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[+] Principles of Operation

[+] Conventional Positive-Pressure Ventilation

[+] Disease-Oriented Mechanical Ventilation Strategies

[+] Monitoring of the Mechanically Ventilated Patient

Complications Associated with Intermittent Positive-Pressure Ventilation

References

[+] Figures

[+] 59 - Mechanical Ventilation Part II: Noninvasive

[+] 60 - Mechanical Ventilation Part III: Discontinuation

[+] 61 - Gas Embolism Syndromes: Venous Gas Emboli, Arterial Gas Emboli, and Decompression Sickness

[+] 62 - Respiratory Adjunct Therapy

[+] 63 - Chest Radiographic Examination

37. Hilbert G, Gruson D, Vargas F, et al: Noninvasive ventilation in immunosuppressed patients with pulmonary infiltrates, fever, and acute respiratory failure. *N Engl J Med* 344:481–487, 2001.

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38. Girou E, Schortgen F, Delclaux C, et al: Association of noninvasive ventilation with nosocomial infections and survival in critically ill patients. *JAMA* 284:2361–2367, 2000.

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39. Azoulay E, Alberti C, Bornstain C, et al: Improved survival in cancer patients requiring mechanical ventilatory support: impact of noninvasive mechanical ventilatory support. *Crit Care Med* 29:519–525, 2001.

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40. Brochard L: Non-invasive ventilation for acute exacerbations of COPD: a new standard of care (editorial). *Thorax* 55:817–818, 2000.

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42. Antonelli M, Conti G, Rocco M, et al: A comparison of noninvasive positive-pressure ventilation and conventional mechanical ventilation in patients with acute respiratory failure. *N Engl J Med* 339:429–435, 1998.

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Improved survival in cancer patients requiring mechanical ventilatory support: Impact of noninvasive mechanical ventilatory support

[Clinical Investigations]

Azoulay, Elie MD; Alberti, Corinne MD; Bornstain, Caroline MD; Leleu, Ghislaine MD; Moreau, Delphine MD; Recher, Christian MD; Chevret, Sylvie MD PhD; Le Gall, Jean-Roger MD; Brochard, Laurent MD PhD; Schlemmer, Benoît MD

From the Medical Intensive Care Unit (Drs. Azoulay, Bornstain, Leleu, Moreau, Recher, Le Gall, and Schlemmer) and the Department of Biostatistics (Dr. Chevret), Saint Louis University Hospital and University Paris 7; and U444-INSERM, Medical Intensive Care Unit (Dr. Brochard), Henri Mondor University Hospital and University Paris XII. Assistance publique des hôpitaux de Paris, France.

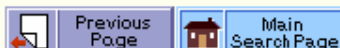
Upstream management of critically ill cancer patients may increase the chances of longer survival with an acceptable quality of life.

Presented, in part, at the Congress of The French Society of Critical Care Medicine in January 2000.

Address requests for reprints to: Elie Azoulay, MD, Medical ICU, Saint Louis University Hospital, 1 avenue Claude Vellefaux, 75010 Paris, France. E-mail: elie.azoulay@sls.ap-hop-paris.fr

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 - [End Points.](#)
 - [Statistical Analysis.](#)
- [RESULTS](#)
 - [Cohort Study](#)
 - [Chronic Health Status and Co-Morbidities.](#)
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Chapter: WOMEN AND CORONARY ARTERY DISEASE

Passage: ... on diagnosis begins with discussion of the ways that CAD presents in asymptomatic women and then reviews models to assess CAD risk with attention

□ 1. **Source:** *Hurst's The Heart*

Editors: Valentin Fuster, R. Wayne Alexander, Robert A. O'Rourke

Publisher: McGraw-Hill, 2001

Chapter: WOMEN AND CORONARY ARTERY DISEASE

Passage: ... on diagnosis begins with discussion of the ways that CAD presents in asymptomatic women and then reviews models to assess CAD risk with attention to gender. Finally, symptomatic CAD in women is discussed, including the management of the women with angina and myocardial infarction. The chapter closes with an update on gender differences in sudden death. **PREVENTION: GENDER-SPECIFIC ISSUES** It is especially important to identify women at high risk for CAD for possible primary prevention. Young women with coronary artery mortality⁴ are more likely to have a history of tobacco exposure, obesity, hypertension, diabetes, early menopause, or, less often, cocaine abuse.⁵ These risk factors are also important in predicting nonfatal myocardial infarction.¹ Gender differences are reviewed in ...

