CURRICULUM

Novel Emergency Medicine Curriculum Utilizing Self-Directed Learning and the Flipped Classroom Method: Endocrine and Metabolic Emergencies Small Group Module

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ABSTRACT:

Audience: This curriculum created and implemented at The Ohio State University emergency medicine residency program was designed to educate our emergency medicine (EM) residents, PGY-1 to PGY-3, as well as medical students and attending physicians.

Introduction: Endocrine and metabolic complaints are commonly seen in the emergency department (ED). In 2014, endocrine and metabolic complaints represented about 2 million ED visits in the United States, representing about 1.5% of all ED visits.[1] The high prevalence of endocrine disease in the US means that endocrine conditions are likely to complicate other ED visits in which endocrine disease was not the primary discharge diagnosis. For example, diabetes was documented as a chronic factor in a further 13.9 million visits. As such, emergency medicine (EM) residents must be proficient in the differential diagnosis and management of the wide variety of endocrine emergencies.

The flipped classroom curricular model emphasizes self-directed learning activities completed by learners, followed by small group discussions pertaining to the topic reviewed. The active learning fostered by this curriculum increases faculty and learner engagement and interaction time typically absent in traditional lecture-based formats.[2-4] Studies have revealed that the application of knowledge through case studies, personal interaction with content experts, and integrated questions are effective learning strategies for emergency medicine residents.[4-6] The Ohio State University EM Residency didactic curriculum recently transitioned to a "flipped classroom" approach.[7-10] We created this innovative curriculum aimed to improve our residency education program and to share educational resources with other EM residency programs. Our

curriculum utilizes an 18-month curricular cycle to cover the defined emergency medicine content. The flipped classroom curriculum maximizes didactic time and resident engagement, fosters intellectual curiosity and active learning, and meets the needs of today's learners. [4,7]

Objectives: We aim to teach the presentation and management of endocrine emergencies through the creation of a flipped classroom design. This unique, innovative curriculum utilizes resources chosen by education faculty and resident learners, study questions, real-life experiences, and small group discussions in place of traditional lectures. In doing so, a goal of the curriculum is to encourage self-directed learning, improve understanding and knowledge retention, and improve the educational experience of our residents.

Methods: The educational strategies used in this curriculum include: small group modules authored by education faculty and content experts based on the core emergency medicine content. This program also includes resident-submitted questions that were developed during review of the content. The question and answer format of the Socratic Method (with a focus on fostering an open learning environment, not negative "pimping"-type questioning) was used during small group sessions to encourage active participation and discussion. Small groups also focus on the synthesis and application of knowledge through the discussion of real-life experiences. The use of free open access medical education (FOAM) resources for pre-learning allows learners to work at their own pace and maximize autonomy.

Topics: Emergency medicine, flipped classroom, medical education, endocrine and metabolic emergencies, pedagogy, teaching.

USER GUIDE

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Learner Audience:
Medical Students, Interns, Junior Residents, Senior Residents, Attending Physicians and Faculty Members

Length of Curriculum:
The entire didactic curriculum was developed to utilize an 18-month curricular cycle; therefore, resident learners experience each curricular topic twice in the course of a three-year residency training. The endocrine emergencies module consists of five 45-60 minute small group sessions.

Topics:
Emergency medicine, flipped classroom, medical education, endocrine and metabolic emergencies, pedagogy, teaching.

Objectives:
Each chapter within our curriculum has individual objectives; however, educational objectives for the curriculum and more specifically, the endocrine emergencies module include:

1. After completing the endocrine emergencies module, resident learners will exhibit mastery within this content area and will critically discuss the pathophysiology, diagnosis, and treatment of various pediatric and adult endocrine emergencies including:
2. Thyroid Disorders
    a. Discuss the classical presentation of hyperthyroidism.
    b. Describe how thyroid hormone is produced.
    c. Discuss the treatment of thyroid storm and list the correct order of medications.
    d. Discuss the presentation of hypothyroidism and myxedema coma.
    e. Describe the treatment of severe hypothyroidism.
3. Electrolyte Abnormalities: Sodium, Potassium, Calcium, Magnesium and Phosphate
    a. Describe the presentation and causes of hyponatremia.
    b. List a differential for possible causes of hyponatremia.
    c. Discuss management of acute and chronic hyponatremia.
    d. Review the sodium content of various saline solutions.
    e. Discuss historical and physical exam findings consistent with untreated end-stage renal disease.
    f. Describe the management of hyperkalemia.
    g. Review ECG findings commonly found in patients with hyperkalemia.
    h. Discuss the symptoms and causes of hypercalcemia.
    i. Describe the management of hypercalcemia.
4. Parathyroid and Pituitary Disorders
    a. Describe the pathophysiology and common etiologies of panhypopituitarism.
    b. List the primary hormones secreted by the pituitary gland.
    c. Describe the common etiologies of Cushing's syndrome.
    d. Discuss the evaluation and management of Cushing's syndrome.
5. Diabetes and Diabetic Ketoacidosis
    a. Describe the laboratory findings and treatment for children presenting with new onset diabetes.
    b. Discuss the risks and benefits of bicarbonate administration for severe acidosis during diabetic ketoacidosis.
    c. Describe the risk factors and treatment of cerebral edema.
    d. Discuss the common presenting symptoms for hypoglycemia and atypical presentations.
    e. List risk factors for development of severe hypoglycemia.
    f. Discuss the diagnosis and management of hyperglycemic hyperosmolar non-ketotic state.
6. Adrenal Disorders
    a. Describe the pathophysiology, evaluation, and management of adrenal insufficiency.

b. Compare the most common testing pathways for diagnosing adrenal insufficiency.
c. Discuss etomidate and prednisone, which could affect the hypothalamic-pituitary-adrenal (HPA) axis, and the literature behind their continued use.
d. Describe treatment of hypertensive crisis in suspected pheochromocytoma.

Brief introduction:

The flipped classroom learning approach is becoming more commonly recognized as a preferred curricular model for mature learners, specifically those in medical education. This particular model is a natural fit for the hands-on, experiential emergency medicine learner.[5] The active learning fostered by this curriculum increases faculty and learner engagement and interaction time, which is typically absent in traditional lecture-based formats.[6] Education literature shows that resident learners prefer learning activities that involve small group discussion, are case/skill based, and emphasize the application of newly obtained knowledge.[2,11] This educational model also provides a clear channel for the incorporation of evidence-based medicine and increases opportunities for educator-learner conversations. A successful flipped classroom curriculum fosters learner accountability and provides robust opportunities for formal assessment of various emergency medicine milestones.[5,9] For these reasons, we developed a flipped classroom curriculum at The Ohio State University emergency medicine residency program. This endocrine emergencies curriculum is one of several topics in our overall didactic curriculum.

Endocrine and metabolic complaints are commonly seen in the emergency department (ED). In 2014, endocrine and metabolic complaints represented about 2 million ED visits in the United States, representing about 1.5% of all ED visits.[1] The high prevalence of endocrine disease in the US means that endocrine conditions are likely to complicate other ED visits in which endocrine disease was not the primary discharge diagnosis. For example, diabetes was documented as a chronic factor in a further 13.9 million visits. As such, emergency medicine (EM) residents must be proficient in the differential diagnosis and management of the wide variety of endocrine emergencies.

Problem identification, general and targeted needs assessment:

Traditional lecture-based didactics may not be the most effective or preferred method for emergency medicine resident education.[7] Previously, we used a traditional lecture format in our residency curriculum despite overwhelming evidence favoring a more hands-on, "flipped classroom" approach.[9,10] From the perspective of resident learners, the chance to remain fully engaged through asking questions developed from personal experiences, in addition to learning from the experiences of others, provides a technique of learning that makes a topic more difficult to forget.[6]

As current literature reveals, both educators and learners benefit from an interactive and collaborative classroom, leading to the creation and implementation of this proposed curricular model at our emergency medicine residency program.[12] This weekly small group curriculum has now replaced three hours of traditional lecture-based didactics. Learners divide into small groups of about 20 participants. Each group is led by both a faculty leader and a designated senior resident who has spent extra time preparing; the senior resident is expected to guide the discussion with a question and answer format while the faculty member is there to add expertise and guidance. Since implementation, residents and educators are engaging in new, valuable flipped classroom learning communities at The Ohio State University. Through the curriculum, we continually seek to foster self-directed learning and increased collaboration between resident learners and education faculty members. This ensures that resident time will be maximized and learning will be more efficient and effective, providing a potential positive impact on patient care and physician wellness. Currently, minimal flipped classroom curricular materials dedicated to the core content of emergency medicine exist. The specific content was based on the EM Model of Clinical Practice to ensure topics essential to emergency physicians would be covered.

Goals of the curriculum:

We aim to teach the presentation and management of endocrine emergencies through the creation of a flipped classroom design. This unique, innovative curriculum utilizes resources chosen by education faculty and resident learners, study questions, real-life experiences, and small group discussions in place of traditional lectures. In doing so, a goal of the curriculum is to encourage self-directed learning, improve understanding and knowledge retention, and improve the educational experience of our residents.

