HANDBOOK OF DRUG INTERACTIONS

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GERALD SWIDLER
ENDO LABORATORIES
Los Angeles, California

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PREFACE

This compilation was begun because I believed that there was a need by the physician and pharmacist for additional help in the safe treatment of patients and for the protection of the physician against possible legal action, especially when more than one drug for the patient must be prescribed.

I have based this book on three premises: the pharmacological complexity and potency of many pharmaceuticals on the market, the time or lack of it on the part of the medical and pharmaceutical professions necessary to research each pharmaceutical preparation or combination of products, and the difficulty of correlating the material.

The *Handbook of Drug Interactions* was compiled from information supplied to the medical profession by the pharmaceutical manufacturers in the form of package inserts, literature, and advertisements, the *Physicians Desk Reference*, medical and pharmaceutical journals, and pharmacology texts. Whenever trade-marked names are used, the language of the manufacturer has been adhered to. When animal studies were used, they are so designated.

Since this book does not intend to masquerade as a pharmacology text, no effort has been made to include the mechanisms for all drug interactions. Many of them are so pharmacologically or physiologically complex that I felt I would be doing the reader a disservice by trying to condense this information when a pharmacology text would give complete understanding. The mechanisms not listed for other interactions are either unknown or self-evident. In several instances the mechanism for a drug interaction listed in one section of the book differs from or contradicts a similar drug interaction mechanism in another. This contradiction or difference occurs because the source material and the information supplied by the drug manufacturer do not agree or because a new study appeared which in my opinion was so good that I felt obligated to include it; e.g., Anticoagulants + Chloral Hydrate.

This book has a dictionary format in that all entries are arranged alphabetically. This was done to make it easier to locate a particular drug or drug

interaction. The interaction is explained in an encyclopedic format, which to conserve reading time presents the information as concisely as possible.

The statement "See listed under the following agent(s)" following a main entry indicates crossfiling. This was done to make the reader aware that a particular drug can interact with any number of drugs, differing not only in name but in pharmacologic action, and to encourage him to look up that drug and all other drugs with which it may interact.

The notation "See" or "See also" refers the reader to a similar mechanism for a drug interaction, a similar grouping of drug interactions under a pharmacologic entry, or a breakdown of a fixed-combination drug into its individual components, in which case the reader can then look up each of the ingredients. I have in most instances interpolated the generic name to a trade-marked name for that drug if the drug is marketed by only one manufacturer, even if it differs from the manufacturer of the combination drug.

For ease in locating a particular drug the index is in two sections. The General Index, pages 351 through 370, is a list of all agents found in the book. The generic names of single-ingredient drugs are given next to them in parentheses. The Generic Name Index, pages 371 through 384, is a listing of drugs in the book by generic or chemical name with the trade-marked name(s) listed beneath.

All entries in the book are crossfiled for ease and rapidity in locating a particular drug.

I make no claim to have covered every pharmaceutical preparation available to the physician or found on the pharmacy shelf. Unfortunately, lack of information, either in the form of definitive studies or from the manufacturer, forced me to omit material from this edition. The study of drug interactions is relatively new and there is much we do not know.

GERALD SWIDLER

West Covina, California
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A

ACETATE FABRICS + Cresatin: see Cresatin + Acetate Fabrics

ACETYLCHOLINE: see listed under the following agents:
Isordil
Pentritol
Sorbitrate

ACETYLSALICYLIC ACID: see listed under the following agents:
Levoprome
Liquamar

ACHROMYCIN
+ Antacids: see Achromycin + Food
+ Drugs, High Calcium Content: see Achromycin + Food
+ Food: Absorption of Achromycin is impaired by concomitant administration of high calcium content drugs such as some antacids and foods and some dairy products such as milk. Oral forms of tetracycline should be given one hour before or two hours after meals. Mechanism: Tetracyclines are chelating agents that form insoluble chelates with multivalent cations (Ca^{++}, Mg^{++}, Al^{+++}) and gastrointestinal absorption is inhibited. (1, 55, 81)
+ Milk and Dairy Products: see Achromycin + Food
+ Trisoralen: see Trisoralen