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□ 2. **Source:** *Oxford Textbook of Clinical Nephrology*

Editors: Alex M. Davison, J. Stewart Cameron, Jean-Pierre Grunfeld, David N.S. Kerr, Eberhard Ritz, Christopher G. Winearls

Publisher: Oxford University Press, 1998

Chapter: Clinical approach to hypertension

Passage: ... over what the target blood pressure should be in treating essential hypertension since it has been suggested that there is a J-shaped curve relating mortality to treated blood pressure. In patients with known ischaemic heart disease the least mortality was seen when the diastolic blood pressure was 85 to 90 mmHg and was significantly greater when the diastolic blood pressure was less than 85 mmHg (Cruickshank et al. 1987). However, a similar pattern was seen in the 'Heart attack primary prevention in hypertension' study, from which patients with ischaemic heart disease were excluded (Wilhelmsen et al. 1987), and others have also been unable to relate increased mortality at lower diastolic pressures to cardiovascular complications at entry (Staessen et al. 1989). It has ...

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□ 3. **Source:** *Drugs in Pregnancy and Lactation*

Editors: Gerald G. Briggs, Roger K. Freeman, Sumner J. Yaffe

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Chapter: TIMOLOL

Passage: ... after myocardial infarction, for the prophylaxis of migraine headache, and topically for the treatment of glaucoma.

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□ 4. **Source:** *Novak's Gynecology*

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Publisher: Lippincott Williams & Wilkins, 2002

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Passage: ... small molecule in biology (33). The dietary influence of cholesterol on atherosclerosis and its relationship to hypertension and cardiovascular events (myocardial infarction and stroke) has been widely debated in both the scientific and lay communities (34). The controversy centers on the influence of dietary cholesterol in risk assessment and prevention of cardiovascular disease (35). Many assume that all cholesterol and fat in the diet have negative health consequences. Furthermore, cholesterol metabolism is complex and our understanding in some cases is extrapolated from animal models. The role of cholesterol testing (who, when, and at what age) is hotly debated among health care professionals. Cholesterol testing is fraught with multiple variables that affect results. The ...

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TABLE OF CONTENTS

[+] I - Procedures and Techniques

[+] II - Cardiovascular Problems and Coronary Care

[-] III - Pulmonary Problems in the Intensive Care Unit

[-] 46 - Respiratory Failure Part I: A Physiologic Approach to Managing Respiratory Failure

[+] Overview

[+] Respiratory Acid-Base Disorders

[+] Clinical Approach to

Conclusions

References

[+] Tables

[+] 47 - Respiratory Failure Part II: The Acute Respiratory Distress Syndrome and Pulmonary Edema

[+] 48 - Respiratory Failure Part III: Status Asthmaticus

[+] 49 - Respiratory Failure Part IV: Chronic Obstructive Pulmonary Disease

[Back](#)

