\hat{A} , the period graphs \hat{B} , \hat{A}^{M} , \hat{G}^{M} , \hat{G}^{M} ate of similar

institute of the state of

And of London

fifth edition

PATHOLOG PHYSIO



W. B. SAUNDERS COMPANY • PHILADELPHIA • LONDON • TORONTO

***Militarian of the system of the second of the second of the system of the second of the second

CONTRIBUTORS

Peter Abramoff, Ph.D. Professor and Chairman, Department of Biology, Marquette University, Milwaukee, Wisconsin.

J. A. ABILDSKOV, M.D. Professor of Internal Medicine, University of Utah College of

Medicine, Salt Lake City, Utah.

EZRA A. AMSTERDAM, M.D. Assistant Professor of Medicine, Assistant Professor of Pharmacology, Director of Coronary Care Unit, Section of Cardiovascular Medicine, Departments of Medicine and Pharmacology, University of California School of Medicine, Davis, California.

CHARLES E. BILLINGS, M.D., M.Sc. Professor and Director, Division of Environmental Health, Department of Preventive Medicine, The Ohio State University; Attending

Physician, Ohio State University Hospitals, Columbus, Ohio.

GEORGE A. BRAY, M.D. Professor of Medicine, UCLA School of Medicine; Director,

Clinical Study Center, Harbor General Hospital, Torrance, California.

THOMAS W. BURNS, M.D. Professor of Medicine and Director, Division of Endocrinology, University of Missouri School of Medicine; Attending Endocrinologist, University of Missouri Medical Center; Consultant in Endocrinology, V.A. Hospital, Columbia, Missouri

BERTRAM D. DINMAN, M.D., Sc.D. Medical Director, Aluminum Company of America,

Pittsburgh, Pennsylvania.

HAROLD T. DODGE, M.D. Professor of Medicine, University of Washington; Co-Director, Division of Cardiology; Director, Cardiovascular Research and Training Center, Seattle, Washington.

LEONARD S. DREIFUS, M.D. Clinical Professor of Medicine and Senior Attending

Physician, Hahnemann Medical College, Philadelphia, Pennsylvania.

RENÉ J. DUQUESNOY, Ph.D. Assistant Professor of Microbiology, The Medical College

of Wisconsin, Milwaukee, Wisconsin.

Louis J. Elsas, II, M.D. Associate Professor of Pediatrics, Assistant Professor of Internal Medicine, Assistant Professor of Biochemistry, Chief, Section of Medical Genetics (Department of Pediatrics), Emory University School of Medicine. Attending Physician, Emory University Hospital, Grady Memorial Hospital, and the Henrietta Egleston Hospital for Children, Atlanta, Georgia.

ALLAN J. ERSLEY, M.D. Cardeza Research Professor of Medicine, Jefferson Medical College of Thomas Jefferson University; Attending Physician (Medicine), Thomas

Jefferson University Hospital, Philadelphia, Pennsylvania.

ROBERT E. FORSTER, II, M.D. Isaac Ott Professor and Chairman of the Department of Physiology, University of Pennsylvania School of Medicine; Physiologist, Hospital of the University of Pennsylvania and Children's Hospital; Consultant, Philadelphia Naval Hospital and Philadelphia General Hospital.

THOMAS G. GABUZDA, M.D. Professor of Medicine, Jefferson Medical College of Thomas Jefferson University; Chief, Department of Hematology, Lankenau Hospital; Con-

sulting Staff, Roxborough Memorial Hospital, Philadelphia, Pennsylvania.

Benjamin Robert Gendel, M.D. Professor and Associate Chairman, Department of Medicine, University of Tennessee College of Memphis; Chief, Medical Service, V.A. Hospital, Memphis, Tennessee.

RAY W. GIFFORD, JR., M.D. Head, Department of Hypertension and Nephrology, Cleveland Clinic Foundation, Cleveland, Ohio.

Franz Goldstein, M.D. Professor of Medicine, Jefferson Medical College of Thomas Jefferson University; Chief, Department of Gastroenterology, Lankenau Hospital, Philadelphia, Pennsylvania.