Objectives of the curriculum:

Each chapter within our curriculum has individual objectives; however, educational objectives for the curriculum and more specifically, the endocrine emergencies module include:

After completing the endocrine emergencies module, resident learners will exhibit mastery within this content area and will critically discuss the pathophysiology, diagnosis, and treatment of various pediatric and adult endocrine emergencies including:

1. Thyroid Disorders

   a. Discuss the classical presentation of hyperthyroidism.
   b. Describe how thyroid hormone is produced.
   c. Discuss the treatment of thyroid storm and list the correct order of medications.
   d. Discuss the presentation of hypothyroidism and myxedema coma.
   e. Describe the treatment of severe hypothyroidism.
2. Electrolyte Abnormalities: Sodium, Potassium, Calcium, Magnesium and Phosphate
   a. Describe the presentation and causes of hyponatremia.
   b. List a differential for possible causes of hyponatremia.
   c. Discuss management of acute and chronic hyponatremia.
   d. Review the sodium content of various saline solutions.
   e. Discuss historical and physical exam findings consistent with untreated end-stage renal disease.
   f. Describe the management of hyperkalemia.
   g. Review ECG findings commonly found in patients with hyperkalemia.
   h. Discuss the symptoms and causes of hypercalcemia.
   i. Describe the management of hypercalcemia.
3. Parathyroid and Pituitary Disorders
   a. Describe the pathophysiology and common etiologies of panhypopituitarism.
   b. List the primary hormones secreted by the pituitary gland.
   c. Describe the common etiologies of Cushing's syndrome.
   d. Discuss the evaluation and management of Cushing's syndrome.
4. Diabetes and Diabetic Ketoacidosis
   a. Describe the laboratory findings and treatment for children presenting with new onset diabetes.
   b. Discuss the risks and benefits of bicarbonate administration for severe acidosis during diabetic ketoacidosis.
   c. Describe the risk factors and treatment of cerebral edema.
   d. Discuss the common presenting symptoms for hypoglycemia and atypical presentations.
   e. List risk factors for development of severe hypoglycemia.
   f. Discuss the diagnosis and management of hyperglycemic hyperosmolar non-ketotic state.
5. Adrenal Disorders
   a. Describe the pathophysiology, evaluation, and management of adrenal insufficiency.
   b. Compare the most common testing pathways for diagnosing adrenal insufficiency.
   c. Discuss etomidate and prednisone, which could affect the hypothalamic-pituitary-adrenal (HPA) axis, and the literature behind their continued use.
   d. Describe treatment of hypertensive crisis in suspected pheochromocytoma.

Educational Strategies: (See curriculum chart)
Please refer to the curriculum chart of linked objectives and educational strategies.

Evaluation and Feedback:
This innovative curriculum was literature based and specifically designed to maximize active learning using the flipped classroom learning model. We overcame initial challenges and skepticism from both educators and learners to execute a successful, novel curricular model. Both resident learners and faculty educators have provided an overwhelming amount of positive feedback. Additionally, a survey was administered to each resident prior to initiation of the curricular innovation and repeated at the conclusion of the first 18-month cycle. Learners and educators were enthusiastic about the conference structure and expressed a preference for it rather than the previous, lecture-based didactics. Resident learner attendance at weekly emergency medicine didactics increased, presumably as a result of our curricular innovation and the associated increase in faculty engagement, active discussions, and learner perceived value of the sessions. More recently during the second 18-month cycle of the flipped classroom curriculum, students were surveyed on their perceived quality of instruction of the various program components. A majority of residents (60.9%) reported that the small group discussions were good or excellent, compared to only 26% of residents that felt that our grand rounds sessions during the same time were good or excellent. This curriculum has been delivered to two cohorts of learners, having delivered the content twice in three years with about 50 residents per cycle. On the most recent iteration, residents evaluated the teaching methods as effective, with an average rating of more than 4.3 out of 5 (4 being agree, 5 being strongly agree). The curriculum is critically evaluated and updated by education faculty members in order to ensure educational material remains current and consistent with the emergency medicine core content.

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Additional Resources:

Educational resources are available within each individual chapter of this Endocrine emergencies curricular module; however, a complete list of resources and educational materials are listed below.

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DIDACTICS AND HANDS-ON CURRICULUM

Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Thyroid Disorders	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 15 minutes for brief topic review and 30-45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of thyroid disorders.	By the end of this session, learners will: 1. Discuss the classical presentation of hyperthyroidism. 2. Describe how thyroid hormone is produced. 3. Discuss the treatment of thyroid storm and list the correct order of medications. 4. Discuss the presentation of hypothyroidism and myxedema coma. 5. Describe the treatment of severe hypothyroidism.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: *(Optional)* Projector or large screen so that instructor can pull up web images during session. Tables and space promoting small group discussion. Instructors: 2 faculty members or content experts. Predetermined senior resident discussion leader. Timing: Small group discussions with no more than 15 learners. Duration: 45-60 Minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric. Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.

DIDACTICS AND HANDS-ON CURRICULUM

Electrolyte Abnormalities	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 15 minutes for brief topic review and 30-45 minutes for case and content discussion.	Pathophysiology, diagnosis and therapies of electrolyte abnormalities.	By the end of this session, learners will: 1. Describe the presentation and causes of hyponatremia. 2. List a differential for possible causes of hyponatremia. 3. Discuss management of acute and chronic hyponatremia. 4. Review the sodium content of various saline solutions. 5. Discuss historical and physical exam findings consistent with untreated end-stage renal disease. 6. Describe the management of hyperkalemia. 7. Review ECG findings commonly found in patients with hyperkalemia. 8. Discuss the symptoms and causes of hypercalcemia 9. Describe the management of hypercalcemia.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: *(Optional)* Projector or large screen so that instructor can pull up web images during session. Tables and space promoting small group discussion. Instructors: 2 faculty members or content experts. Predetermined senior resident discussion leader. Timing: Small group discussions with no more than 15 learners. Duration: 45-60 Minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric. Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.

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Pituitary Disorders	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 15 minutes for brief topic review and 30-45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of parathyroid and pituitary disorders.	By the end of this session, learners will: 1. Describe the pathophysiology and common etiologies of panhypopituitarism. 2. List the primary hormones secreted by the pituitary gland. 3. Describe the common etiologies of Cushing's syndrome. 4. Discuss the evaluation, and management of Cushing's syndrome.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: *(Optional)* Projector or large screen so that instructor can pull up web images during session. Tables and space promoting small group discussion. Instructors: 2 faculty members or content experts. Predetermined senior resident discussion leader. Timing: Small group discussions with no more than 15 learners. Duration: 45-60 Minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric. Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.

DIDACTICS AND HANDS-ON CURRICULUM

Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Diabetes and Diabetic Ketoacidosis	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 15 minutes for brief topic review and 30-45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of diabetes and DKA.	By the end of this session, learners will: 1. Describe the laboratory findings and treatment for children presenting with new onset diabetes. 2. Discuss the risks and benefits of bicarbonate administration for severe acidosis during diabetic ketoacidosis. 3. Describe the risk factors and treatment of cerebral edema. 4. Discuss the common presenting symptoms for hypoglycemia and atypical presentations. 5. List risk factors for development of severe hypoglycemia. 6. Discuss the diagnosis and management of hyperglycemic hyperosmolar non-ketotic state.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: *(Optional)* Projector or large screen so that instructor can pull up web images during session. Tables and space promoting small group discussion. Instructors: 2 faculty members or content experts. Predetermined senior resident discussion leader Timing: Small group discussions with no more than 15 learners. Duration: 45-60 Minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric. Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.

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Adrenal Disorders	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 15 minutes for brief topic review and 30-45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of adrenal disorders.	By the end of this session, learners will: 1. Describe the pathophysiology, evaluation, and management of adrenal insufficiency. 2. Compare the most common testing pathways for diagnosing adrenal insufficiency. 3. Discuss etomidate and prednisone, which could affect the hypothalamic-pituitary-adrenal (HPA) axis, and the literature behind their continued use. 4. Describe treatment of hypertensive crisis in suspected pheochromocytoma.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: *(Optional)* Projector or large screen so that instructor can pull up web images during session. Tables and space promoting small group discussion. Instructors: 2 faculty members or content experts. Predetermined senior resident discussion leader. Timing: Small group discussions with no more than 15 learners. Duration: 45-60 Minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric. Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.

Appendix A:
Thyroid Disorders

Objectives

1. Discuss the classical presentation of hyperthyroidism.
2. Describe how thyroid hormone is produced.
3. Discuss the treatment of thyroid storm and list the correct order of medications.
4. Discuss the presentation of hypothyroidism and myxedema coma.
5. Describe the treatment of severe hypothyroidism.

Case Studies

Case 1: A 54-year-old woman presents with the sudden onset of palpitations and mild shortness of breath. She denies any chest pain or lower extremity swelling. She has no history of alcohol use. Review of systems is positive for an unintentional 20-lb weight loss over last 2 months, despite an increased appetite. Vital signs are as follows: pulse 180, blood pressure 150/86, respiratory rate 26, temperature 101.3^{o}F. On exam, she is diaphoretic and appears anxious. Heart and lung exams reveal tachycardia with no murmurs and clear lung sounds.

Question Prompts:

1. What is the most likely condition causing this patient's presentation? Describe the classic presentation and causes of this condition
    a. This patient's presentation is consistent with hyperthyroidism.
        i. Presentation can include:
            - Constitutional: Weight loss despite hyperphagia, fatigue, generalized weakness
            - Hypermetabolic: Heat intolerance, cold preference, excessive perspiration
            - Cardiorespiratory: Palpitations, tachycardia, atrial fibrillation, dyspnea, dyspnea on exertion, chest pains, poor exercise tolerance
            - Gastrointestinal: Nausea, vomiting, diarrhea, dysphagia
            - Neuropsychiatric: Anxiety, restlessness, hyperkinesis, emotional lability, confusion, insomnia, poor attention
            - Neuromuscular: Myopathy, myalgias, tremor, proximal muscle weakness (difficulty getting out of a chair or combing hair)
            - Ophthalmologic: Tearing, irritation, wind sensitivity, diplopia, foreign body sensation
            - Thyroid gland: Neck fullness, dysphagia, dysphonia
            - Dermatologic: Flushed feeling, hair loss, pretibial swelling

- Reproductive: Oligomenorrhea, amenorrhea, menometrorhaggia, decreased libido, gynecomastia, erectile dysfunction, infertility

b. How can the presentation of hyperthyroidism change in elderly patients?
   i. Remember that hyperthyroidism can manifest is more subtle ways in elderly patients. They are often asymptomatic or with nonspecific symptoms including weight loss, shortness of breath, and cognitive dysfunction. Older adults are more prone to cardiac manifestations, and often present with atrial fibrillation. Older adults who smoke or have higher circulating thyroid hormone levels appear to have more severe symptoms.

c. Causes:
   i. Graves' disease is the most common cause of hyperthyroidism. It is caused by autoantibodies that bind to the receptors for thyroid stimulator hormone (TSH) and cause its chronic release.
   ii. Toxic multinodular goiter is the second leading cause (usually in women older than 50). Usually present with more mild symptoms compared to Grave's.
   iii. Toxic adenoma is a hyperactive thyroid nodule.
   iv. Thyroiditis, which may be autoimmune, drug-induced, infectious, or traumatic.
      - Hashimoto's thyroiditis, or chronic lymphocytic thyroiditis, is the most common cause of autoimmune thyroiditis in the US.
      - Silent thyroiditis is a painless, or silent, thyroiditis typified by a small non-tender goiter and mild symptoms. It is more common in women than men, with a peak from age 30 to 40 years. It has an autoimmune cause. More commonly seen in areas of adequate iodine intake.
      - Postpartum thyroiditis affects 5-10% of all pregnant women and can last 2-3 months.
      - Subacute thyroiditis is viral infection mediated. It is more common in women than men, with a peak age from 40-50.
      - Suppurative thyroiditis presents with fever & anterior neck pain. It is caused by bacterial infection, most commonly in immunocompromised patients.
      - Drug-induced thyroiditis, usually related to amiodarone or lithium
      - Factitious thyroiditis caused by exogenous administration of thyroid hormone (which may be found in some weight loss preparations).

2. Briefly describe how thyroid hormone is produced in the body? What testing needs to be ordered to work up the cause of the thyroid disorder?
   a. Thyroid releasing hormone (TRH) produced in the hypothalamus stimulates the pituitary to generate thyroid stimulating hormone (TSH), which subsequently stimulates the thyroid gland to generate T3 and T4. T4 is at high concentrations in blood (14:1), but T3 is the active form. T4 is converted to T3 intracellularly.
   b. When thyroid dysfunction is on the differential, laboratory workup should include serum levels of TSH, T3, and free T4, as well as serum electrolytes.
3. What differentiates patients with thyroid storm from thyrotoxicosis?

a. Signs and symptoms of thyroid storm are constitutional (fever), central nervous system related (agitation, confusion, delirium, stupor, coma, and seizure), and cardiovascular (tachycardia, arrhythmia, and heart failure). CNS symptoms differentiate thyroid storm from thyrotoxicosis.

4. What is the proper order of treatment for this patient? What are the mechanisms of action of the treatments?
    a. The recommended order of treatment for the hyperthyroidism is as follows:
        i. **#1 Beta blockade:** Mitigates hypermetabolic state
            - Propranolol is the most commonly used agent as it also inhibits conversion of T4 to T3
            - Proper dosing of propranolol is essential as it has dose dependent effects on the peripheral conversion of thyroid hormone. Esmolol can be considered as an alternative.
            - Oral dosing of labetalol is 60 – 80 mg q4h
            - Alternatively, labetalol can be given intravenously (IV) via a 2-step process. This may potentially avoid beta-blocker associated hypotension in those with associated congestive heart failure secondary to thyroid storm.
                a. Test dose: 0.5 mg to 1 mg as a slow IV push administered over 10 minutes
                b. Subsequent doses: 1 to 3 mg IV over 10 to 15 minutes every few hours to desired effect with monitoring of cardiac rhythm
        ii. **#2 Thioamides:** Reduce thyroid hormone production by inhibiting thyroid peroxidase, an enzyme involved in the production of T3 and T4. There are two agents, propylthiouracil (PTU) and methimazole (MMI)
            - **Propylthiouracil**
                a. Loading dose: 600 to 1000 mg orally (PO), maintenance: 200 to 250 mg PO q4h
                b. Preferred thioamide in early pregnancy
                c. Black box warning for severe hepatotoxicity
            - **Methimazole**
                a. Loading dose: 20 to 25 mg PO, maintenance dose: 20 to 25 mg PO q4h
            - Note that PTU and MMI can both be administered through the rectal route as a suppository and retention enema. Intravenous (IV) thioamides are currently not commercially available in the United States
            - Note also, that agranulocytosis can occur with both MMI and PTU
        iii. **#3 Inorganic Iodine:** Inhibit release of pre-formed thyroid hormone from the thyroid gland. It is very important to remember that administration of inorganic iodine should be delayed for at least one hour after initiation of thioamide therapy because the iodine load can serve as a substrate for thyroid hormone synthesis and exacerbate thyroid storm.
            - Several forms are available:

a. Saturated solution of potassium iodide (SSKI): 5 drops PO, via nasogastric tube (NG), or per rectum (PR) q6h
b. Lugol's solution: 8 drops PO, NG, or PR q6h
c. Sodium iodide: 500 mg IV q12h
d. Remember that dosages of the above formulations are by the dropper-full and they are not interchangeable as the amount of iodine per drop varies widely. The dose should be dissolved in 3 to 4 ounces of milk or fruit juice to mask the bitter taste and ensure that the full dose is ingested.

iv. **Other Adjuncts:**

- **Corticosteroids**: Used to treat the depression of the hypothalamic-pituitary axis that commonly occurs in the setting of thyroid storm. They also help inhibit conversion of T4 to T3.
  a. Improved survival is seen with early steroid administration
  b. Hydrocortisone: loading dose of 300 mg IV followed by maintenance dose of 100 mg IV q8h
  c. Steroids can be given immediately, without the need to wait for iodine treatment. The most important order is that thioamides must come before iodine. Beta blockers should be used ASAP to blunt symptoms of thyrotoxicosis.
- **Cholestyramine resin:** Can be used to interrupt enterohepatic recirculation of thyroid hormone, leading to decreased circulating levels of thyroid hormone
  a. Dose: 4 g PO q6h

Case 2: A 67-year-old man presents from an extended care facility with increased lethargy and inability to assist with his activities of daily living. He has been undergoing an aggressive bowel regimen for constipation over the last week, which has largely been unsuccessful. A chart review shows a history of congestive heart failure and longstanding hypertension. His current medications include hydrochlorothiazide and amlodipine. He is unable to participate in the history. Vitals are as follows: Pulse 54, blood pressure 100/85, respiratory rate 12, and temperature of 95.6°F. He is sleepy but arousable. He appears as in the photo below. Laboratory analysis shows: Na 125, Cl 98, K 3.8, BUN 60, Creatinine 1.77, glucose 78. His TSH is elevated and free T4 is low.

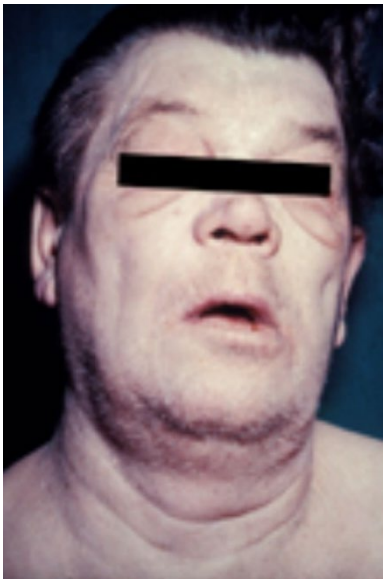

Image source: Fred HL, van Dijk HA. Case 82. In: *Images of Memorable Cases: 50 Years at the Bedside*. Openstax CNX. https://cnx.org/contents/WCJhOoMc@3/Images-of-Memorable-Cases-Case. Updated December 8, 2008. CC BY 2.0.

Question Prompts:

1. What is the most likely diagnosis of this patient? Describe the physical findings in the picture that lead you to suspect this condition.
    a. This patient has physical exam findings and labs consistent with hypothyroidism. Given his altered mental status, hypothermia and hypotension, this patient meets criteria for myxedema coma. Myxedema coma is decompensated hypothyroidism characterized by altered mental status and hypothermia. Myxedema coma is usually precipitated by a physiologic stress in a patient with known underlying hypothyroidism. Non-pitting edema ('myxedema') is a common finding, though not necessary to make the diagnosis. This patient demonstrates thinning of the lateral 1/3 of the brow, periorbital edema, broadened nose, flat facial expression, swelling of the lips.
2. What other physical exam findings might be expected in hypothyroidism?
    a. Macroglossia
    b. Body and scalp hair are brittle and coarse, some degree of alopecia
    c. Pallor
    d. Decreased bowel sounds
    e. Puffiness of the hands
    f. Ascites
3. Discuss the common precipitants for myxedema coma?
    a. Infection, sepsis (especially pneumonia)
    b. Exposure to cold
    c. Cerebrovascular accident
    d. Drug effect
        i. Altered sensorium: Sedative-hypnotics, narcotics, anesthesia, neuroleptics

ii. Decreased T_4 and T_3 release: Amiodarone, lithium, iodides
iii. Enhanced elimination of T_4 and T_3: Phenytoin, rifampin
iv. Inadequate thyroid hormone replacement: Noncompliance; interference with absorption (iron, calcium, cholestyramine)

e. Myocardial infarction
f. Gastrointestinal bleeding
g. Trauma or burns
h. Congestive heart failure (CHF)
i. Hypoxia
j. Hypercapnia
k. Electrolyte abnormalities (hyponatremia, hypoglycemia, hypercalcemia)
l. Diabetic ketoacidosis

4. What is the initial treatment of myxedema coma?
   a. Initial evaluation should focus on basic airway, breathing and circulation (ABCs). Ensure patients protect their airway and provide ventilator support as needed. Initiate fluid resuscitation for hypotension with 0.9% NS (or D5/0.9% NS if hypoglycemic). Avoid hypotonic fluids. Watch for unmasking of CHF with volume expansion. Passively rewarm with regular blankets and take measures to prevent further heat loss. Avoid mechanical stimulation.
   b. Hydrocortisone: 100 mg IV every 8 hrs. Patients suffering from severe hypothyroidism often have relative adrenal suppression, owing to high TSH levels (suppresses corticotropin-releasing hormone). Corticosteroids should be given prior to thyroid hormone replacement as replacing thyroid in the setting of adrenal insufficiency could theoretically cause clinical deterioration.
   c. Thyroid hormone replacement: Administer T4 200–400 µg IV loading dose. Use lower doses to patients who are smaller, have coronary artery disease, or a history of arrhythmia. Subsequent daily replacement of 1.6 µg/kg body weight PO (give 75% of this dose if given IV). Other treatment protocols call for using combination T4 and T3 replacement. Because of potential complications of using these medications in these already critically ill patients, it is recommended to determine the best course of action in close consultation with an endocrinologist.
   d. Correction of hyponatremia: Consider fluid restriction. If sodium is less than 120 mEq/L, consider 3% sodium chloride in 50 to 100 ml boluses.
   e. Consider and treat any precipitating illnesses with special attention to infectious causes.

Suggested Reading:

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Thiessen MEW. Thyroid and Adrenal Disorders. In: Walls RM, Hockberger RS, Gausche-Hill M, eds. *Rosen's emergency medicine: Concepts and clinical practice.* 9th ed. Philadelphia, PA: Elsevier/Saunders; 2018:1557-1571.

Papi G, Corsello SM, Pontecorvi A. Clinical concepts on thyroid emergencies. *Front Endocrinol (Lausanne).* 2014; 5:102. doi: 10.3389/fendo.2014.00102

Angel TE, Lechner MG, Nguyen CT, Salvato VL, Nicoloff JT, LoPresti JS. Clinical features and hospital outcomes in thyroid storm: a retrospective cohort study. *J Clin Endocrinol Metab.* 2015;100(2):451-459. doi: 10.1210/jc.2014-2850

Appendix B:
Electrolyte Abnormalities

Objectives

1. Describe the presentation and causes of hyponatremia.
2. List a differential for possible causes of hyponatremia.
3. Discuss management of acute and chronic hyponatremia.
4. Review the sodium content of various saline solutions.
5. Discuss historical and physical exam findings consistent with untreated end-stage renal disease.
6. Describe the management of hyperkalemia.
7. Review ECG findings commonly found in patients with hyperkalemia.
8. Discuss the symptoms and causes of hypercalcemia.
9. Describe the management of hypercalcemia.

Case Studies

Case 1: A 45-year-old male group home resident is brought in from his residence after having a seizure. He has a history of traumatic brain injury but no previous history of seizures and no recent illnesses. For the last week, he's been experiencing more frequent behavioral issues and has been spending prolonged periods secluded in his bathroom as "time outs." Vitals are as follow: pulse 110, blood pressure 150/90, respiration rate 22, temperature 37°C. On exam he appears post-ictal, he has no current seizure activity, and no signs of trauma. Initial blood work shows: Na 108, Cl 95, K 3.0, Bicarbonate 22, and glucose 110.

Question Prompts:

1. What are the possible causes of profound hyponatremia? Which is most likely given this patient's history?
    a. Pseudohyponatremia
        i. Hyperlipidemia.
        ii. Hyperproteinemia (multiple myeloma, macroglobulinemia).
    b. Dilutional
        i. Hyperglycemia: Referred to by some as "pseudohyponatremia," but this form of hyponatremia represents a true hyponatremia secondary to osmotic dilution.
    c. Hypovolemic: decreased total body water and sodium, with a relatively greater decrease in sodium.
        i. Body fluid losses: sweating, vomiting, diarrhea, gastrointestinal suction.
        ii. Third spacing: bowel obstruction, burns, pancreatitis, rhabdomyolysis.
        iii. Renal causes: diuretics, mineralocorticoid deficiency, osmotic diuresis, renal tubular acidosis, salt-wasting nephropathies.

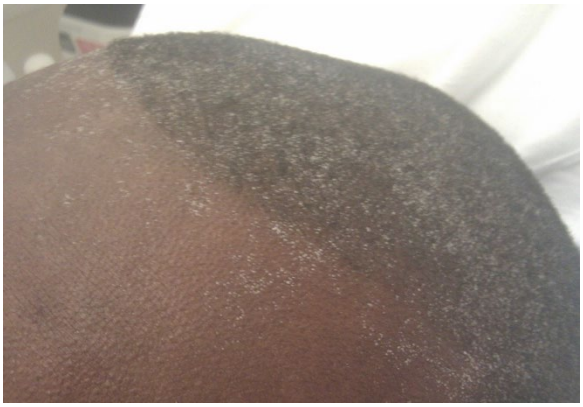
d. Hypervolemic hyponatremia: Increased total body sodium with a relatively greater increase in total body water.
    i. Heart failure.
    ii. Chronic renal failure.
    iii. Hepatic failure or cirrhosis.
e. Euvolemic Hyponatremia: Increased total body water with nearly normal total body sodium.
    i. Syndrome of inappropriate anti-diuretic hormone secretion (SIADH).
        1. Drugs that can cause SIADH: Diuretics, barbiturates, carbamazepine, chlorpropamide, clofibrate, opioids, tolbutamide, vincristine.
    ii. Psychogenic polydipsia: Excessive water intake in patients with underlying psychiatric disorder (most commonly schizophrenia) or developmental disorders.
    iii. Beer potomania: Massive consumption of beer, which is low in solutes and electrolytes.
    iv. Hypothyroidism: Low thyroid stimulating hormone (TSH) associated with hyponatremia, especially extreme such as in myxedema coma.
    v. Adrenal insufficiency: Lack of steroids leads to salt wasting.
    vi. MDMA (ecstasy): Etiology controversial, but possibly drug-induced secretion of anti-diuretic hormone (ADH) in the setting of over hydration with water.
    vii. Accidental or intentional water intoxication: Exceedingly rare but may be seen in "water drinking competitions" or hazing rituals.
f. Given this patient's past medical history and recent behavioral issues, it is likely he is suffering from hyponatremia due to psychogenic polydipsia.

2. How fast can the hyponatremia be corrected and what is the risk of rapid correction?
    a. Treatment of hyponatremia is guided by the patient's clinical presentation, severity of symptoms, estimated duration of illness, fluid status, and underlying cause of the sodium disturbance. Typically, sodium should be corrected during a time course of 48 to 72 hours.
    b. The neurological changes, including flaccid paralysis, dysarthria, dysphagia, and hypotension, associated with *overly rapid sodium correction* are referred to as *osmotic demyelinating syndrome (ODS),* previously termed *central pontine myelinolysis.* Correcting at rates higher than 0.5mEq/L/hour (12mEq/L/day) put patients at increased risk for ODS. Most ODS cases occur in the alcoholic, malnourished, and elderly population, although this devastating side effect can occur in young healthy patients as well.
    c. There is no consensus regarding the optimal treatment of symptomatic hyponatremia (for example seizures, altered mental status, delirium or other symptoms that are otherwise unexplained in the setting of hyponatremia). However, there is agreement that correction should occur at a sufficient pace and magnitude to reverse the manifestations of hypotonicity but not be so rapid as to pose a risk for development of ODS.

3. How do you calculate the correction of hyponatremia?
    a. Given this patient's level of hyponatremia in association with change in mental status and history of seizing, 3% hypertonic saline is recommended.
    b. Calculations: $Change\ in\ serum\ Na = \frac{Fluid\ Na - Serum\ Na}{Total\ Body\ Water + 1}$

INFUSATE	INFUSATE SODIUM (mEq/L)	EXTRACELLULAR FLUID DISTRIBUTION (%)
23.4% hypertonic	4000	100
3% Hypertonic saline	513	100
0.9% Normal saline solution	154	100
Lactated Ringer's solution	130	97
Half-normal saline solution	77	73
0.2% Sodium chloride + D_5W	34	55
D_5W	0	45

Case 2: A 58-year-old undocumented citizen from Honduras presents with his family who states he needs hemodialysis. He has recently arrived in the United States (US) and had been on hemodialysis in his home country, but is not established in the US medical care system and unable to get in to a dialysis center. His last treatment was 8 days ago. He is sleepy, but arousable. States he has had some nausea and anorexia. Vitals are as follows: pulse 101, blood pressure 170/90, respiration rate 24, temperature 37°C, SpO2 93% on room air. His skin exam is shown below. He has jugular venous distension and pedal edema bilaterally, with crackles in both lung bases. His laboratory exam shows: Na 140, K 6.5, Cl 97, BUN 140, Creat 7.9, Glucose 145. Exam of his skin shows:



Classic skin findings of uremic frost, a skin finding seen in chronic kidney disease caused by crystallized urea deposits.
Image source: Fythrion. Uremic frost on forehead and scalp of young Afro-Caribbean male. In: Wikimedia Commons. https://commons.wikimedia.org/wiki/File:Uremic_frost_on_forehead_and_scalp_of_young_Afro-Caribbean_male.jpg. Nov 12, 2012. GNU FDL.

Question Prompts:

1. What is the diagnosis and what are the initial management and medications indicated?
    a. Calcium for cardiac membrane stabilization (see #3 for dosing).
    b. Beta-agonists via continuous albuterol to shift potassium intracellularly.
    c. Insulin 10 units intravenously (IV) to shift potassium intracellularly.
    d. Dextrose can be given in amp or continuous infusion. Dextrose also shifts potassium intracellularly and provides glucose after insulin to prevent hypoglycemia.

e. Furosemide can be considered in patients who still make urine. The dose is 20-40mg IV.
f. Dialysis is the definitive management for hyperkalemia, especially in this patient with severe renal failure.
g. General supportive care as well, such as considering Bipap for fluid overload pulmonary edema.

2. Is sodium polystyrene sulfonate indicated? What are the potential complications?
    a. Sodium polystyrene sulfonate (Kayexalate), a potassium-binding ion exchange resin, has long been administered with sorbitol to promote elimination of potassium from the body, but is no longer considered to be effective or free of adverse effects.
    b. Common side effect includes constipation. Life-threatening side effects include possible colonic necrosis. This is thought to be mainly from the sorbitol component, as formulations with less or no sorbitol have not been associated with this somewhat rare complication. Patients with decreased gut motility and post-surgical patients are at increased risk of colonic necrosis with SPS + sorbitol formulations.
    c. Efficacy: not proven effective at treating hyperkalemia.
3. What electrocardiogram (ECG) findings are found with increasing potassium?
    a. Classic electrocardiographic changes include peaked T wave, flattened P waves with prolonged PR interval or a totally absent P wave, wide QRS, and sine wave pattern (portending imminent cardiac arrest). Peaked T waves usually appear as serum potassium levels exceed 5.5 to 6.5 mEq/L; P wave disappearance and PR prolongation are common with levels above 6.5 to 7.5 mEq/L; and levels above 7.0 to 8.0 mEq/L can result in QRS prolongation. Facilitators should consider drawing out this progression for learners, or showing the ECG progression figure from the Critical Care Emergency Medicine, electrolyte disorders chapter, cited in the references.
4. What is the difference between calcium chloride and calcium gluconate and how much should be given/what route?
    a. Intravenous calcium stabilizes the cardiac membrane by restoring the electrical gradient. Calcium increases the depolarization threshold and the calcium gradient across the cardiac membrane, reducing myocyte excitability, and increasing cardiac conduction speed, narrowing the QRS. Calcium does not decrease serum potassium levels, and its effect is rapid (within 1 to 3 minutes), but transient (30 to 60 minutes or less). The dose is one ampule, or 10 mL of 10% calcium chloride solution. Calcium chloride is preferably administered through a central venous line due to the risk of tissue necrosis should it extravasate at the injection site.
    b. More than 10 mL of calcium gluconate will often be required, because it contains only one-third the elemental calcium contained in calcium chloride. Calcium gluconate is preferred in pediatric cases, as well as in patients with less emergent (more chronic) hyperkalemic patients when a slow infusion is desired.
5. Discuss special circumstances in hyperkalemia (DKA and digoxin toxicity).
    a. Diabetic ketoacidosis (DKA) may result in serum hyperkalemia, although most hyperkalemic patients with DKA are actually total body deficient in potassium. In this insulin-deficient and acidotic state, serum potassium levels rise as intracellular potassium shifts out of cells.

Simply treating the patient's underlying DKA will also treat the hyperkalemia. In fact, the mainstay of treatment of DKA—fluids and insulin—closely mirrors the treatment of isolated hyperkalemia. As DKA patients are often total body deficient in potassium, other treatment modalities for hyperkalemia such as kayexalate, furosemide, or dialysis are not indicated.

b. In the case of hyperkalemia associated with digitalis toxicity:
    i. In acute poisoning, serum potassium concentration may begin to rise rapidly within 1 to 2 hours of ingestion. Potassium should be withheld, even if mild hypokalemia is measured initially. The initial serum potassium concentration may in fact be a better predictor of mortality than the initial digoxin concentration. Before digoxin immune fab treatment was available, up to 50% of the patients with serum potassium concentrations between 5.0 and 5.5 mEq/L died. In the setting of digoxin toxicity, digoxin immune fab is indicated with potassium levels above 5.0 mEq/L
    ii. The decision to administer calcium to patients with hyperkalemia and digoxin poisoning represents a clinical dilemma. Classic teaching is that in the setting of the increased intracellular calcium concentration from digoxin poisoning, administration of exogenous calcium will result in “stone heart” from excessive intracellular calcium. This concept has been in the literature since 1927, based primarily on animal studies. Documented cases of cardiac arrest after calcium administration are exceedingly rare in the literature, and the temporal relationship is dubious.
    iii. Digitalis ECG:

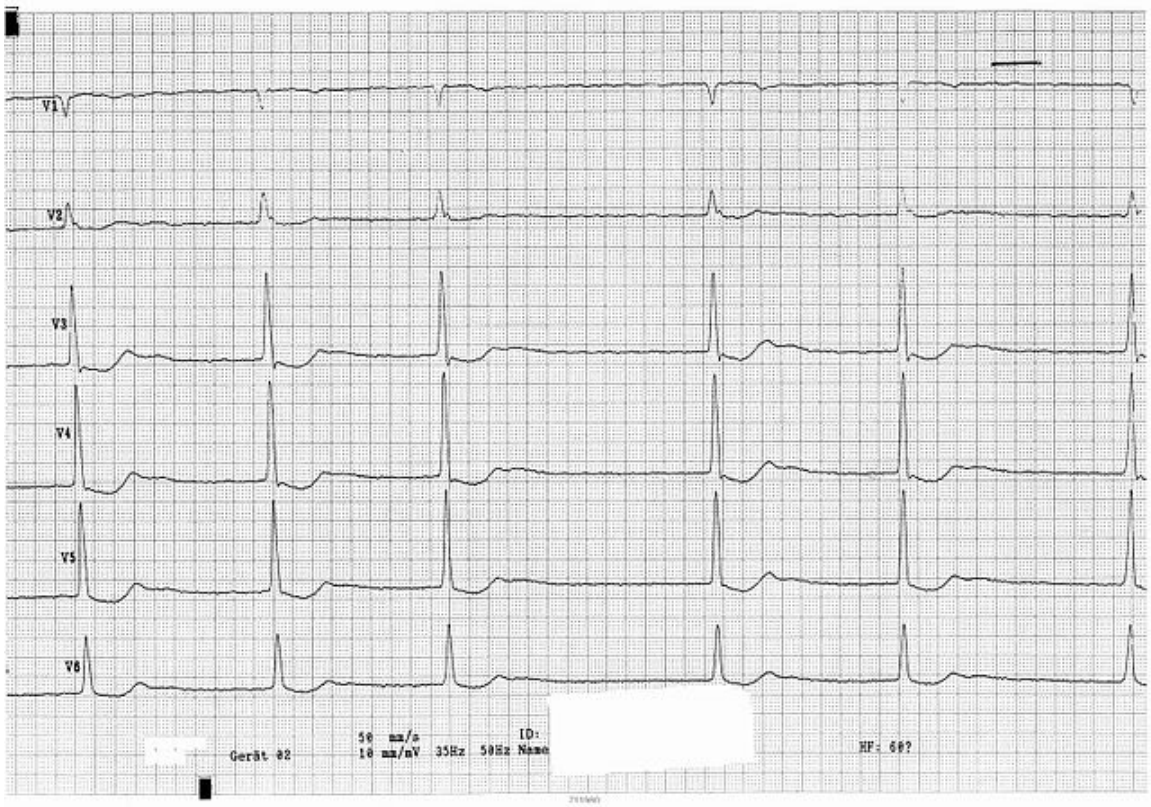


Image source: Patho. ECG *atrial fibrillation *Serum digitoxin concentration 31,5ng/mL. In: Wikimedia Commons. https://commons.wikimedia.org/wiki/File:ECG_005_b.jpg. Public domain.

Case 3: A 78-year-old man with a history of multiple myeloma presents with constipation, generalized fatigue, nausea, and diffuse abdominal pain. He is in the midst of a work up for his myelodysplastic syndrome (MDS), and he has not yet started treatment. His ECG shows a short QTC and his serum calcium level is 14.5 mg/dl..

Question Prompts:

1. What are likely causes of his hypercalcemia?
    a. His hypercalcemia is likely related to multiple myeloma or his MDS. Other cancers which commonly cause hypercalcemia are cancers metastatic to bone, breast, lung, and hematologic, kidney, and prostate cancers. Malignancy-associated hypercalcemia occurs in up to 10% of all patients with advanced cancer, and generally conveys a poor prognosis.
2. What are the classic signs and symptoms of hypercalcemia?
    a. Symptoms of hypercalcemia can be remembered with the mnemonic "stones, bones, abdominal groans, psychiatric overtones."
        i. Stones – nephrolithiasis
        ii. Bones – osteoporosis, osteomalacia, arthritis
        iii. Abdominal groans – constipation, nausea, vomiting, peptic ulcers and pancreatitis
        iv. Psychiatric overtones – effects on the central nervous system range from lethargy, depression, memory loss, psychosis, ataxia, delirium to coma
3. Discuss primary disease of the parathyroid gland:
    a. Main function of parathyroid glands is to regulate the body's calcium and phosphate levels. Parathyroid hormone (PTH) increases calcium levels by stimulating gastrointestinal and renal calcium absorption, as well as bone breakdown.
    b. Hyperparathyroidism can be caused primarily due to increased proliferation of cells in the parathyroid, or secondarily due to renal disease. It leads to increased calcium circulation, which can cause bony pain and tenderness, myalgias, kidney stones, depression, constipation, peptic ulcer disease, and dehydration. It can be seen in multiple endocrine neoplasia (MEN) syndrome type 1 or 2a.
    c. Hypoparathyroidism leads to decreased circulating calcium, which can lead to muscle cramps and spasms, headaches, fatigue, insomnia, and bony pain.
4. What are initial steps for management?
    a. Remember, when checking calcium levels to order ionized calcium which measures free calcium in the blood instead of calcium which is bound to proteins in the blood such as albumin. Most hypercalcemic patients are volume contracted and therefore initial treatment is with rehydration. Use of loop diuretics has fallen out of favor. Osteoclast-inhibiting therapies for severe hypercalcemia include the bisphosphonates, mithramycin, calcitonin, and glucocorticoids. Generally, these therapies are considered in the inpatient settings and are not initiated in the emergency department. In general, all patients with hypercalcemia should be admitted.

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Appendix C:
Pituitary Disorders

Objectives

1. List the primary hormones secreted by the pituitary gland.
2. Describe the common etiologies of Cushing's syndrome.
3. Discuss the evaluation and management of Cushing's syndrome.

Case Studies

Case 1: A 58-year-old man with no significant past medical history presents to the emergency department complaining of several months of progressive fatigue and diffuse weakness. Today he had difficulty getting out of bed. Review of systems reveals intermittent headaches and nausea, as well as new onset sexual dysfunction.

Vitals signs are as follows: heart rate 85, blood pressure 90/65, respiration rate 14, pulse oximetry 98% on room air, temperature 37°C.

Physical exam reveals a tired-appearing man with evidence of dehydration. Heart and lung exams reveal no abnormalities. Abdomen is soft and non-tender. Neurologic exam reveals normal strength in all extremities and 1+ reflexes diffusely. Skin appears coarse and dry.
His electrocardiogram (ECG) shows a 1st degree atrioventricular (AV) block with U waves noted. Initial labs are notable for hypokalemia and hypomagnesemia, with low thyroid stimulating hormone (TSH) and low free T4.

Question Prompts:

1. What is the likely diagnosis? Describe the pathophysiology and common etiologies of this condition
    a. This presentation is consistent with panhypopituitarism, a clinical condition in which a disorder of the pituitary gland or hypothalamus cause deficiency of all pituitary hormones.
    b. Pituitary adenoma is by far the most common cause, usually related to iatrogenic causes (surgery, radiation).
    c. Other causes include traumatic brain injury, subarachnoid hemorrhage, stroke, neoplasm, and autoimmune disorders.
    d. Remember that pituitary adenomas can cause visual impairment due to chiasmatic compression. Also remember that partial hypopituitarism can occur which is deficiency of one or several (but not all) pituitary hormones. Presentation of hypopituitarism has a wide range of presentations, from asymptomatic to acute hemodynamic collapse.
2. How should this patient be managed in the emergency department?

a. Initial management should focus on airway, breathing and circulation (ABCs). Treat hypotension and electrolyte abnormalities with fluid resuscitation.
b. Consider thyroid function tests in all patients that you are considering endocrine abnormalities.
c. Initiate treatment for adrenal insufficiency with crisis with hydrocortisone 100 mg. This provides both glucocorticoid and mineralocorticoid effects. If an adrenocorticotropic hormone stimulation test is anticipated, then could consider IV dexamethasone 4mg bolus which will not affect this testing. If patients are hypotensive, first administer steroids prior to initiating pressors for those that are not fluid responsive.
d. Patient will likely require imaging with magnetic resonance imaging (MRI) of the brain to help identify the underlying cause.

3. List and briefly describe the functions of hormones secreted by the pituitary gland.
   a. Adrenocorticotrophic hormone (ACTH): Stimulates cortisol release by the adrenal gland. Deficiency produces adrenal insufficiency.
   b. Thyroid-stimulating hormone (TSH): Stimulates thyroid gland to release T3 and T4. Deficiency produces hypothyroidism.
   c. Gonadotropins: Follicle stimulating hormone (FSH) and luteinizing hormone (LH): Act on the gonads to release sex hormones. Deficiency causes sexual dysfunction, decrease in libido and osteoporosis. Men lose facial and pubic hair. Women will display oligo- or amenorrhea.
   d. Growth hormone (GH): Acts on tissues of the body as an anabolic steroid. Deficiency causes weakness and fatigue in adults and failure to thrive and short stature in children.
   e. Antidiuretic hormone (ADH) also known as vasopressin: Acts on the kidney to increase free water absorption, and constricts arterioles to raise blood pressure. Deficiency causes diabetes insipidus (polyuria and polydipsia).
4. Describe pituitary apoplexy and Sheehan's syndrome.
   a. Pituitary apoplexy is bleeding or impaired blood supply to the pituitary gland (usually in the presence of pituitary tumor) presenting with a sudden headache, worsening visual field deficit and acute hypopituitarism – predominantly adrenal insufficiency with hemodynamic collapse.
      i. Diagnosed with MRI and usually requires surgical decompression.
   b. Sheehan's syndrome is hypopituitarism caused by ischemic necrosis of the pituitary due to blood loss and hypovolemic shock post-partum.

Case 2: A 38-year-old female presents with a concern for a mass on her back. She has no significant medical history and takes no medications. Review of symptoms is positive for irregular menses for eight months. Vitals are as follows: heart rate 78, blood pressure 140/90, respiration rate 14, temperature 37°C. Lung and heart exam are normal. When you enter the room, this is how the patient looks:

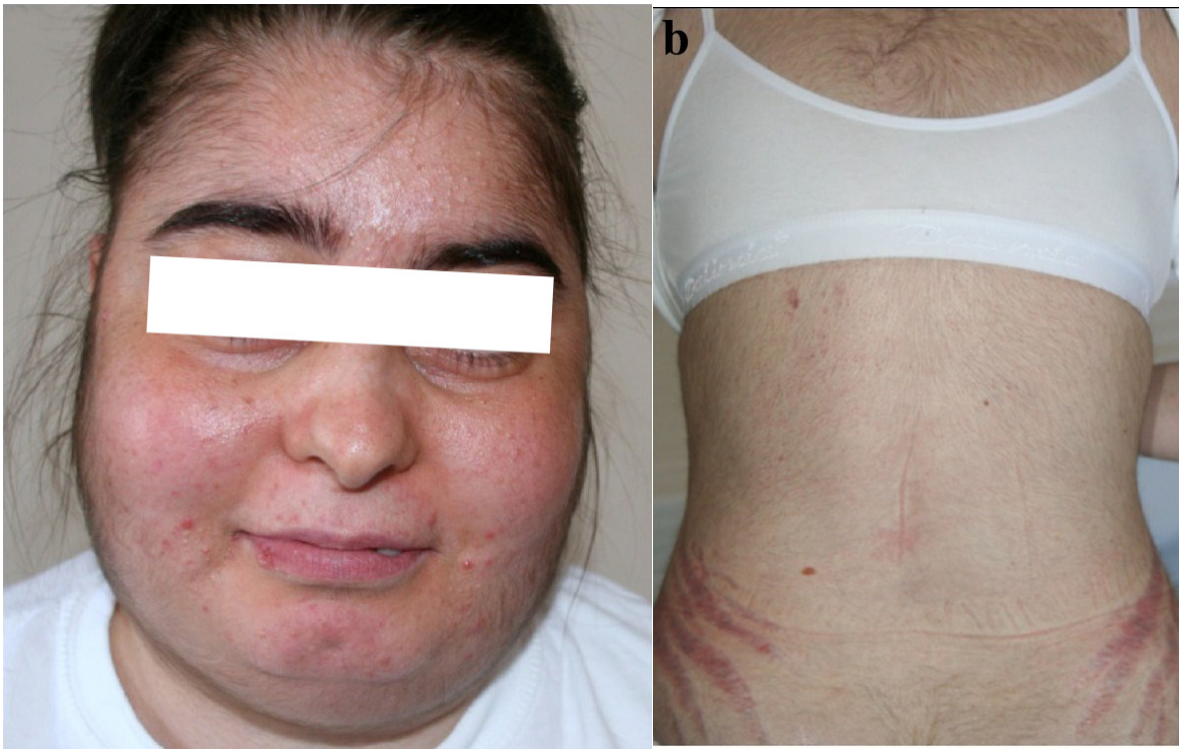

Image source: Celik O, Niyazoglu M, Soylu H and Kadioglu P. Iatrogenic Cushing's syndrome with inhaled steroid plus antidepressant drugs. In: Multidisciplinary Respiratory Medicine. https://doi.org/10.1186/2049-6958-7-26. 2012; 7:26. CC BY 2.0.

Question Prompts:

1. Describe the physical findings pictured in the photos.
    a. The diagnosis is Cushing's syndrome. This patient demonstrates moon facies (abnormal facial distribution of fat in the face), facial acne, striae of the trunk, and hirsutism (facial male pattern hair growth).
2. What is the patient's complaint of a back mass most likely?
    a. The mass on her back is likely caused by lipodystrophy with a dorsocervical fat pad.
3. What other physical exam abnormalities might you expect?
    a. Supraclavicular fat pads may also occur. Truncal obesity with thin arms and legs is a common finding. Osteoporosis and poor wound healing are also seen. Hypertension is common.
    b. In patients with a pituitary adenoma causing their symptoms, central compression of the optic nerve can cause loss of *peripheral* visual fields so remember to check visual fields by confrontation.
4. What are the most common causes of Cushing's syndrome? What is the difference between Cushing's syndrome and Cushing's disease?

a. Cushing's syndrome is a constellation of signs and symptoms caused by prolonged exposure to excess cortisol. By far the most common cause of Cushing's syndrome is iatrogenic due to corticosteroid use.
b. ACTH-producing pituitary adenoma ("Cushing's disease") is the second most common cause.
c. Other causes include ectopic ACTH syndrome (certain lung tumors can produce ACTH) and adrenal adenomas.
d. The most common findings associated with Cushing's syndrome are decreased libido, weight gain/obesity, plethora, round face (moon facie), menstrual irregularity, hair growth, hypertension, bruising, fatigue/lethargy, dorsal fat pad (hump back).
e. Less common findings include nonspecific ECG abnormalities, atherosclerosis, abdominal striae (stretch marks), edema, proximal muscle weakness, osteopenia/fracture, headaches, backaches, recurrent infections, abdominal pain, acne, female balding.

5. How would you make this diagnosis in the emergency department?
   a. Careful history taking should be able to guide whether Cushing's syndrome from exogenous steroid use is likely. If a pituitary adenoma is suspected, an MRI should be done; make sure that radiology knows the indication for the imaging because the MRI should be protocoled particularly for this. Computed tomography (CT) is notoriously unreliable at diagnosing pituitary or imaging the sella.

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Appendix D:
Diabetes and Diabetic Ketoacidosis

Objectives

1. Describe the laboratory findings and treatment for children presenting with new onset diabetes.
2. Discuss the risks and benefits of bicarbonate administration for severe acidosis during diabetic ketoacidosis.
3. Describe the risk factors and treatment of cerebral edema.
4. Discuss the common presenting symptoms for hypoglycemia and atypical presentations.
5. List risk factors for development of severe hypoglycemia.
6. Discuss the diagnosis and management of hyperglycemic hyperosmolar non-ketotic state.

Case Studies

Case 1: A 7-year-old girl presents with her mother for lethargy and weight loss. Her mother reports she has lost seven pounds over the past several weeks, has excessive thirst, and has been wetting the bed. This morning, she appeared to have difficulties breathing and was difficult to arouse from sleep. She has minimal verbal response and only states her "tummy aches." Her vitals are as follows: heart rate 130, blood pressure 70/50, respiration rate 34, temperature 37°C (98.6°F).

Question Prompts:

1. What laboratory analysis would most help you in this situation? What results would you expect? Explain the pathophysiology behind these results.
    a. This patient is presenting with a clinical picture highly suggestive of diabetic ketoacidosis (DKA). Labs ordered should include basic metabolic panel, blood gas and beta-hydroxybutyrate.
    b. Laboratory findings would include hyperglycemia associated with acidemia, anion-gap metabolic acidosis, and ketosis, usually seen indirectly as ketonuria on urinalysis.
    c. Electrolyte abnormalities are common, including derangements in potassium and sodium. Total body potassium is depleted, though serum potassium levels may vary as acidosis leads to shifting of intracellular potassium into the intravascular space. Serum hyponatremia is commonly seen, though a significant component of this is usually due to dilutional hyponatremia from the significant hyperglycemia. Classically a corrected sodium can be calculated by adding 1.6 to the measured sodium value for every 100 mg/dl glucose over 100 mg/dl. A quick bedside "ballpark" test can be to add 1 for every 100 mg/dl glucose, knowing that this will under-estimate true sodium. For more accurate calculations there are online calculators.

2. Outline your treatment algorithm for this patient, highlighting the difference between adult management of this disease process and pediatric management.
    a. Treatment of DKA focuses on insulin replacement, as well as repletion of volume and electrolytes lost through hyperglycemic osmotic diuresis. The first priority in managing DKA is intravenous (IV) fluid replacement as patients will be significantly volume depleted.
    b. Insulin infusion using regular insulin should be initiated at 0.1 units/kg/hr without preceding bolus, with a goal of closing the anion gap. However, insulin infusion should be held until potassium has been adequately restored. Common recommendations are to hold insulin with serum potassium levels lower than 3.5, supplant potassium with replacement IV fluids for levels between 3.6 and 5.4, and to hold potassium replacement for levels of 5.5 or greater.
    c. Dextrose can be added to maintenance IV fluids as needed once glucose is below 250; either 5% or 10% dextrose can be used and it is wise to abide by your institution's guidelines.
    d. Continue insulin infusion until resolution of ketoacidosis: pH greater than 7.3, bicarb greater than16 mEq/L. At that point transition the patient to subcutaneous insulin.
    e. Bicarbonate administration is not recommended because it has no documented improvement in outcomes and is associated with a 4-fold increased risk of cerebral edema. Bicarb should only be considered in patients with pH less than 7 with associated hemodynamic compromise (administer 0.5-2 mEq/kg over 1-2 hours).
    f. Treatment for adult and pediatric DKA is generally similar. Main differences would be to include a broad differential for inciting causes of adult DKA such as MI (myocardial infarction), infection, etc. These would be unlikely in a pediatric patient with new onset DKA. Also, with pediatric patients it can be easy to over fluid resuscitate, which can pre-dispose cerebral edema. Appropriate fluid resuscitation is 10ml/Kg of 0.9% normal saline as a bolus, and then a maintenance rate at 1.5-2x typical maintenance rate to correct the fluid deficit over 48 hours.
3. What are the most feared immediate complications of this child's presentation? What interventions would you propose?
    a. Cerebral edema occurs in about 1% of all children presenting with DKA, and accounts for 57-87% of pediatric DKA-related deaths.
    b. Risk factors include young age, severe hyperosmolality, persistent hyponatremia, and severe acidosis.
    c. Symptoms of cerebral edema include headache, declining mental status, seizures, and papillary edema.
    d. Standard treatment of cerebral edema includes mannitol (0.5-1 gm/kg IV bolus) and intubation if needed.
4. What illnesses are associated with her primary diagnoses? What baseline tests would you consider to evaluate for these?
    a. Even in a first-time diagnosis, often times there is another inciting cause that tips the newly diabetic patient into DKA. These include infections such as viral infections, gastroenteritis,

urinary tract infections, or even a stressful event. Consider workup such as chest X-ray or urinalysis as warranted by clinical history and exam to evaluate for these inciting causes.

b. Risk factors for DKA in established diabetic patients on insulin therapy also include medication non-compliance.
c. However, an inciting cause for DKA may not be present in the newly diabetic patient not taking insulin.

Case 2: A 32-year-old female with a history of diabetes presents with loss of strength in her right hand and arm and an inability to speak. Her family member states that she was fine when she went to bed last night, but on awakening this morning had difficulties walking downstairs and was unable to speak and unable to move her right arm. The patient's diabetes is managed with an insulin pump, and she has been dieting recently.

Question Prompts:

1. What simple laboratory evaluations will provide a diagnosis, even in a limited lab setting, and what treatment should be initiated? What do you do if you do not have IV access?
    a. This patient is experiencing focal weakness mimicking stroke, a classic finding of severe hypoglycemia. Standard therapy is to administer IV dextrose, 25-75 g for adults or 0.5-1g/kg for children. If IV access cannot be established, consider administering glucagon, 1-2mg in adults or 0.025-0.1 mg/kg in children intramuscularly (IM) or subcutaneously. Onset of action is 10-20 minutes. Glucagon stimulates hepatic gluconeogenesis and is therefore ineffective in cases of glycogen absence, such as alcohol-induced hypoglycemia. Having depleted the hepatic glucose stores, patients are at increased risk of rebound hypoglycemia if not provided glucose supplementation (oral or parenteral) after glucagon administration.
2. What are more classic signs or symptoms associated with this diagnosis?
    a. Classic symptoms of hypoglycemia are shakiness, jitteriness, sweating, tachycardia, and a sense of being "unwell" and fatigued. General symptoms of headache, nausea, and lightheadedness are also common. Focal neurologic symptoms are not common but can occur.
    b. Hypoglycemia without warning or "hypoglycemia unawareness" is a complication of type 1 diabetes caused by previous hypoglycemia episodes blunting a normal neuro-hormonal counter-regulatory response to later hypoglycemia episodes. These patients may become abruptly unarousable without warning. Risk factors for hypoglycemia unawareness include: overaggressive insulin therapy, longer history of type 1 DM, autonomic neuropathy.
3. What risk factors can be identified associated with this case as possible precipitants?
    a. Limited carbohydrate intake is a major risk factor for development of hypoglycemia. Other precipitants include missing a meal (decreased intake), increased energy output (exercise), and increased insulin dosage. It can also occur in the absence of any precipitant.
4. Juxtapose treatment of this patient to a type 2 diabetic with hypoglycemia who is on insulin and oral therapy.

a. Both patients on exogenous insulin and oral antihyperglycemic require IV dextrose, but patients on a sulfonylurea will frequently require an additional agent to further inhibit insulin release, such as a somatostatin analogue (octreotide). It is important to give the patient a meal with simple and complex carbohydrates and protein. Giving only IV dextrose or simple sugars will make it more likely the patient will have recurrent hypoglycemia. Adults on oral agents may need to be monitored for many hours and require an admission/observation stay for further monitoring of blood glucose.

Case 3: An 86-year-old woman with a history of cerebral vascular accident, hypertension, type 2 diabetes and chronic kidney disease presents to the emergency department with confusion, low grade fever, and tachycardia per the ECG. She is unable to add to the history. Vital signs are as follows: heart rate 130, blood pressure 88/56, respiration rate 28, temperature 38.5°C (101.3°F). Her mucous membranes are dry, neck veins are flat, and she has poor skin turgor. Paramedics reported a bedside glucose as "high" (greater than 500).

Question Prompts:

1. What laboratory findings would be expected in this case?
    a. Hyperglycemic hyperosmolar non-ketotic state (HHNK) is defined by severe hyperglycemia (glucose greater than 600), elevated calculated osmolality (greater than 315), associated with mild acidosis (bicarb greater than 15, arterial pH greater than 7.3) and the absence of significant ketosis.
    b. Metabolic acidosis may be present through a combination of lactic acidosis from hypoperfusion, starvation ketosis, and renal hypoperfusion causing retention of inorganic acids. Ketoacidosis with an elevated beta-hydroxybutyrate differentiates DKA from HHNK. In contrast to HHNK, patients in DKA typically have more significant acidosis, low bicarb, positive ketones, elevated anion gap, and can have variable osmolarity.
2. What common precipitants would need to be considered?
    a. Patients are usually debilitated, have limited access to water, and often a precipitating medical event (infection, pulmonary embolism, gastrointestinal hemorrhage, mesenteric ischemia, myocardial infarction, burns, cerebrovascular accident). Physical findings non-specific, usually appear dehydrated and may be hypotensive
3. How would you start treatment? How rapidly would you expect to need to correct total body water deficit?
    a. Treatment of HHNK includes aggressive fluid resuscitation, identifying and treating precipitating causes, correcting electrolyte abnormalities, and gradual correction of hyperglycemia and osmolarity.
    b. Patients usually have an 8-12 L total body water deficit. Usually correct about half in the first 12 hours and remainder in next 12-24 hours. Fluid resuscitation status can be assessed both by vital signs (improvements in tachycardia and hypotension), urine output and physical exam findings such as skin turgor and capillary refill.

c. Insulin drip at 0.1 units/kg/hr with hourly glucose monitoring, add dextrose-containing fluids once glucose <300. However, insulin therapy should only be initiated after adequate volume resuscitation.
d. Potassium repletion with close monitoring to ensure it does not drop too precipitously.

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Appendix E:
Adrenal Disease

Objectives

1. Describe the pathophysiology, evaluation, and management of adrenal insufficiency.
2. Compare the most common testing pathways for diagnosing adrenal insufficiency.
3. Discuss etomidate and prednisone, which could affect the hypothalamic-pituitary-adrenal (HPA) axis, and the literature behind their continued use.
4. Describe treatment of hypertensive crisis in suspected pheochromocytoma.

Case Studies

Case 1: A 26-year-old graduate student presents to the emergency department (ED) with generalized fatigue and lightheadedness. She was in class today feeling weak and dizzy and had a syncopal episode upon standing. She also reports almost daily nausea and intermittent diarrhea, which she attributes to a "fussy stomach."

Vital signs: heart rate 100, blood pressure 70/50, respiratory rate 12, temperature 37°C (98.6°F). On exam, she appears in no apparent distress, but generally thin and somewhat tanned. You note hyperpigmented palmar creases, as well as darkening of her tongue and buccal mucosa.

Two large-bore intravenous (IV) lines are established and she is given a 2L normal saline bolus. Despite this, her vital signs remain unchanged. Laboratory studies are remarkable for hyponatremia (Na 128), hyperkalemia (K 5.0), chlorine of 107, and glucose of 60.

Question Prompts:

1. Discuss the hormone feedback loops involved in the hypothalamic-pituitary-adrenal (HPA) axis.
    a. The hypothalamus secretes corticotropin releasing hormone (CRH), which stimulates the anterior pituitary to release adrenocorticotropic hormone (ACTH) which stimulates the adrenal cortex to produce cortisol. This has negative feedback on both the anterior pituitary and hypothalamus.

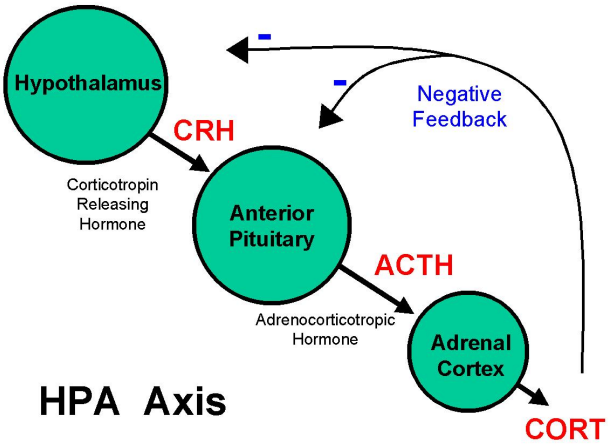

Image Source: Malisch J, Garland T. Basic HPA Axis. In: Wikimedia Commons. https://commons.wikimedia.org/wiki/File:Basic_HPA_Axis.jpeg. Dec 20, 2011. Public Domain.

2. What is your differential diagnosis? Why is this patient in crisis?
    a. In this young woman with refractory hypotension without signs of infectious, hypovolemic or anaphylactic etiology, adrenal insufficiency should be suspected. Physical exam clues include hyperpigmentation of skin and mucous membranes. Chronic symptoms of adrenal insufficiency can be easily confused with anorexia nervosa, occult malignancy, chronic fatigue syndrome, hypothyroidism, influenza, and toxicological causes. Refractory hypotension can be attributed to sepsis, gastrointestinal bleed, or anaphylaxis. As with many endocrine disorders, the symptoms are vague and therefore the differential is broad.
    b. Clinical features of adrenal insufficiency: weakness, fatigue, anorexia and weight loss are nearly universal in patients with adrenal insufficiency. Blood pressures are typically less than 110/70. Gastrointestinal symptoms are very common. Hyponatremia is found in the majority of cases. Mild fevers are common. Other features include depression, myalgias and arthralgias. Auricular calcifications are seen in a small percentage of cases.
    c. Without additional information, it can be difficult to determine the exact etiology of the patient's apparent adrenal crisis. The most common etiology is an autoimmune disorder of the adrenal gland (Addison's disease). The patients gradually waste salts and have other symptoms/complications of adrenal insufficiency. Often an inciting cause, such as otherwise mild infection, will trigger the crisis episode that leads to their ED presentation.
3. What laboratory testing would confirm your diagnosis?
    a. Many testing pathways exist to diagnose adrenal insufficiency, but if there is high suspicion for adrenal insufficiency treatment should be initiated immediately before a definitive diagnosis can be made.
    b. Cortisol measurement is the mainstay of diagnosis. This is done with an ACTH stimulation test. If the adrenal gland is unable to secrete an appropriate level of cortisol after corticotropin administration, a diagnosis of adrenal insufficiency is made. This test cannot differentiate between primary and secondary adrenal insufficiency.

   c. Random basal serum cortisol concentrations are of limited value for assessment of HPA axis reserve, but can confirm an intact adrenocortical reserve if basal morning concentrations are above 500 nmol/L
4. What therapy should be started?
   a. Dexamethasone, 4 mg IV bolus (preferred) *or* hydrocortisone, 100 mg IV bolus
   b. 0.9 NS, 2–3 L in the first few hours
   c. Switch to D_5/NS if hypoglycemia
   d. Treat precipitating illness
   e. There is a distinct advantage in using dexamethasone as initial therapy because its administration will preserve the ability to complete an ACTH stimulation test. Hydrocortisone will significantly interfere with the ACTH axis yielding unreliable testing results.
5. What are the differences between primary, secondary & tertiary adrenal insufficiency?
   a. Primary adrenal insufficiency is caused by an impairment of the adrenal gland, most commonly due to an autoimmune disease called Addison's disease. Other causes include congenital adrenal hyperplasia, adrenal tumors, infectious destruction (HIV, tuberculosis), and adrenal infarction (sepsis, disseminated intravascular coagulation, Waterhouse-Friderichsen syndrome).
   b. Secondary adrenal insufficiency (more common than primary) is when the pituitary gland does not release ACTH. This is most commonly due to pituitary adenoma, though this can also be seen in pathology that causes pituitary destruction, as in Sheehan Syndrome (post-partum pituitary hemorrhage).
   c. Tertiary adrenal insufficiency, *the most common cause of adrenal insufficiency in the United States*, is caused by sudden cessation of chronic steroid use.
   d. Primary adrenal insufficiency characteristically has more pronounced clinical manifestations than secondary and tertiary adrenal insufficiency. Clinical manifestations of primary adrenal insufficiency involve deficiencies of glucocorticoids, mineralocorticoids, and androgens. These include skin hyperpigmentation (particularly in areas exposed to the sun or subject to friction or pressure), salt craving, hyperkalemia, and acidosis. These patients may show signs of sodium and volume depletion (orthostatic hypotension and tachycardia). In secondary and tertiary adrenal insufficiency, patients more often present with pale skin, loss of axillary and pubic hair, decreased libido, and impotence. Glucocorticoid deficiency and low ACTH concentrations may result in hypotension and hyponatremia with relatively unaffected potassium level.
6. Given the literature about adrenal suppression with use of etomidate or prednisone, would you have any concerns about using these drugs? Is etomidate safe to use for intubating critically ill patients? How long of a course of prednisone requires a taper?
   a. Is etomidate associated with acute adrenal insufficiency?
      i. Mechanism: selective inhibitor of adrenal 11 beta-hydroxylase (which converts deoxycortisol to cortisol).
      ii. Mixed evidence: 2015 Cochrane Review did not find strong evidence that single-bolus etomidate increases mortality in critically ill patients (compared with other

induction agents). It did find positive ACTH stimulation test associated with etomidate use, especially at 4-6 hours after dose administered.

1. Conclusion: Etomidate has been shown to cause adrenal suppression based on laboratory testing[3] but a Cochrane review failed to show any strong evidence that etomidate increases mortality in critically ill patients when compared to other induction agents.[4]

b. Prednisone: duration, dosage, underlying disease, and circadian rhythm/timing of dosage may determine magnitude of effect on HPA axis function and likelihood of developing adrenal insufficiency. In most cases, a prednisone regimen lasting less than 14 days will not require a taper.

Case 2: A 32-year-old man with no significant past medical history presents with what he describes as a "panic attack." He had an episode of headache, palpitations, sweating, and intense anxiety while at work. Symptoms lasted about an hour and have since resolved. He reports similar episodes over the last several months, but none this severe. He has no prior diagnosis of anxiety and cannot pinpoint a trigger for these "attacks." He denies illicit drug use and has occasional light alcohol use. He is adopted and has an unknown family history. Vitals: heart rate 110, blood pressure 190/90, respiratory rate 14, temperature 99° F, and pulse oximetry 98% on room air. Exam reveals a generally well but slightly anxious-appearing man, slightly tachycardic but no murmurs or rubs, clear lung sounds, and a non-tender abdomen.

Question Prompts:

1. What condition do you suspect in this patient? Describe the pathophysiology and presentation of this condition?
    a. In this patient presenting with episodes of tachycardia, headache and anxiety, you should suspect pheochromocytoma. Pheochromocytoma is a rare neuroendocrine tumor of the adrenal gland that secretes high levels of catecholamines. The classic triad of signs/symptoms is headache, diaphoresis and tachycardia. Many of the presenting symptoms have significant overlap with primary psychiatric disease, particularly anxiety. Up to 25% of cases may be familial.
2. Describe the diagnosis and treatment of this condition and what should be avoided in treatment of this patient?
    a. Diagnosis is made via imaging and laboratory analysis. Abdominal computed tomography or magnetic resonance imaging (more sensitive) can visualize an adrenal mass. Laboratory studies include 24-hr urine catecholamines and metanephrines (more specific), or plasma fractionated metanephrines (more sensitive).
    b. Acute treatment for pheochromocytoma involves catecholamine antagonists. Alpha-blockade (doxazosin, terazosin) should precede beta-blockade (propranolol or atenolol), because beta blockade in isolation could precipitate an hypertensive crisis due to unopposed alpha stimulation. Definitive management comes though surgical resection of the tumor.

Suggested Readings:

Thiessen MEW. Thyroid and Adrenal Disorders. In Walls RM, Hockberger RS, Gausche-Hill M, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice.* 9th ed. Philadelphia, PA: Elsevier/Saunders; 2018:1557-1571.e2.

Idrose A. Adrenal insufficiency. In: Tintinalli JE, Stapczynski J, Ma O, Yealy DM, Meckler GD, Cline DM, eds. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. 8th ed. New York, NY: McGraw-Hill; 2016:1479-1482.

Additional References:

Hildreth AN, Mejia VA, Maxwell RA, Smith PW, Dart BW, Barker DE. Adrenal suppression following a single dose of etomidate for rapid sequence induction: a prospective randomized study. *J Trauma.* 2008;65-573-579. doi: 10.1097/TA.0b013e31818255e8

Bruder EA, Ball IM, Ridi S, Pickett W, Hohl C. Single induction dose of etomidate versus other induction agents for endotracheal intubation in critically ill patients. Cochrane Database Syst Rev. 2015;1:CD010225. doi: 10.1002/14651858.CD010225.pub2

Karan RS, Pandhi P, Behera D, Saily R, Bhargava BK. A comparison of non-tapering vs tapering prednisolone in acute exacerbation of asthma involving use of the low-dose ACTH test. *Int J Clin Pharmacol Ther.* 2002; 40:256-262.

Schuetz P, Leuppi JD, Bingisser R, et al. Prospective analysis of adrenal function in patients with acute exacerbations of COPD: the Reduction in the Use of Corticosteroids in Exacerbated COPD (REDUCE) trial. *Eur J Endocrinol.* 2015; 173:19-27. doi: 10.1530/EJE-15-0182

Tekwani KL, Watts HF, Chan CW, Nanini S, Rzechula KH, Kulstad EB. The effect of single-bolus etomidate on septic patient mortality: a retrospective review. *West J Emerg Med.* 2008; 9:195-200.

Kadiyala R, