LEE A. FLEISHER • STANLEY ROSENBAUM

COMPLICATIONS IN ANESTHESIA

THIRD EDITION

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All experienced educators realize that those whom we teach are also our own most enthusiastic and involved teachers. So we dedicate this volume to our residents at Penn and Yale, who have been our students, our teachers, and our colleagues.

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Preface

"Complications are the natural byproduct of the search for perfection"

The third edition of *Complications in Anesthesia,* like the two previous editions, is designed to provide practitioners of anesthesia and critical care medicine with a comprehensive source of information for a large number of complications they might be faced in clinical practice. The first two editions were edited by John L. Atlee, MD, who established an outstanding framework for addressing complications using a highly structured format of a case synopsis, problem analysis, management, and prevention. In the current edition, the chapters have been grouped into Preoperative Conditions, Procedure-Related Complications, Intraoperative Agents and Potential Complications, Equipment-Related Complications, Perioperative Events, and Pediatric Perioperative Events.

While many of these individual chapter topics were covered in previous editions, the vast majority of the authors have changed, and new topics have been added. The editors wish to acknowledge Dr. Atlee and the previous edition authors; some of the chapters were based upon their contributions to the previous edition.

It is our hope that this approach to the treatment of complications in anesthesia and critical care will serve as a reference for those currently in practice and as a tool for residents to learn how to both prepare for and manage complications.

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1 Adrenal Insufficiency

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Case Synopsis

A 68-year-old, 5-foot 10-inch, 100-kg man develops refractory hypotension toward the end of a laparotomy to remove the left colon because of recurrent diverticulitis and suspected peridiverticular abscess. The patient remains intubated at the end of the procedure and is taken to the intensive care unit (ICU), where a pulmonary artery catheter is placed and transthoracic echocardiogram (TTE) is obtained. The pulmonary artery occlusion pressure is 6 mm Hg, systemic vascular resistance is 475 dynes/cm^5, cardiac output is 10 L/min, and cardiac index is 6 L/min/m^2. TTE shows a hyperdynamic left ventricle with end-systolic cavity obliteration, a small hypercontractile right ventricle, and a small inferior vena cava with marked respiratory variations. The patient is mechanically ventilated and has a heart rate of 128 beats per minute in sinus rhythm and blood pressure of 88/42 mm Hg on infusions of norepinephrine 0.1 μg/kg per minute, epinephrine 0.1 μg/kg per minute, and vasopressin 0.03 units per minute. The patient's medical history is remarkable for hypertension and type 2 diabetes chronically treated with lisinopril and glucophage, respectively. Both were withheld on the day of surgery. Shortly after his admission to the ICU, a diagnostic test was performed and a new medication was added to the therapeutic regimen. After several hours the patient was hemodynamically stable and vasopressors had been discontinued.

PROBLEM ANALYSIS

Definition

Adrenal insufficiency (AI) is a relatively rare but potentially life-threatening condition that can be quiescent until unmasked by medical stressors such as sepsis, traumatic insults, hemorrhagic shock, or surgical stress.

Sir Thomas Addison described primary AI in 1855. Approximately a century later Harvey Cushing developed the concept of secondary AI. Causes for primary and secondary AI are listed in Box 1.1.

The hypothalamic-pituitary-adrenocortical (HPA) axis (Fig. 1.1) regulates the amount of cortisol released by the adrenals. The cycle begins with the release of corticotropin-releasing factor (CRF) from the hypothalamus, which stimulates the release of adrenocorticotropic hormone (ACTH) from the anterior pituitary. ACTH then stimulates the release of cortisol from the adrenal cortex at a rate of about 20 mg/day. Cortisol (or a synthetic analog) acts on the hypothalamus to inhibit the release of CRF and on the anterior pituitary to inhibit the release of ACTH. The associated diurnal variation in cortisol release peaks in the morning and midafternoon and then tapers off to a nadir in the evening. Although normal adults secrete about 5 to 10 mg/m^2 of cortisol (or hydrocortisone) each day, during periods of acute stress the adrenal cortex can secrete as much as 100 mg/m^2 per 24 hours.

Primary adrenal insufficiency is rare and is a result of adrenal destruction or surgical resection. Causes include autoimmune etiologies, trauma, hemorrhage, infection, infiltrative disease, or surgical removal. Secondary adrenal insufficiency develops with any process that involves the hypothalamus or pituitary and interferes with CRF and/or ACTH secretion. Tertiary adrenal insufficiency may be brought about by adrenal atrophy due to acute or chronic glucocorticoid therapy. Patients with adrenal atrophy may show no symptoms of AI; however, when subjected to the stress of even modest surgery or acute illness, these patients may develop life-threatening symptoms of AI.

Along with the classification of AI as a primary or secondary process, there is now recognition of absolute or relative AI. Classic Addison disease due to autoimmune destruction of the adrenals is an example of primary, absolute AI. In contrast, the normal stress-induced increase in cortisol production may be blunted during life-threatening illnesses (e.g., sepsis) in some patients owing to relative AI. Alternatively, there may be down-regulation of cortisol binding and adrenergic receptors despite the normal stress-induced increase in steroidogenesis, another explanation for relative AI. Etomidate transiently inhibits normal adrenal steroidogenesis (see Box 1.1) and appears to result in relative AI in critically ill patients. It is no longer used as a continuous infusion for sedation in the critically ill because of its reported deleterious impact on survival. Finally, as illustrated in the case synopsis, relative AI may underlie life-threatening hemodynamic instability. However, if it is recognized as such and treated with stress doses of glucocorticoids, this process may be reversed.