ARTHUR C. GUYTON, M.D. Professor of Physiology and Biophysics, University of

Mississippi School of Medicine, Jackson, Mississippi.

W. PROCTOR HARVEY, M.D. Professor of Medicine; Director, Division of Cardiology, Georgetown University Medical School; Georgetown University Hospital; Consultant, Washington V.A. Hospital; Bethesda Navy Hospital; Walter Reed Army Medical Center; National Heart and Lung Institute, Washington, D.C.

Frank L. Iber, M.D. Professor of Medicine, University of Maryland; Chief of Gastroenterology, University Hospital, Baltimore; Chief of Gastroenterology, Loch Raven

Veterans Administration Hospital, Baltimore, Maryland.

J. WARD KENNEDY, M.D. Associate Professor of Medicine, University of Washington; Chief, Department of Cardiology, Veterans Administration Hospital, Seattle, Washington.

JOHN H. KILLOUGH, Ph.D., M.D. Associate Professor of Medicine and Associate Dean, Jefferson Medical College; Attending Physician, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania.

JOSEPH B. KIRSNER, M.D., Ph.D. Louis Block Professor of Medicine, Deputy Dean for Medical Affairs, University of Chicago; Attending Physician, A. M. Billings Hospital,

University of Chicago, Chicago, Illinois.

HERBERT C. MANSMANN, JR., M.D. Professor of Pediatrics, and Associate Professor of Medicine, Jefferson Medical College; Medical Director, Children's Heart Hospital of Philadelphia; Pediatrician, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania.

DEAN T. MASON, M.D. Professor of Medicine, Professor of Physiology, Chief, Section of Cardiovascular Medicine, Departments of Medicine and Physiology, University of

California, School of Medicine, Davis, California.

RASHID A. MASSUMI, M.D. Professor of Medicine, Director, Electrophysiology, Section of Cardiovascular Medicine, Department of Medicine, University of California, School of Medicine, Davis, California.

PAUL H. MAURER, B.S., Ph.D. Professor of Biochemistry and Chairman of the Department, Jefferson Medical College of Thomas Jefferson University, Philadelphia,

Pennsylvania.

WILLIAM W. PARMLEY, M.D. Associate Professor of Medicine, UCLA School of Medicine; Associate Director, Department of Cardiology, Cedars-Sinai Medical Center, Los Angeles, California.

Spencer O. Raab, M.D. Associate Professor of Medicine, University of Arkansas School of Medicine; Attending Physician, University Hospital; Consultant (Hematology), Veterans Administration Hospital, Little Rock, Arkansas.

WILLIAM D. ROBINSON, M.D. Professor and Chairman, Department of Internal Medi-

cine, University of Michigan Medical Center, Ann Arbor, Michigan.

DAVID B. SKINNER, M.D. Dallas B. Phemister Professor and Chairman, Department of Surgery, University of Chicago; Chief of Surgery, University of Chicago Hospitals and Clinics (Billings Hospital), Chicago, Illinois.

WILLIAM A. SODEMAN, JR., M.D., F.A.C.P. Associate Professor of Medicine; Chief of the Division of Gastroenterology, Medical College of Ohio at Toledo, Toledo, Ohio.

JOHN F. STAPLETON, M.D. Professor of Medicine, Georgetown University Medical School; Medical Director, Georgetown University Hospital, Washington, D.C.

H. J. C. SWAN, M.D., Ph.D. Professor of Medicine, UCLA School of Medicine; Director, Department of Cardiology, Cedars-Sinai Medical Center, Los Angeles, California. MORTON N. SWARTZ, M.D. Professor of Medicine, Harvard Medical School; Chief, Infectious Disease Unit, Massachusetts General Hospital, Boston, Massachusetts.

ROBERT C. TARAZI, M.D. Staff Member, Research Division, Cleveland Clinic Foundation, Cleveland, Ohio. 1 12

MILTON TOPOREK, B.A., M.A., Ph.D. Professor of Biochemistry, Jefferson Medical College of Thomas Jefferson University, Philadelphia, Pennsylvania.

YOSHIO WATANABE, M.D. Professor of Medicine, Fujita Garken University School of Medicine, Toyo-Ake-Shi, Japan.

DAVID W. WATSON, M.D. Associate Professor of Medicine, University of California, Davis; Attending Physician, Sacramento Medical Center; Consultant in Gastro-

enterology, San Joachin General Hospital, California.

LOUIS WEINSTEIN, Ph.D., M.D. Professor of Medicine, Tufts University School of Medicine; Lecturer in Medicine, Harvard Medical School; Chief, Infectious Disease Service, Tufts-New England Medical Center; Associate Physician, Medical Service, Massachusetts General Hospital, Boston, Massachusetts.

JOHN M. WELLER, M.D. Professor of Internal Medicine, University of Michigan; Director, Nephrology Division, University Hospital, Ann Arbor, Michigan.

K. LEMONE YIELDING, M.D. Professor, Biochemistry, University of Alabama School of

Medicine, Birmingham, Alabama.

ROBERT ZELIS, M.D. Associate Professor of Medicine, Associate Professor of Physiology, Director, Clinical Physiology and Cardiac Catheterization Laboratories, Section of Cardiovascular Medicine, Departments of Medicine and Physiology, University of California, School of Medicine, Davis, California.

enterplays were concluded for the two set of Machine, This parties belond an Marken Level of the enterplay of the deficient This parties belond to Marken Level of the enterplay of the enterplay

the first that Aprended the armin Made of their Constitution is not

and the tribute of the property of the management of the property of the prope

This edition of Pathologic Physiology, the fifth, represents a complete rewriting of the text. This restructuring coincides with an extensive turnover in the list of contributors. It is in the spirit of the dramatic changes and staggering advances in medical science that the editors accepted the fact that many of the contributors who had remained with the book since the First Edition in 1950 should now give way to a younger group. Thus, the text has undergone a complete rewriting and reorientation of approach, especially in fields such as immunology, genetics, and molecular biology. Importantly, these new contributors have joined with us in capturing the concept presented in the First Edition, the goal of the text being to present and interpret the clinical picture of disease and the genesis of symptoms and signs as physiologic dysfunctions.

The W. B. Saunders Company and its staff have been more than patient and cooperative as the work has progressed. We are grateful and thank them. The Editors also wish to thank their wives, Marjorie Christian Sodeman and Mary Agnes Sodeman, for their support and tolerance as the work on the text and the reading of manuscripts progressed.

WILLIAM A. SODEMAN, JR., M.D. WILLIAM A. SODEMAN, SR., M.D.

PREFACE TO THE FIRST EDITION

This volume, a collaborative effort by 25 authors, approaches problems of disease in the field of internal medicine from the standpoint of disturbed physiology. Unlike the usual text, which is devoted to discussions of etiology, pathology, symptoms and treatment, this work analyzes symptoms and signs and the mechanisms of their development. The monograph is not intended to take the place of standard texts on physiology or textbooks of medicine. It does not aim at the completeness of either, but does try to bridge the gap between them by presenting a clinical picture of disease seen as physiologic dysfunction. An attempt is made to promote understanding of how and why symptoms appear, so that the student or physician may have a reasonable explanation for the findings he elicits. Neurologic problems are considered only as they are related to the various disease groups. The same is true of metabolic disturbances and disorders of acid-base balance.

The Editor thanks the contributors for their ready cooperation in covering certain aspects of disease in which presentation of material is at times most difficult. He thanks the Saunders Company for their help and guidance, and also Miss Brent S. Robertson for her long hours of hard work and patience in reading and checking manuscripts.

WILLIAM A. SODEMAN, M.D.

d cyna)

de tal<mark>ingm</mark> ette kab<mark>i</mark>m. Gordschettet tätteen begit et gotte.

