The PHYSICIAN'S BOOK OF LISTS

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Churchill Livingstone

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Preface

Everyone makes lists—it has, in recent years, even become something of a national pastime—but few do so with the fervor and sheer profusion as physicians. Whether hastily scribbled on a blackboard during rounds or meticulously copied into a little black book and stuffed into one's jacket pocket, lists comprise a fundamental part of one's clinical armamentarium: differential diagnoses, signs and symptoms, drug interactions and side effects, diagnostic protocols, etiologic considerations. Their number is limitless, a function of the vast wealth of clinical information and the urgent necessity to organize it all into focused, functional portions.

Lists are efficient and effective. However, they contain little clinical judgment. A list merely provides terse data to be processed within the framework of clinical judgment. One must bring one's education and experience *to* a list. Only then can the dangers of oversimplification and missed subtleties be avoided. Lists jog the memory, organize one's thoughts, clarify one's approach. They do not in themselves educate or instill clinical acumen. Lists are merely the tersest condensation of ideas which are developed, defended, and given context elsewhere.

Nevertheless, used with care, lists make better, more confident physicians of us all. We have tried here to select those lists that have proven most useful for us and for many other active clinicians. This book is hardly exhaustive. It could easily have been ten times as large and the paring down to portable size was often a difficult process. We hope the end result addresses a wide sampling of the key questions encountered in the everyday practice of medicine. The emphasis is clearly on diagnosis and approach. Therapeutics are

represented only sporadically, largely because of the large number of outstanding therapeutic manuals already available.

We view this book as an on-going project, and hope that you will send us some of your favorite lists for possible inclusion in future editions. Please send them to:

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We wish to give special thanks to Mr. Lewis Reines, who conceived this project and whose warm humor, gentle guidance, and deft prodding made this book a reality. Acknowledgements plunge too readily into the sentimental, but to Lew we simply cannot overstate our appreciation and feelings of friendship. We would also like to express our deepest gratitude to our families and friends for their constant support, and especially to Else Smedemark and Nancy Klinghoffer, whose love and encouragement are the best medicine of all.

M.T.
D.M.

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Cardiology

IMPORTANT CAUSES OF SHOCK

1. Cardiogenic
   a. Myocardial infarction (15% of acute MIs)
   b. Valvular disease (especially acute mitral regurgitation)
   c. Congestive failure
   d. Arrhythmias (especially ventricular tachycardia)
   e. Obstructive disease (especially pulmonary embolism, pneumothorax and cardiac tamponade)
   f. Toxic: (1) endogenous—circulating vasoactive toxins, such as myocardial depressant factors; and (2) exogenous—cardiotoxic drug overdoses, e.g., tricylic antidepressants
2. Hypovolemic
   a. Hemorrhage
   b. Burns
   c. Trauma
   d. Ruptured aneurysm
   e. Anaphylaxis
   f. Disseminated intravascular coagulation (DIC)
   g. Pancreatitis (due to both volume loss and the release of myocardial depressant factors)
   h. Peritonitis
3. Septic (generally gram negative sepsis)
4. Other
   a. Addison's disease
   b. Neurogenic (spinal cord injuries, narcotic/barbiturate overdose)

HYPODYNAMIC VS. HYPERDYNAMIC SHOCK

	Hypodynamic Shock	*Hyperdynamic Shock*
Major causes	Cardiogenic, hypovolemic	Septic
Patient's skin	Cool, clammy	Warm, flushed
Blood pressure	Decreased	Decreased
Pulse	Increased	Increased
Cardiac output	Decreased	Increased early, decreased later
AV O_2 difference	Increased	Decreased early, increased later
Total peripheral resistance	Increased	Decreased early, increased later
Pulmonary capillary wedge pressure	Increased in cardiogenic shock Decreased in hypovolemic shock	Decreased
Central venous pressure	Increased in cardiogenic shock Decreased in hypovolemic shock	Decreased
Lactate levels	Increased	Increased

MANAGEMENT OF THE PATIENT IN SHOCK: Essential Measurements

1. Vital signs: blood pressure, pulse, temperature, respiratory rate
2. Assessment of mental status, adequacy of distal perfusion
3. Serum electrolytes, glucose, creatinine, BUN
4. Urine output and osmolality; urinary sodium concentration
5. Hemoglobin, hematocrit, white blood cell and platelet counts
6. Arterial blood gases
7. Central venous pressure (or, preferably, pulmonary capillary wedge pressure)
8. ECG and chest x-ray
9. In appropriate patients:
   a. blood cultures
   b. fibrin, fibrin split products, fibrinogen and complement levels
10. Record all fluid input and output and time and amount of all drugs

Specific Therapeutic Measures

1. Maintain intravascular volume with goal of CVP 10–12 mm Hg; PCW 18–20 mm Hg. Give whole blood, saline, or colloids as appropriate
2. Correct hypoxemia to $Po_2 > 60$ mm Hg. Use increasing FIo_2 values; intubate and use pressure cycled respirator technique if needed
3. Correct acidosis
4. If hypotension persists, use pressors. In general, isoproterenol (Isoprel) is used when generalized beta adrenergic effects are desired; dopamine (Inotropin) is used in low doses for maintaining renal perfusion, and in higher doses as a mixed alpha and beta agent; norepinephrine (Levophed) is used for its alpha effects and dobutamine (Dobutex) is used for cardiac inotropic beta stimulation.
5. Treat specific precipitants (CHF, sepsis, adrenal insufficiency, hemorrhage, etc.)

THE AMERICAN HEART ASSOCIATION'S CLASSIFICATION OF PATIENTS WITH HEART DISEASE *

Functional Classification

Class I: Patients with cardiac disease but without resulting limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.
Class II: Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.
Class III: Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnea, or anginal pain.
Class IV: Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome are present even at rest. If any physical activity is undertaken, discomfort is increased.

Therapeutic Classification

Class A: Patients with a cardiac disease whose ordinary physical activity need not be restricted.
Class B: Patients with cardiac disease whose ordinary physical activity need not be restricted, but who should be advised against severe or competitive physical efforts.
Class C: Patients with cardiac disease whose ordinary physical activity should be moderately restricted, and whose more strenuous efforts should be discontinued.
Class D: Patients with cardiac disease whose ordinary physical activity should be markedly restricted.
Class E: Patients with cardiac disease who should be at complete rest, confined to bed or chair.

*Excerpted from Diseases of the Heart and Blood Vessels—Nomenclature and Criteria for Diagnosis, 6th edition, Boston, Little Brown and Company, copyright 1964 by the New York Heart Association, Inc. These classifications are not included in the 7th edition, revised 1973, nor in the 8th edition, revised 1979.

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