synthesis of hepatic microsomal enzymes. Reportedly has antidiuretic activity. Acetaminophen is available OTC both alone and in a variety of combination formulations including: Bromo Seltzer, Capron, Duradyne, Dolor, Excedrin, S-A-C, Trigesic, Vanquish. Also available, by prescription, with butabarbital Minotal, Phrenilin, and others) and with codeine (Empracet, Pavadon, and others).

Used for temporary relief of mild to moderate pain, such as simple headache, minor joint and muscle pains, neuralgia, and dysmenorrhea, and for control of fever. Widely used as an alternative to aspirin when the latter is contraindicated or not tolerated.

ABSORPTION AND FATE

Rapidly and almost completely absorbed from GI tract and well-distributed in body fluids. Peak blood levels in ½ to 1 hour, detectable in plasma for about 5 hours. Elimination half-life about 1 to 3.5 hours. Metabolized by liver microsomes; 80% excreted in urine as conjugated acetaminophen and other metabolites; 2% to 4% excreted unchanged. Crosses placenta.

CONTRAINDICATIONS AND PRECAUTIONS

Hypersensitivity to acetaminophen or phenacetin; children under 3 years of age unless directed by a physician; repeated administrations to patients with anemia, or hepatic, renal, cardiac, or pulmonary disease; G6PD deficiency. *Cautious use:* arthritic or rheumatoid conditions affecting children under 12 years of age; alcoholism, malnutrition.

ADVERSE REACTIONS

Neglible with recommended dosage. **Acute poisoning:** *3 to 24 hours postingestion:* anorexia, nausea, vomiting, dizziness, lethargy, generalized weakness, diaphoresis, chills, epigastric or abdominal pain, diarrhea; *24 to 48 hours postingestion* (often no symptoms): onset of hepatotoxicity: elevations of serum transaminases (SGOT, SGPT) and bilirubin; *3 to 5 days postingestion:* vomiting, jaundice, RUQ tenderness, hepatic necrosis, abnormal liver function tests, hypoglycemia, metabolic acidosis, increased prothrombin time, hepatic coma, acute renal failure, CNS stimulation or depression, hypothermia, circulatory failure. **Chronic ingestion:** hemoglobinemia, neutropenia, pancytopenia, leukopenia, hemolytic anemia (rare), thrombocytopenic purpura, agranulocytosis, methemoglobinemia (rare), sulfhemoglobinemia, hypoglycemia or hypergly-

cemia, splenomegaly, acute pancreatitis, psychological changes, hepatic and renal damage. **Hypersensitivity:** erythematous or urticarial skin rash, drug fever, mucosal lesions, laryngeal edema.

ROUTE AND DOSAGE

Adults: Oral, rectal: 300 to 650 mg at 4-hour intervals, as needed; maximum daily dosage: 2.6 Gm. For OTC use, it is recommended that acetaminophen not be used for more than 10 days at a time unless otherwise directed by a physician. **Pediatric:** Oral, rectal: **under 1 year:** 60 mg (highly individualized); **1 to 3 years:** 60 to 120 mg; **3 to 6 years:** 120 mg, not to exceed 480 mg/day; **6 to 12 years:** 150 to 325 mg, not to exceed 1.2 Gm/day. Pediatric doses may be repeated every 4 to 6 hours, as required, for no more than 5 doses/24 hours or for more than 5 days. Available as tablets, chewable tablets, capsules, drops, elixir, oral suspension, syrup, rectal suppositories.

NURSING IMPLICATIONS

Coadministration with a high carbohydrate meal may significantly retard absorption rate.

Caution patient not to exceed recommended dosage. Overdosing and chronic use can cause liver damage and other serious toxic effects.

Patients on prescribed high doses or long-term therapy are advised to have monitoring of hepatic, renal, and hematopoietic function.

Individuals with poor nutrition or who have ingested alcohol over prolonged periods tend to be prone to hepatotoxicity even from moderate doses.

Remind patient that reduction of fever by acetaminophen may mask serious illness.

Most poisonings result from suicide attempts or accidental ingestion by children. Caution patient to keep acetaminophen out of the reach of children.

Treatment of acute toxicity: Plasma half-life may be used to predict degree of liver damage: if greater than 4 hours hepatic necrosis is probable; if greater than 12 hours hepatic coma is likely. Emesis is induced with syrup of ipecac, or gastric lavage is used to evacuate stomach followed by administration of activated charcoal. Magnesium or sodium sulfate solution may be instilled into stomach following lavage. Use of acetylcysteine (Mucomyst) as antidote reportedly beneficial in preventing hepatic necrosis, if administered within 16 hours of acetaminophen overdose. If activated charcoal has been used it should be lavaged until clear because it may interfere with acetylcysteine absorption. Acetylcysteine (Mucomyst) 140 mg/kg loading dose diluted: 1 part acetylcysteine to 3 parts liquid (cola, grapefruit juice, or plain water). Treatment continued with acetylcysteine (Mucomyst) 70 mg/kg every 4 hours for 17 doses or 3 days. If patient vomits within an hour after a given dose repeat dose and continue treatment protocol; duodenal tube may be used, if necessary. Follow treatment with serum acetaminophen assays (in about 4 hours postingestion), liver function tests, coagulation, and electrolyte studies.

Patient who has ingested a toxic dose should be hospitalized because the onset of hepatic damage is usually insidious and may not be apparent until several days after overdosage.

High abuse potential; psychological dependence can occur. Self-administered acetaminophen is intended for temporary use only.

Withdrawal of acetaminophen following long-term use may be associated with restlessness and excitement.

There is no basis for the claim that acetaminophen is safer than aspirin. There is little evidence that combination analgesic formulations have any clinical advantage over single component products.

Preserved in tightly covered, light-resistant containers.

4 *DIAGNOSTIC TEST INTERFERENCES:* Acetaminophen may cause (1) false increases in urinary **5-HIAA** (5-hydroxyindoleacetic acid) determinations by qualitative tests using nitrosonaphthol reagent and (2) false decrease in **blood glucose** as measured by glucose oxidase/peroxidase procedure.

DRUG INTERACTIONS

Plus	Possible Interactions
Oral anticoagulants	In therapeutic doses, acetaminophen (also true of phenacetin) produces only slight increase, if any, in hypoprothrombinemic response, but chronic administration of large doses may cause significant potentiation.

ACETAZOLAMIDE, USP *(a-set-a-zole' a-mide)*

(Ak-Zol, Diamox, Diamox Sequels, Hydrazo Rozolamide)

ACETAZOLAMIDE SODIUM, USP

(Diamox Parenteral)

Diuretic, anticonvulsant *Carbonic anhydrase inhibitor; Sulfonamide*

ACTIONS AND USES

Nonbacteriostatic sulfonamide derivative. Diuretic effect is due to inhibition of carbonic anhydrase activity in proximal renal tubule, thereby preventing formation of carbonic acid (H_2CO_3), source of hydrogen (H^+) and bicarbonate (HCO_3^-) ions. Absence or reduced availability of hydrogen ions inhibits renal tubular reabsorption of sodium and enhances its elimination along with that of potassium, bicarbonate, and water. The net result is diuresis of alkaline urine, which is accompanied by chloride and ammonia conservation. After 3 or 4 days of continuous inhibition, mild metabolic acidosis develops with concomitant reduction in diuresis. Inhibition of carbonic anhydrase in the eye reduces rate of aqueous humor formation with consequent lowering of intraocular pressure. This effect is apparently independent of systemic acid–base balance and diuretic action. Mechanism of anticonvulsant action unknown, but thought to involve inhibition of carbonic anhydrase in CNS which retards abnormal paroxysmal discharge from CNS neurons.

Used adjunctively in treatment of several forms of epilepsy, especially absence seizures, generalized tonic-clonic and focal seizures, and to reduce intraocular pressure in open angle glaucoma, secondary glaucoma, and for short-term preoperative treatment in acute angle closure glaucoma. Also used as adjunct in treatment of edema due to congestive heart failure and drug-induced edema. May be used to correct metabolic alkalosis or to potentiate or restore the effectiveness of mercurial diuretics. Given occasionally to alkalinize urine, and to relieve symptoms of acute mountain sickness.

ABSORPTION AND FATE

Rapidly absorbed from GI tract with beginning effects in 30 minutes to 1 hour, following tablet administration; plasma levels peak within 2 to 4 hours, and persist 6 to 8 hours. Sustained-release form peaks in 8 to 12 hours with duration of 18 to 24 hours. Acts within 2 minutes after IV administration; duration: 4 to 5 hours. Wide distribution, including CNS with especially high concentrations in erythrocytes, pancreas, gastric mucosa, and renal cortex. Highly bound to plasma proteins. Excreted unchanged in urine; 90% within 24 hours. Crosses placenta.

CONTRAINDICATIONS AND PRECAUTIONS

Hypersensitivity to sulfonamides; renal and hepatic dysfunction; Addison's disease or other types of adrenocortical insufficiency, hyponatremia, hypokalemia, hyperchlo-

remic acidosis, prolonged administration to patients with chronic noncongestive angle closure glaucoma, pregnancy. *Cautious use:* history of hypercalciuria, diabetes mellitus, gout, digitalized patients, obstructive pulmonary disease.

ADVERSE REACTIONS

CNS: paresthesias, especially of face and extremities, and drowsiness are common; disorientation, depression, nervousness, excitement, fatigue, flaccid paralysis, headache, dizziness, convulsions. **GI:** anorexia, nausea, vomiting, weight loss, constipation, diarrhea, melena. **Blood chemistry and electrolytes:** hypokalemia, hyperchloremic acidosis, hyperglycemia, hyperuricemia. **GU:** urinary frequency, polyuria, glycosuria, ureteral colic, nephrolithiasis. **EENT:** transient myopia, tinnitus. **Hypersensitivity:** pruritus, rash, urticaria, fever. **In common with other sulfonamides:** hematuria, crystalluria, renal calculi, hepatic dysfunction, bone marrow depression with agranulocytosis, thrombocytopenic purpura, hemolytic anemia (infrequently), leukopenia, pancytopenia. **Other:** hirsutism, loss of libido, impotence, exacerbation of gout, altered taste and smell, thirst.