É LAMBACO FOLITORIA E NEL CONTRO O MICAGO O MAGAZO EL POLATORIO DE POL

Value terreit on off reconstant according to

.*				HELD TRANSFORMEN A COMPANY
SEC	TION I. SCIENTIFIC FOUND			
N	Chapter 1			the second
MET	ABOLIC BIOCHEMISTRY			in i marataret en englis Santo North
	Chapter 2			No. of the second second
Moli	ECULAR BIOLOGY		54****	
	K. Lemone Yielding	. "	** * 127 * 5. \$	en e
	Chapter 3			El aigers
MED	Benjamin R. Gendel and Louis J. Elsas, II	e fire the same	ingragation of	
	Chapter 4			h. Tagaid
[₩₩	Peter Abramoff and René J. Duquesnoy	<i>ભૂત</i> ધૂર અને ક પોર્કાસિક અલે કર્યો		til eretsesse tsjeckskeisere i 97 SNN 1881 NOVER
	Chapter 5			
Import	UNODEFICIENCY DISEASES AND TUMOR René J. Duquesnoy and Peter Abramoff	Тимп иові		
	TION II. CARDIORENAL AN	ID RESPII		BAARRI TABBAR
	Chapter 6		1,	Line 1 Rulen
Inte	GRATIVE HEMODYNAMICS			149
	Arthyr Groygon All Yes Liba Yes			я стои ш
Sveri	Chapter 7 EMIC ARTERIAL PRESSURE	· ·		T
31811	Robert C. Tarazi and Ray W. Gifford, Jr.		33	Adamadda. 177 XBS(U arxiddadd Nathally y U

Chapter 8	
MECHANISMS OF CARDIAC CONTRACTION: STRUCTURAL, BIOCHEMICAL, AND FUNCTIONAL RELATIONS IN THE NORMAL AND DISEASED HEART	206
Dean 1. Mason, Robert Zetts, Ezra A. Amsterdam, and Rashda A. Massama	
Chapter 9	
CARDIAC OUTPUT, CARDIAC PERFORMANCE, HYPERTROPHY, DILATATION, VALVULAR DISEASE, ISCHEMIC HEART DISEASE, AND PERICARDIAL DISEASE	235
Harold T. Dodge and J. Ward Kennedy	
Chapter 10	
Congestive Heart Failure	273
H. J. C. Swan and William W. Parmley Chapter 11	
HEART SOUNDS, MURMURS, AND PRECORDIAL MOVEMENTS	295
Chapter 12	
THE ELECTROCARDIOGRAM	312
J. A. Abildskov	
Chapter 13	
ARRHYTHMIAS – MECHANISMS AND PATHOGENESIS	329
Chapter 14	
RENAL DISEASE: WATER AND ELECTROLYTE BALANCE	345
Chapter 15	
PULMONARY VENTILATION AND BLOOD GAS EXCHANGE	371
Chapter 16	
PROTECTIVE MECHANISMS OF THE LUNGS; PULMONARY DISEASE; PLEURAL DISEASE	393
John H. Killough	
SECTION III. RHEUMATOLOGY, ALLERGY, INFECTIOUS DISEASE, AND HEMATOLOGY	
Chapter 17	
RHEUMATIC DISEASES	117
William D. Rahinsan	

Chapter 18	88 mm
ALLERGY: ITS NATURE AND RELATION INDUCED DISEASE STATES	
Herbert C. Mansmann	1
Chapter 19	CA He HO
PATHOGENIC PROPERTIES OF INVADIA	Mast (Mont and Al) to You do Richer His 15 and 457
Louis Weinstein and Morton N. Swa	rtz 170000103AVISMIS. 1700 VISCO
Chapter 20	$\mathbf{C}^{\xi_{i}} + \mathbf{q}_{i,i}$)
HOST RESPONSES TO INFECTION	62.9 ค.ศ. 6 พ.ศ. 60 - ค.ศ. 62 - ค.ศ. 473
Louis Weinstein and Morton N. Swa	
Chapter 21	A Company
PATHOPHYSIOLOGIC CHANGES DUE TO	DECALIZATION OF
INFECTIONS IN SPECIFIC ORGANS	
Louis Weinstein and Morton N. Swan	rtz \$ £ - 40,000
Chapter 22	・ 「「「・・・・・・・・・・・・・・・・・・・・・・・・・・・・・・・・・
•	Disorders 511
Allan J. Erslev and Thomas G. Gabu	•
NO CHEMICAL 23 ADIVERD CH	SECTION IN TOXIC PHYSICAL
THE SPLEEN AND RETICULOENDOTHER	LIAL SYSTEM
Spencer O. Raab	Breach in the River
SECTION IV GASTPOENTE	ROLOGY, ENDOCRINOLOGY,
AND METABO	et ainne?
948 WEIND	CHARLES OF THE STATE OF DESIGNARY
Chapter 24	Compared to the second
The Esophagus	697
Tree David B. Skinner	NDP
Chapter 25	
THE STOMACH	700
Joseph B. Kirsner	
gampa 2. Italian	
Chapter 26	
THE SMALL INTESTINE	
David W. Watson and William A. Son	
Chapter 27	
THE LARGE INTESTINE	
William A. Sodeman, Jr., and David V	