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125

Anticonvulsants

Cynthia K. Aaron

Anticonvulsants can be divided into four groups based on their primary mechanism of action: those that primarily act on neuronal membranes (membrane-active agents), those that act on neurotransmitters or their receptor sites (synaptic agents), those with multiple sites of action, and those that are not yet understood. Membrane-active agents alter ion fluxes and include carbamazepine (CBZ), oxcarbazepine, ethosuximide, zonisamide, phenytoin, and lamotrigine (LTG). Synaptic agents primarily affect the activity of gamma-aminobutyric acid (GABA) and include barbiturates, benzodiazepines, gabapentin (GBP), tiagabine, and vigabatrin. Agents that have multiple sites of action include valproate, GBP, felbamate, and topiramate, and those for which mechanisms of action still are not understood are levetiracetam, stiripentol, and remacemide [1,2 and 3]. Barbiturates and benzodiazepines are discussed in [Chapter 149](#). The precise mechanisms of action of many of the newer anticonvulsants also remain unknown. Even within groups, the site or mechanism of action may differ. Pharmacologic differences are important from a therapeutic standpoint. In the treatment of seizures, combining agents from different groups may be effective whenever a single agent is ineffective or requires a toxic dose for efficacy. Such therapeutic synergism may also occur when different agents of the same group are combined (e.g., benzodiazepines and barbiturates).

Phenytoin

Phenytoin (diphenylhydantoin) is the most commonly used anticonvulsant medication [4]. It is also used in the treatment of trigeminal neuralgia. Phenytoin was the antidysrhythmic of choice for digitalis toxicity before the advent of digitalis Fab fragments [5]. Iatrogenic intoxications may occur with drug interactions because distribution, protein binding, and clearance of phenytoin are affected by other medications and disease states. Toxicity may occur when the daily administered dose exceeds endogenous metabolism and elimination [6,7].

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[+] XII - Neurologic Problems in the Intensive Care Unit

[+] XIII - Transplantation

[+] XIV - Metabolism and Nutrition

[+] XV - Rheumatologic and Immunologic Problems in the Intensive Care Unit

[+] XVI - Psychiatric Issues in the Intensive Care Unit

[+] XVII - Moral, Ethical, Legal, and Public Policy Issues in the Intensive Care Unit

BACK OF BOOK

[-] Resources - References

[-] Appendix

[+] Calculations Commonly Used in Critical Care

[-] Index

A	B	C	D
E	F	G	H
I	J	K	L
M	N	O	P
Q	R	S	T
U	V	W	X
Y	Z		

Mallory-Weiss tear

gastrointestinal hemorrhage in [1091-1092](#)

iatrogenic [1136](#)

Malnutrition [2053-2054](#)

assessment of [2053-2054](#) [2059](#)

in renal failure [2071](#)

hypoglycemia in [1201](#)

in liver disease [2072-2073](#)

pancreatitis in [1677](#)

protein-calorie [2057](#)

in liver failure [2074](#) [2075](#)

in renal failure [2069](#)

in renal failure [2069](#) [2071](#)

respiratory disorders in [801](#) [801](#) [2075](#)

nutritional therapy in [807](#)

in respiratory failure [2075](#)

survival time in [2058-2059](#)

Wernicke-Korsakoff syndrome in [750](#) [1875](#) [1882](#)

Managed care systems [2249](#)

decision-making process in [2220-2221](#)

Mania

in cancer patients [2203](#)

in HIV infection and AIDS [2207](#)

lithium therapy in [1508](#)

Manikin training on CPR, prevention of infection transmission in [262](#)

Mannitol [843](#) [843](#)

in brain tumors [1895](#)

in cerebral ischemia [1890](#)

in compartment syndrome of extremities [1852](#)

dosage of [845](#) [1791](#)

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[+] XIII - Transplantation

[+] XIV - Metabolism and Nutrition

[+] XV - Rheumatologic and Immunologic Problems in the Intensive Care Unit

[+] XVI - Psychiatric Issues in the Intensive Care Unit

[+] XVII - Moral, Ethical, Legal, and Public Policy Issues in the Intensive Care Unit

BACK OF BOOK

[-] [Resources - References](#)[-] [Appendix](#)[+] [Calculations Commonly Used in Critical Care](#)

[-] Index

A	B	C	D
E	F	G	H
I	J	K	L
M	N	O	P
Q	R	S	T
U	V	W	X
Y	Z		

entity [75] or instances of renal failure enhancing intercurrent disorders that predispose to hypoglycemia [76].