Recognition

The presentation of acute AI varies from a gradual onset over many days in a patient who is not stressed to a sudden fall in blood pressure associated with major stress such as an operation, trauma, or infection. Hypotension associated with AI can be severe and refractory to treatment. Chronic AI can be insidious and nonspecific in onset and remain undiagnosed for months. The prevalence of signs and symptoms associated with AI is detailed in Table 1.1. The most specific sign of primary AI is hyperpigmentation of the skin and mucosal surfaces caused by the high levels of corticotropin resulting from decreased cortisol feedback.

BOX 1.1 Causes of Adrenal Insufficiency

Primary Adrenal Insufficiency

- Autoimmune
  - Polyglandular autoimmune syndrome types I and II
- Infectious
  - Tuberculosis
  - Histoplasmosis
  - Blastomycosis
  - Coccidiomycosis
  - Cryptococcosis
  - Human immunodeficiency virus
  - Cytomegalovirus
  - *Mycobacterium avium-intracellulare*
  - Cryptococcus
  - Toxoplasmosis
  - Kaposi sarcoma
- Fibrosis
- Infarction
- Adrenal hemorrhage
  - Waterhouse-Friderichsen syndrome
  - Lupus anticoagulant
  - Antiphospholipid antibodies
  - Immune thrombocytopenic purpura
  - Heparin induced
  - Thrombocytopenia
  - Anticoagulants
- Metastatic disease
  - Lung
  - Gastric
  - Breast
  - Malignant melanoma
  - Lymphoma
- Drugs
  - Decreased steroid synthesis
    - Metyrapone
    - Aminoglutethimide
    - Mitotane
    - Etomidate*
    - Ketoconazole
  - Increased steroid catabolism
    - Rifampin
    - Dilantin
    - Phenobarbital
- Familial
  - Familial glucocorticoid deficiency
  - Adrenoleukodystrophy
  - Adrenomyeloneuropathy
- Iatrogenic
  - Bilateral surgical removal
  - Bilateral embolization

Secondary Adrenal Insufficiency

- Exogenous steroid administration (often referred to as tertiary or iatrogenic)
- Pituitary or hypothalamic diseases
  - Infiltrative tumor (adenoma)
  - Sarcoid
  - Hemorrhage
  - Autoimmune
- Isolated ACTH deficiency
- Surgical
  - Pituitary surgery
  - Removal of a functioning adrenal adenoma

*Still unproven and therefore speculative.
ACTH, Adrenocorticotropic hormone.

Because primary AI (Addison disease) develops from failure of the adrenal gland itself, there is evidence of both glucocorticoid and mineralocorticoid deficiencies. Because secondary AI develops from an interruption of the HPA axis that stimulates the adrenal glands to secrete cortisol, but spares the gland itself, it presents as pure glucocorticoid deficiency. In this case the patient may also have hyponatremia; this is not related to sodium excretion but rather to water intoxication secondary to an elevated level of antidiuretic hormone, as well as a primary defect in free water excretion related to glucocorticoid deficiency.

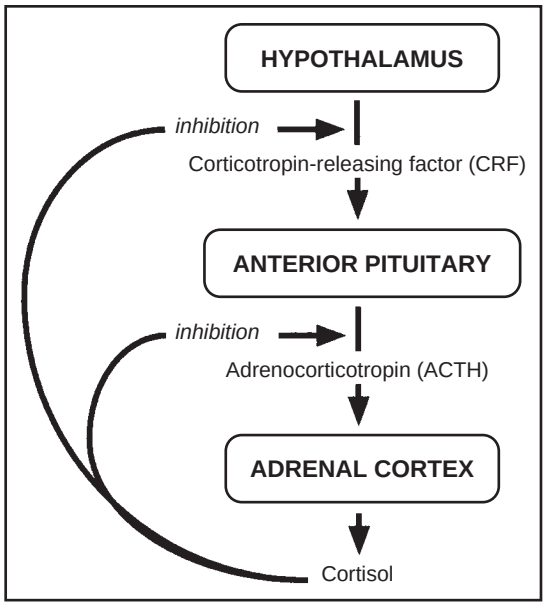

Fig. 1.1 Hypothalamic-pituitary-adrenocortical axis.

TABLE 1.1 Prevalence of Signs and Symptoms of Chronic Adrenal Insufficiency

Signs and Symptoms	Prevalence (%)
Weakness and fatigue	74–100
Weight loss	56–100
Hyperpigmentation	92–96
Hypertension	59–88
Hyponatremia	88–96
Hyperkalemia	52–64
Gastrointestinal symptoms	56
Postural dizziness	12
Adrenal calcification	9–33
Hypercalcemia	6–41
Muscle and joint pain	6
Vitiligo	4

Data from De Rosa G, Corsello SM, Cecchin L, et al: Clinical study of Addison's disease. *Exp Clin Endocrinol* 90:232-242, 1987.

Hypotension can be a common finding in both chronic and acute AI. Hypotension associated with acute AI has been reported as high-output circulatory failure with hallmarks of elevated cardiac output and index, low or normal pulmonary artery occlusion pressure, and decreased systemic vascular resistance. The pathogenesis of such hypotension is unknown but may include a combination of three possible mechanisms: (1) impairment of the direct effect of glucocorticoids on vascular smooth muscle, (2) loss of the "permissive" glucocorticoid effect on catecholamine synthesis and action, and (3) a decrease in the effects of glucocorticoids on vasoactive peptides. Dehydration can also be a factor in the hypotension associated with acute and chronic AI.

Risk Assessment