Chapter 28	Section 1995 - Secti
•	plocy of the Liver 790
F. L. Iber	the property of the second
Chapter 29	
PATHOPHYSIOLOGY OF GALLBLADD	ER DISEASE 818
Chapter 30	
PATHOPHYSIOLOGY OF THE PANCE Franz Goldstein	EAS
Chapter 31	
NUTRITIONAL FACTORS IN DISEAS	E
George A. Bray Chapter 32	 And the second of the experience of the second of the secon
·	865
Thomas W. Rurns	The Model of Society (Mark 1997) and the Society of the Society (Mark 1997) and the Society of t
***	SICAL AND CHEMICAL AGENTS
Effects of Physical Agents Charles E. Billings	917
YOC. 3490098 Chapter 34 CHEMICAL AGENTS AND DISEASE.	SOCIMER ON CASTROCHTSPOLOK
UHEMICAL AGENTS AND DISEASE . Bertram D. Dinman	949
Index	and the second s
INDEX	
4.C.,	File Appelled to the second of
	Bury 26
-87 · · . · . · . · . · . · . · . · .	Constitution of the second section of the
	re _{gories} .
en e	and the second of the second o

SECTION I

SCIENTIFIC FOUNDATIONS

CHAPTER 1

METABOLIC BIOCHEMISTRY

MILTON TOPOREK PAUL H. MAURER

The ultimate goal of metabolic biochemistry or intermediary metabolism is to determine all the reactions undergone by the various molecules which enter the body, by normal or other means, from the time they enter until they or their derivatives leave the body. Although much has been accomplished in this field in the last 25 years. since radioactive isotopes became available, much more remains to be learned. However, much of the information available has already provided the medical profession with many useful procedures for diagnosis and therapy based on objective criteria of clinical laboratory determinations which, when compared to normal metabolic patterns, can indicate the health status of the patient. It is therefore the purpose of this chapter to review, in limited form, the major metabolic pathways, with some examples of abnormal changes correlated with clinical conditions.

CARBOHYDRATE METABOLISM

A general summary of carbohydrate metabolism is presented in Figure 1-1. This diagram shows the possible uses of glucose in the liver and muscle to be as follows:

Liver
Storage as glycogen
Oxidation for energy

purposes

Muscle
Storage as glycogen
Oxidation for energy
purposes

Conversion to other metabolites:

Fat Amino Acids Other carbohydrates

USES OF GLUCOSE

Storage as Glycogen (Glycogenesis)

Glucose not used for other purposes as listed below may be converted to glycogen and deposited in the liver or muscle tissues in relatively small and limited amounts. Remaining available glucose is disposed of in pathways discussed below.

Oxidation for Energy Purposes

Complete oxidation of glucose to CO2 and H2O can furnish energy for the body as required. This is accomplished in two stages: (1) glycolysis (Embden-Meyerhof pathway), taking glucose to the 3-carbon pyruvate or lactate point; and (2) the tricarboxylic acid cycle (Krebs, or citric acid cycle), which converts the 3-carbon pyruvate to CO2 and H2O. Under anaerobic conditions, as in muscle after prolonged or strenuous exercise, glycolysis produces lactic acid, a reduction product of pyruvic acid. By way of the blood, the lactic acid reaches the liver for reprocessing or further oxidation. Under aerobic conditions, lactate is converted back to pyruvate, and the oxidation of pyruvate is completed in liver and muscle by way of the tricarboxylic acid cycle.

Liver and adipose tissue can also degrade glucose by the pentose phosphate pathway (pentose shunt), a pathway which is relatively unimportant in skeletal muscle.