P.1201

These might include drug ingestion, liver disease, and adrenal or pituitary insufficiency.

Fasting Hypoglycemia Due to the Unavailability of Gluconeogenic Substrate.

The prototypic condition in which substrate deficiency leads to hypoglycemia is ketotic hypoglycemia of childhood [77]. Patients with this condition, a variant of the normal response to starvation, are usually diagnosed between 18 months and 5 years of age. The hallmark of the condition is a low basal blood concentration of the gluconeogenic precursor alanine, and the hypoglycemia can be corrected by glucose or by infusion of alanine.

In adult populations, low levels of alanine are associated with chronic renal disease, and extrapolation from animal data suggests that glucocorticoid deficiency may result in the suboptimal release of alanine and other gluconeogenic precursors from muscle. Severe malnutrition from any cause diminishes the supply of gluconeogenic precursors and can lead to hypoglycemia. In severe malnutrition, glycogen, fat, and lean body mass are reduced, adding to the propensity to develop hypoglycemia.

OTHER CAUSES***Sepsis.***

Sepsis has occasionally been implicated as a cause of hypoglycemia [78,79,80 and 81]. Shock and liver failure were concurrent problems in some reported cases [79]. Under conditions of decreased hepatic reserve, the combination of circulatory failure and impairment of gluconeogenesis by endotoxin might be postulated to lead to hypoglycemia.

Septic hypoglycemic patients are often acidotic, and the fatality rate is high [78,79]. In one study, only 1 of 15 such patients survived 1 month after the onset of hypoglycemia and hypotension [79].

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- Home
- Clinical
- Drug Reference
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- PREFACE

- ACKNOWLEDGMENTS

TABLE OF CONTENTS



[\(+\)](#) PART 1 - ESSENTIALS OF MODERN ONCOLOGIC SCIENCE

[\(+\)](#) PART 2 - PRINCIPLES OF ONCOLOGY

[\(+\)](#) PART 3 - PRACTICE OF ONCOLOGY

[\(+\)](#) PART 4 - NEWER APPROACHES IN CANCER TREATMENT

BACK OF BOOK

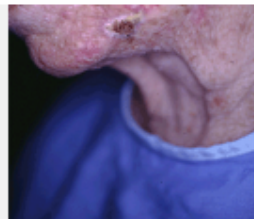


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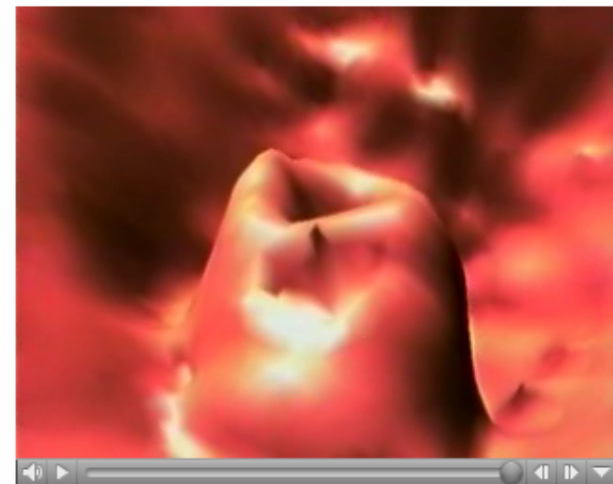
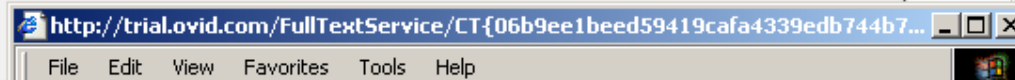
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[+] Tables

[+] 11 - Infant Dental Care

[+] 12 - Normal Speech and Language Development

[+] 13 - Normal Motor and Cognitive Development

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























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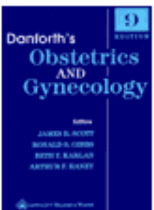



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