ACHROMYCIN Parenteral
+ Calcium-Containing Solutions: The use of solutions containing calcium should be avoided, since they tend to form precipitates (especially in neutral or alkaline solution) and therefore should not be used unless necessary. Ringer's Injection U.S.P. and Lactated Ringer's Injection U.S.P. can be used with caution, however, for the calcium ion content in these diluents does not normally precipitate tetracycline in an acid media. (1)
+ Hemastix: see Hemastix

ACIDIC AGENTS
+ Alkaline Media: The following drugs which are weak acids will not be absorbed well in an alkaline media if, for example, the patient is taking antacids at the same time. Mechanism: Unionized drugs generally are more lipid soluble and poorly water soluble. The ionized drugs are poorly lipid soluble and are water soluble. The membranes in the body (cell membrane, blood vessels, stomach

ACIDIC AGENTS (continued)

+ Alkaline Media (continued): and intestinal lining) are lipoidal and therefore uncharged forms of drugs pass freely through them; charged forms transfer across cell membranes relatively slowly. Any drug that influences the pH of the fluid in a given compartment will influence absorption of a drug from there. The proportion of ionization is greater when acidic agents are in an alkaline media and therefore less lipoid soluble. (50, 55, 124)

Butazolidin	Furadantin	Salicylic Acid
Coumarins	NegGram	Sulfonamides

+ Alkanizing Agents (Urinary): Weak acids are excreted at a higher clearance in highly alkaline urine. Mechanism: The proportion of ionization is greater when acidic agents are in an alkaline media and therefore more soluble in water and less lipoid soluble. This assumes that the compound has not been primarily metabolized. (50, 55, 124) Drugs that are known to show this phenomenon of pH-dependent excretion include the weak acids:

Furadantin	Phenobarbital	Sulfonamides
NegGram	Salicylic Acid	

ACIDIC DRUGS: see listed under the following agents:
Antacids
Paveril Phosphate

ACIDIC MEDIA + Alkaline Agents: see Alkaline Agents + Acidic Media

ACIDIC SOLUTIONS: see listed under the following agents:
Brevital Sodium
Keflin
Premarin I.V.

ACIDIFYING AGENTS (Urinary) + Alkaline Agents: see Alkaline Agents + Acidifying Agents (Urinary)

ACIDITY: see listed under the following agents:
Mandelamine
Thrombin

ACIDS: see listed under the following agents:
Merthiolate
Thrombin, Topical

ACNE MIXED UBA + Antiseptic: If any of the antiseptic used to cleanse the rubber stopper is carried into the vial by the needle, denaturation of the proteins may occur and thus the special advantage of the antigen will be lost. (1)

ACRIDINE + Trisoralen: see Trisoralen

ACTH: see Adrenal Corticosteroids, Adrenocorticosteroids, Corticosteroids, and Steroids

ACTH: see listed under the following agents:

- Anhydron
- Anticoagulants
- Biavax
- Brucellergen
- Dicumarol
- Diuril
- Esidrix
- Hedulin
- HydroDIURIL
- Measles Virus Vaccine, Inactivated, Aluminum Phosphate Adsorbed
- Metahydrin
- Naqua
- Naturetin
- Panwarfin
- Poliomyelitis Vaccine (Lilly)
- Rabies Vaccine (Duck Embryo), Dried Killed Virus
- Smallpox Vaccine, Dried
- Tetanus Toxoid, Alum Precipitated
- Tetra-Solgen
- Tri-Solgen
- Typhoid Vaccine
- Typhus Vaccine

ACTHAR + Diabetes: Corticotrophin may aggravate diabetes mellitus so that higher insulin dosage may become necessary or manifestations of latent diabetes mellitus may be precipitated. (1)

HP ACTHAR GEL: see Acthar

ACTOSPAR

+ Oxytocics: In patients refractory to Actospar, at least three hours should elapse between oxytocics; e.g., synthetic oxytocin (Syntocinon) is administered to prevent potential synergistic action between oxytocin and Actospar. (1)
+ Syntocinon: see Actospar + Oxytocics

ACUSUL + Sulfonylureas, Hypoglycemic: Sulfonamide therapy may potentiate the hypoglycemic action of sulfonylureas. Mechanism: Displacement of the sulfonylurea from its protein binding site. (1)