Conversion to Other Metabolites

Fut. Excess glucose may be converted to fatty acids and glycerol and deposited as triglycerides

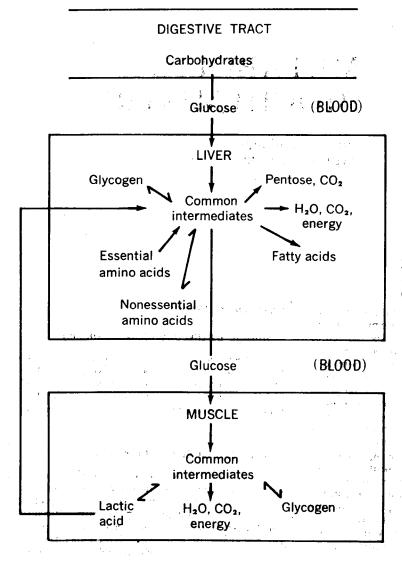


Figure 1-1 General summary of carbohydrate metabolism. (From Toporek: Basic Chemistry of Life, 1968. Courtesy of Appleton-Century-Crofts, Inc.)

in adipose tissue, a process which, to the sorrow of many, is unlimited except by restrictions on intake of carbohydrates. As will be noted later, there are certain features common to the metabolic pathways of glucose and the fatty acids, but the conversion of glucose to fatty acids is irreversible on the basis of energy accounting, i.e., it would require a net expenditure of energy to convert fatty acids to glucose.

Amino Acids. The carbon skeletons of the nonessential amino acids may be derived from glucose in a reversible process. The essential amino acids can also contribute to the carbon skeleton of glucose, but the reverse is not possible in this case. Other Carbohydrates. Some glucose is used for the synthesis of other important sugars such as ribose and deoxyribose, components of the nucleic acids, and galactose, a component of cerebrosides, gangliosides, and glycolipids.

OXIDATION OF GLUCOSE

The complete oxidation of glucose is divided into two major phases: (1) glycolysis (Embden-Meyerhof pathway), an anaerobic pathway (does not require oxygen but can occur in its presence) which includes glycogenesis and glycogenolysis, interconversions of galactose, fructose, and glucose, and ends with the production of pyruvate (under aerobic conditions) or lactate (under

anaerobic conditions) and a small amount of energy; and (2) the tricarboxylic acid cycle (Krebs cycle, citric acid cycle), an aerobic pathway which completes the oxidation of glucose to H₂O and CO₂, with the production of a much larger amount of energy.

Glycolysis

The metabolic reactions of glycolysis are outlined in Figure 1-2. The solid line arrows indicate the reactions in the direction of glycogenesis and the anaerobic metabolism of glucose to the pyruvate or lactate stage (glycolysis). A singleheaded arrow (-----) indicates a reaction which is irreversible as written. A double-headed arrow (indicates a reversible reaction. A broken arrow (----) indicates a reaction which has the effect of reversing an irreversible reaction by a reaction which is different from the forward reaction. Thus, there is complete biologic reversibility, but it occurs at the price of a loss of energy, i.e., since the reactions in the direction of glucose to pyruvate generate energy, the reactions in reverse must require an input of energy. The individual reactions are listed below by numbers corresponding to those in Figure 1-2.

Reactions to Glycolysis

Reaction 1-Glucose is converted to glucose-6-phosphate (G-6-P) under the influence of hexokinase and adenosine triphosphate (ATP). This is an irreversible reaction because a high-energy phosphate group in ATP is used to form a low-energy ester phosphate bond in G-6-P.

Reaction 2—G-6-P is converted back to glucose in the liver by glucose-6-phosphatase, not by reversal of reaction 1. Muscle does not have this phosphatase.

Reaction 3-G-6-P is converted to glucose-1-phosphate (G-1-P) by phosphoglucomutase.

Reaction 4-This summarizes a series of reactions resulting in the formation of glycogen, as follows:

G-1-P + UTP Pyrophosphorylase UDPG + pyrophosphate

 $\frac{\text{Glycogen symmetase}}{\text{Branching enzyme}} \quad \text{Glycogen} + \text{UDP}$

UDP + ATP Phosphokinase UTP + ADP

UTP = uridine triphosphate UDP = uridine diphosphate

UDPG = uridine diphosphate-glucose

Glycogen synthetase: forms 1,4-linkages between glucose moieties, making straight chains

Branching enzyme: forms 1,6-linkages between glucose moieties, making branches between straight chains

Reaction 5 – Glycogen is broken down to G-1-P in the presence of a debranching enzyme (breaks 1,6-linkages) and phosphorylase (breaks 1,4-linkages). Reaction 3 converts G-1-P to G-6-P. If required for blood sugar, G-6-P can be converted back to glucose by reaction 2.

Reaction 6-G-6-P is converted to fructose-6-phosphate (F-6-P) in a reversible reaction catalyzed by phosphohexose isomerase.

Reaction 7-F-6-P is phosphorylated to fructose-1,6-diphosphate (F-1,6-P₂) in an irreversible kinase reaction in the presence of phosphofructokinase.

Reaction 8—Reaction 7 can be biologically reversed by way of a phosphatase reaction.

Reaction 9 – F-1,6-P₂ is split into two triose phosphate molecules, glyceraldehyde-3-phosphate (glycerald-3-P) and dihydroxyacetone phosphate [(OH)₂-acetone-P], in a reversible reaction catalyzed by fructose diphosphate aldolase.

Reaction 10—The triose phosphates, glycerald-3-P and (OH)₂-acetone-P, are in equilibrium with each other in the presence of an isomerase. Thus, both halves of the original glucose molecule are available for subsequent reactions.

Reaction 11—The conversion of glycerald-3-P to 1,3-diphosphoglycerate (1,3-P₂-glycerate) is catalyzed by glycerald-3-P-dehydrogenase in a reversible reaction. The carboxyl phosphate bond is a high-energy bond, the energy coming from the oxidation of the aldehyde to a carbo yl group.

Reaction 1.—In the conversion of 1,3-P₂-glycerate to 3-phosphoglycerate (3-P-glycerate) under the influence of phosphoglycerate kinase, the energy of the carboxyl phosphate bond is captured in the concomitant conversion of ADP to ATP.

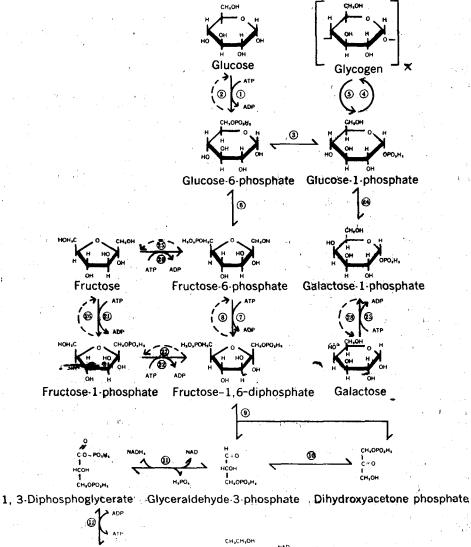
Reaction 13-3-P-glycerate forms 2-P-glycerate in the presence of phosphoglyceromutase in a reversible reaction.

Reaction 14—The dehydration of 2-P-glycerate to phosphoenolpyruvate (P-E-pyr) with the production of a high-energy phosphate bond is catalyzed by enolase.

Reaction 15 - The high energy in P-E-pyr is captured in the formation of ATP from ADP in an irreversible reaction in the presence of pyruvate kinase, resulting in the formation of pyruvate.

Reaction 16—Under anaerobic conditions, pyruvate is converted to lactate in the presence of lactate dehydrogenase in a reaction which reverses itself under aerobic conditions. This concludes the glycolysis reactions.

Reaction 17-In the liver, reaction 15 can be



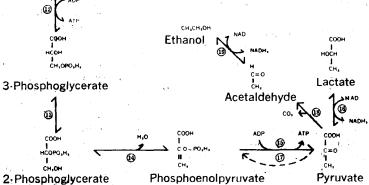


Figure 1-2 Anaerobic metabolism of glucose (glycolysis) and related hexoses. (~ = high energy bond.) (From Toporek: Basic Chemistry of Life, 1968. Courtesy of Appleton-Century-Crofts, Inc.).