ROUTE AND DOSAGE

Adults: *Glaucoma:* Oral: 250 mg every 6 hours (timed-release preparations may be prescribed every 12 to 24 hours); intravenous, intramuscular: initially 500 mg repeated in 2 to 4 hours, if necessary; *anticonvulsant, diuretic:* 250 to 1 Gm daily in divided doses. **Pediatric:** oral, intravenous, intramuscular: *anticonvulsant, glaucoma:* 5 to 10 mg/kg every 6 hours; *diuretic:* 5 mg/kg once daily.

NURSING IMPLICATIONS

Not to be confused with acetohexamide.

Teach patient not to accept brand interchange, as it is not recommended for carbonic anhydrase inhibitor products.

If necessary, regular tablet (not sustained release form) may be softened in hot water and added to 1 or 2 teaspoonsful of honey or syrup (e.g., chocolate, raspberry, cherry to disguise bitter taste). Not stable in fruit juices.

Reportedly, timed-release capsule may be better tolerated than tablet form, but may not be as effective in some patients.

Highly alkaline pH (approximately 9.2) of the parenteral solution causes intense pain when injected intramuscularly; therefore, this route is not commonly employed.

Diuretic dose is generally administered in the morning to avoid interference with sleep as a result of diuresis.

Monitor intake and output in patients receiving the drug to reduce edema. Effectiveness as a diuretic diminishes in a few days; therefore, it is usually given on alternate days or for 2 days followed by a day without medication.

An adequate fluid intake should be maintained during acetazolamide therapy. Check with physician for optimum intake.

Patient should be weighed under standard conditions before drug therapy is initiated and daily thereafter: use same scale, with similar clothing, preferably in morning after voiding but before eating or defecating. Daily weight is a useful index of patient's response to diuretic action.

Advise patient to report numbness, tingling and other paresthesias, and drowsiness (common side effects). Note implications for the ambulatory patient. Caution patient to avoid driving a car and other potentially hazardous activities if these symptoms are prominent.

It is reported that acetazolamide may cause substantial increases in blood glucose in diabetics and in prediabetics. Observe these patients closely; changes in antidiabetic drug dose or diet may be indicated.

Hypokalemia and severe metabolic acidosis are direct extensions of the pharmacologic actions of acetazolamide. Note that concomitant use of steroids or ACTH

may contribute to hypokalemia. Hypokalemia can sensitize the heart to toxic effects of digitalis.

Observe for and advise patient to report signs of hypokalemia (malaise, fatigue, muscle weakness, leg cramps, rapid, irregular pulse, vomiting, abdominal distention, polyguria), and signs of metabolic acidosis (malaise, headache, weakness, abdominal pain, nausea, vomiting, dyspnea, hyperpnea, dehydration).

When acetazolamide is given in high doses or for prolonged periods physician may prescribe potassium-rich diet and potassium supplement, if necessary. Foods high in potassium include: bananas, oranges, and other citrus fruits; melons; avocado; potato; cabbage; brussel sprouts; among others.

Periodic blood pH, blood gases, complete blood cell counts, and serum electrolyte determinations are recommended during prolonged drug therapy or during concomitant therapy with other diuretics or digitalis.

Because the parenteral solution contains no antibacterial preservative, its use within 24 hours of reconstitution is strongly recommended by manufacturer. (Each 500 mg vial should be reconstituted with at least 5 ml sterile water for injection prior to use.)

Oral preparations are preserved in tightly covered, light-resistant containers.

DIAGNOSTIC TEST INTERFERENCES: False positive **urinary protein** determinations; falsely high values for **urine urobilinogen.**

DRUG INTERACTIONS

Plus	Possible Interactions
Ammonium chloride	Concomitant administration may diminish diuretic effect.
Amphetamines	Enhanced amphetamine effect. By alkalinizing urine, acetazolamide decreases amphetamine excretion.
Anticonvulsants	Predisposes patient to anticonvulsant osteomalacia (adult rickets) by increasing calcium excretion.
Antidiabetic agents	Acetazolamide increases blood sugar in diabetics on insulin or oral hypoglycemics and prediabetics.
Lithium carbonate	Increases lithium excretion, therefore decreases lithium effect.
Methenamine compounds	By alkalinizing urine, acetazolamide inhibits antibacterial effect of methenamine.
Primidone (Mysoline)	Decreases primidone effect. (Acetazolamide may reduce GI absorption of primidone in some patients.)
Quinidine Quinine	Alkaline urine (acetazolamide effect) causes decreased excretion of these drugs, and possible toxicity.
Salicylates	Acetazolamide may increase risk of salicylate toxicity in patients receiving large doses, by shifting salicylates from plasma into tissues.