ADRENAL CORTICOSTEROIDS: see listed under the following agents:

- Hygroton
- Measles Virus Vaccine, Inactivated, Aluminum Phosphate Adsorbed
- Poliomyelitis Vaccine (Lilly)
- Rabies Vaccine (Duck Embryo), Dried Killed Virus
- Tetanus Toxoid, Alum Precipitated
- Tetra-Solgen
- Tri-Solgen
- Typhoid Vaccine
- Typhus Vaccine

ADRENAL CORTICOSTEROIDS + Diabetes: Close observation of diabetic patients and sometimes an increase in insulin requirements may be necessary due to the hypoglycemic effects of adrenal corticosteroids. The gluconeogenesis, when not compensated by an adequate insulin output, leads to hyperglycemia and glycosuria. (2)

ADRENERGIC AGENTS

+ Diabetes: Because of the glycogenolytic effect, adrenergic agents should be very carefully administered to diabetic patients. (2)
+ Isoniazid: see Isoniazid + Adrenergic Agents
+ Monoamine Oxidase Inhibitors: Adrenergic agents should not be given to patients receiving monoamine oxidase inhibitors, since this combination may precipitate a hypertensive crisis. For mechanism see MAO Inhibitors + Sympathomimetic Drugs. (2)

ADRENERGIC NEURON BLOCKING AGENTS: see listed under the following agents:

Pertofrane
Tofranil

ADRENOCORTICAL STEROIDS: see listed under the following agents:

Anhydron
Aquatag
Diuretics, Thiazide
Enduron
Esidrix
Exna
HydroDIURIL
Hydromox
Metahydrin
Naqua
Renese
Saluron
Smallpox Vaccine

ADRENOCORTICOSTEROIDS: see listed under the following agents:

Dicumarol
Digitalis
DPT Vaccine

ADRENOCORTICOSTEROIDS

+ Anticoagulants: Concomitant use may cause a reversal of hypoprothrombinemic state.
+ Diabetes: Diabetic patients frequently need an increase in insulin dosage if they are taking adrenocorticosteroids.
+ Digitalis: Potassium depletion due to the adrenocorticosteroid may precipitate digitalis intoxication and arrhythmias without actual overdosage of the digitalis.
+ DPT Vaccine: Concomitant administration should be avoided because the steroids may interfere with antibody response.
+ Measles Virus Vaccine, Inactivated: Adrenocorticosteroids may suppress the antibody response to the vaccine; therefore, if possible,

ADRENOCORTICOSTEROIDS (continued)

+ Measles Virus Vaccine, Inactivated (continued): it would seem advisable to avoid administration of the vaccine concomitantly with these hormones.
+ Measles Virus Vaccine, Live, Attenuated: In patients under treatment with therapeutic doses of steroids, vaccination against smallpox and attacks of measles, chicken pox, and other acute contagious diseases has resulted in serious or fatal illness. At present it is not definitely established that the administration of a live attenuated measles virus vaccine has the same potential hazard. Therefore, until further definitive data are available, the physician should weigh carefully the use of this vaccine in patients under treatment with steroids. Most pharmaceutical companies consider concomitant usage contraindicated.
+ Mumps Vaccine: see Adrenocorticosteroids + Rabies Vaccine
+ Rabies Vaccine: Adrenocorticosteroids may reduce host resistance to certain infectious agents by suppression of antibody response or by other still poorly understood mechanisms. Therefore they should not be administered after exposure to infectious agents (mumps, rabies, tetanus) for which no satisfactory antimicrobial therapy is available. To do so may alter the host-parasite relationship enough to cause severe or fatal illness in spite of the prophylactic administration of a vaccine. .
+ Smallpox Vaccine: see Adrenocorticosteroids + Measles Virus Vaccine, Live, Attenuated
+ Stoxil: In superficial infections Stoxil should not be used in combination with steroids. In deep infections, if such combined therapy is judged necessary, it must be employed with caution and the patient must be observed.
+ Tetra-Solgen: Concomitant administration should be avoided because the steroids may interfere with antibody response.
+ Thiazide Diuretics: Hypokalemia is more likely to develop or a preexistent potassium deficiency may be aggravated during periods of brisk diuresis.
+ Tri-Solgen: Concomitant administration should be avoided because the steroids may interfere with antibody response.

ADRENOLYTIC AGENTS + Aramine: see Aramine + Adrenolytic Agents

ADRENOSEM + Antihistamines: Antihistamines tend to inhibit the efficacy of Adrenosem and should be discontinued 48 hours before Adrenosem is started. If conditions do not permit sufficient time for withdrawal of antihistamines, the usual dose of Adrenosem should be increased to

ADRENOSEM + Antihistamines (continued): 2 cc (10 mg) for the initial injection. (1)

ADRESTAT + Antihistamines: Antihistamines may inactivate Adrestat. If a patient has received antihistamines, at least 12 hours should be allowed to elapse before starting therapy. In emergencies, however, Adrestat may be used if the dose is doubled and repeated in two or three hours if necessary. (1)

AEROLONE COMPOUND: see Isoproterenol

AIR + Surital: see Surital + Air

AKRINOL + Soap: All soap must be removed by thorough rinsing and followed by thorough drying with a towel before Akrinol cream is applied. Soap can drastically reduce the antifungal activity of Akrinol. (1, 2)

ALCOHOL: see listed under the following agents:

Ansolysen
Antabuse
Anticoagulants
Antiemetics, Phenothiazine-type
Antihistamines
Anhydron
Anti-Nausea Suprettes
Apresoline
Atarax
Aventyl
Benzodiazepines
Bristuron
Compazine
Coronary Vasodilators (Nitrates)
Coumadin
Dalmane
Dartal
Deprol
Diabinese
Dicumarol
Disophrol
Diuretics, Thiazide
Diuril
Doriden
Dymelor
Edecrin
Elavil
En-Chlor
Equanil
Esidrix
Eutonyl
Flagyl
Furoxone
Haldol
Harmonyl
HydroDIURIL
Hydromox
Hygroton
Hypoglycemic Drugs, Oral
Insulin
Inversine
Ismelin
Isordil
Lenetran
Levoprome
Librium
Lipan
Listica
MAO Inhibitor
Marplan
Matulane
Mellaril
Meprobamate
Miltown

ALCOHOL (continued):

Nardil
Navane
Niamid
Noludar
Orinase
Pacatal
Parest
Parnate
Pentritol
Periactin
Permitil
Pertofrane
Phenobarbital
Phenothiazine
Placidyl
Plaquenil
Plasmanate
PMB-200
PMB-400
Probanthine with Dartal
Proketazine
Prolixin
Quaalude
Quiactin
Repoise
Seconal Sodium
Serax
Serentil
Seromycin
Sinequan
Sintrom
Solacen
Somnofac
Somnos
Sopor
Sorbitrate
Sparine
Stelazine
Striatran
Sulfonylurea, Hypoglycemic Oral
Symmetrel
Tacaryl
Taractan
Temaril
Thorazine
Tindal
Tofranil
Tolinase
Torecan
Trancopal
Trepidone
Trilafon
Tybatran
Ultran
Valium
Valmid
Vesprin
Vistaril

ALCOHOL

+ Barbiturates: see Alcohol + CNS Depressants. Mechanism: The inhibition of drug-metabolizing enzymes by alcohol may contribute to the increased sensitivity of inebriated persons to barbiturates. (47)
+ Chlorpromazine: see Alcohol + CNS Depressants
+ CNS Depressants: The synergism of the narcotic action of alcohol with a number of central nervous system depressant drugs may produce coma or even death by respiratory depression. In this group are barbiturates, meprobamate, chlorpromazine, and similar phenothiazine derivatives. (26)

ALCOHOL (continued)

