

<<水、电解质和酸碱平衡生理学>>

图书基本信息

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### 内容概要

《水、电解质和酸碱平衡生理学(英文影印版)》由加拿大多伦多大学医学院的Mitchell L.Halperin教授和Marc B.Goldstein教授编写，是国际医学图书市场上畅销的参考书，目前已修订至第3版。全书以专题的形式，围绕水、电解质及酸碱平衡有关问题，将生理学和病理生理学结合起来，逐一进行阐述，重点明确，并附有问答题及讨论答案。全书列出了许多程序图、模式图，方便读者快速、准确的参考、查阅所需要的内容。

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## 章节摘录

版权页：插图：1. Arrest methanol metabolism with ethanol: Establish therapeutic ethanol levels ( 100 mg/dL or 22 mmol/L ) by giving 0.6 g ethanol/kg intravenously or orally or give 4 oz whiskey orally. Maintain a therapeutic level of ethanol in the blood for the duration of the intoxication. Give chronic drinkers ethanol at a rate of 0.15 g/kg/h intravenously or 2 oz whiskey per hour orally. Give nondrinkers 0.07 g/kg/h intravenously or 1 oz whiskey per hour orally. Monitor the ethanol levels during therapy. 2. Correct the acidemia: At high blood methanol levels, acidemia may be severe, although the rate of production of formic acid is not usually that rapid in most cases. Nevertheless, if acidemia is severe, therapy with  $\text{NaHCO}_3$  might be indicated. 3. Remove methanol: If levels of methanol exceed 50 mg/dL ( 15 mmol/L ) , dialysis should be instituted. Hemodialysis is most efficient, but, if not available, peritoneal dialysis removes some methanol. Ethanol, which is also removed by dialysis, must be replaced. Adding ethanol to the dialysate is the easiest way to ensure that adequate blood levels are maintained.

Ethylene Glycol ( Antifreeze ) Intoxication Suspect ethylene glycol intoxication if the patient, is intoxicated, has metabolic acidosis with an increased plasma anion gap and osmolal gap, oxalate crystals in the urine, and acute tubular necrosis. Ethylene glycol, which is readily available, relatively inexpensive, and pleasant-tasting, might be ingested as an intoxicant. It causes fulminant metabolic acidosis, severe CNS toxicity, and acute tubular necrosis. As with methanol, toxicity results from the products of ethylene glycol metabolism ( see Table 3.14 ) . Following the initial toxicity associated with profound metabolic acidosis and CNS manifestations ( confusion, coma, seizures ) , patients may develop congestive heart failure during therapy because of the large load of  $\text{NaHCO}_3$  given coupled with acute renal failure. Those who survive usually have acute tubular necrosis that is generally of the oliguric form. Ethylene glycol intoxication should always be suspected in patients with metabolic acidosis and an increased plasma anion gap, especially if the patient appears intoxicated and denies intake of ethanol or if the odor of ethanol is not evident. The index of suspicion should be increased greatly by the finding of oxalate crystals in the urine. As with methanol, finding an increased plasma osmolal gap is helpful in the absence of ethanol ( see the discussion of Question 3.23 ) . The diagnosis is confirmed by detecting ethylene glycol in the blood.

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