+ Diphenylhydantoin: It has been demonstrated that heavy drinking speeds up the metabolism of diphenylhydantoin. Mechanism: It is postulated that this effect is the result of stimulation of the hepatic microsomal enzyme system in the human liver responsible for the metabolizing of diphenylhydantoin. (45)
+ Insulin: Alcohol has a glucose-lowering action that augments the hypoglycemic effects of insulin and may cause severe hypoglycemia and irreversible neurological changes. Studies have shown that alcohol inhibits the formation of new glucose from amino acids and other precursors. The interference with gluconeogenesis has been attributed to an increase in the ratio of reduced to oxidized nicotinamide adenine dinucleotide ($NADH_2/NAD$) within the hepatic cell during the oxidation of alcohol. Increases in the $NADH_2/NAD$ ratio inhibits the entrance of glycerol, lactic acid, and specific amino acids into the metabolic pathways through which these metabolites are converted to glucose. (49)
+ Meprobamate: see Alcohol + CNS Depressants
+ Nitroglycerin: If nitroglycerin and alcohol are given at approximately the same time, an Antabuse-alcohol type reaction may occur. (26)
+ Orinase: It has been demonstrated that heavy drinking speeds up the metabolism of tolbutamide. Mechanism: It is postulated that this effect is the result of stimulation by alcohol of a hepatic microsomal enzyme system in the human liver responsible for the metabolism of Orinase. (35, 45) See Alcohol + Sulfonylurea, Hypoglycemic.
+ Phenothiazines: see Alcohol + CNS Depressants
+ Sulfonylurea, Hypoglycemic: The oral antidiabetic drugs of the sulfonylurea series such as Orinase and Diabinese when used concomitantly with alcohol may cause an Antabuse-alcohol reaction but milder. Mechanism: The reaction is probably due to a similar mechanism which occurs with Antabuse-alcohol. Alcohol is oxidized in the body to acetaldehyde, acetic acid, and carbon dioxide. Antabuse blocks the enzyme system responsible for the conversion of acetaldehyde into acetate and large amounts of acetaldehyde accumulates in the blood. This evokes a fall in blood pressure, gastrointestinal distress, and the faintness characteristic of the Antabuse-alcohol syndrome. (26, 41) See Alcohol + Orinase.
+ Warfarin: It has been demonstrated that heavy drinking speeds up the metabolism of Warfarin. Mechanism: It is postulated that this effect is the result of stimulation by alcohol of the hepatic microsomal enzyme system in the human liver responsible for the metabolizing of Warfarin. (45)

ALDACTAZIDE: see Aldactone and Hydrochlorothiazide

ALDACTAZIDE

+ Diabetes: Thiazides may decrease glucose tolerance, thus temporarily exaggerating abnormalities of glucose metabolism in diabetic patients or causing them to appear in those latent diabetes. Aldactazide may have similar effects on glucose tolerance. (1)
+ Sodium: The most common electrolyte disturbance encountered with Aldactazide therapy is dilutional hyponatremia. Administration of sodium in this situation is usually contraindicated. (1)

ALDACTONE: see listed under the following agents:

Dyazide
Dyrenium
Lasix

ALDACTONE

+ Antihypertensive Agents: Aldactone may potentiate the action of other antihypertensive drugs. The dosage of such drugs, particularly the ganglionic blocking agents, should be reduced at least 50% when Aldactone is added to the regimen. (1)
+ Diuretics, Mercurial: see Aldactone + Diuretics, Thiazide
+ Diuretics, Thiazide: Aldactone exerts a true synergistic effect when administered concomitantly with thiazide or organic mercurial diuretics. When administered in combination with thiazide or organic mercurial diuretics, the potassium loss induced by these diuretics is offset. Supplemental administration of potassium is contraindicated during such therapy unless glucocorticoids are administered. When Aldactone is used concomitantly with thiazide or organic mercurial diuretics, it has a synergistic effect on sodium excretion. The possible development of hyponatremia, manifested by dryness of the mouth, thirst, lethargy, drowsiness, and a low serum sodium, must be considered. (1, 2)
+ Ganglionic Blocking Agents: see Aldactone + Antihypertensive Agents
+ Glucocorticoids: see Aldactone + Diuretics, Thiazide and Aldactone + Potassium
+ Potassium: Potassium supplementation, either in the form of medication or as a potassium rich diet, is not indicated unless a glucocorticoid is also given. Such supplementation to Aldactone alone may cause hyperkalemia in rare instances. (1, 50)

ALDOCLOR: see Aldomet and Diuril

ALDOMET: see listed under the following agents:

Dopar	Nardil
Esidrix	Naturetin
Eutonyl	Parnate
Larodopa	Raudixin
Levodopa	Rauwiloid
Levophed	Sympathomimetics, Direct-Acting
MAO Inhibitor	Unitensin

ALDOMET

+ Anesthesia: Concomitant usage may prevent the surgical patient from compensating for the hypotensive challenge of anesthesia. (55) Mechanism: see Ismelin + Anesthesia.
+ Antihypertensive Agents: Therapy with Aldomet may be initiated in most patients already under treatment with antihypertensive agents by terminating all previous medication except thiazide drugs which may be continued. By gradually decreasing the dosage of ganglionic blocking agents and Ismelin and gradually adding Aldomet a smooth transition in therapy can be accomplished. (1) See also Aldomet + Drugs.
+ Aramine: Aldomet may mildly potentiate the action of Aramine. (55) Mechanism: see Aldomet + Wyamine.
+ Diuretics, Thiazide: When a thiazide diuretic is given simultaneously, the antihypertensive effect is enhanced. In addition, the thiazide counteracts the retention of sodium and the increased plasma volume that often occur after long-term administration of Aldomet. (1, 2)
+ Drugs: When Aldomet is used in combination with other drugs, potentiation of the action may occur. See also Aldomet + Diuretics, Thiazide and Aldomet + Antihypertensive Agents. (1)
+ Ganglionic Blocking Agents: see Aldomet + Antihypertensive Agents
+ Ismelin: see Aldomet + Antihypertensive Agents
+ Levophed: see Aldomet + Sympathomimetics, Direct-Acting
+ Methamphetamine: Methamphetamine may inhibit the effects of Aldomet to some extent. (55)
+ Sympathomimetics, Direct-Acting: Direct-acting vasopressors such as Levophed given to counteract severe hypotension in a patient who has taken Aldomet, will induce a much greater response than would be expected under normal circumstances. Mechanism: Aldomet works to prevent the uptake of Levophed into inactivation sites and therefore potentiates the effect of the vasopressor. (55)
+ Sympathomimetics, Indirect-Acting: see Aldomet + Aramine and Aldomet + Wyamine
+ Wyamine: Aldomet may mildly potentiate the action of Wyamine. (55) Mechanism: It is postulated that Aldomet is metabolized to alpha

ALDOMET (continued)

+ Wyamine (continued): methylnorepinephrine (a false neurotransmitter) which displaces norepinephrine and is stored in its place in the nerve endings. Alpha methylnorepinephrine may be less potent than norepinephrine or is more tightly bound and less susceptible to release by nerve stimulation. The indirect-acting sympathomimetics act by releasing this false neurotransmitter which may be the cause of the potentiation. (104)

ALDORIL: see Aldomet and HydroDIURIL

ALKALIES + Thrombin, Topical: see Thrombin, Topical + Alkalies

ALKALINE AGENTS

+ Acidic Media: The following drugs which are weak bases will not be absorbed well in an acidic media. Mechanism: Unionized drugs generally are more lipid soluble and poorly water soluble. The ionized drugs are poorly lipid soluble and are water soluble. The membranes in the body (cell membrane, blood vessels, stomach and intestinal lining) are lipoidal and therefore uncharged forms of drugs pass freely through them; charged forms transfer across cell membranes relatively slowly. Any drug that influences the pH of the fluid in a given compartment will influence absorption of a drug from there. The proportion of ionization is greater when alkaline agents are in an acidic media and therefore less lipoid soluble. (50, 55, 124)

Amphetamine	Meperidine
Antipyrine	Procaine
Aralen	Quinidine
Inversine	Theophylline

+ Acidifying Agents (Urinary): Weak bases are excreted at a higher clearance in highly acidic urine. Mechanism: The proportion of ionization is greater when alkaline agents are in an acidic media and therefore are more water soluble and less lipoid soluble. This assumes that the compound has not been primarily metabolized. (50, 55, 124) Drugs that are known to show the phenomenon of pH-dependent excretion include the weak bases:

Amphetamine	Meperidine
Antipyrine	Nicotine
Aralen	Procaine
Atabrine	Quinine
Elavil	Theophylline
Inversine	Tofranil
Levo-Dromoran	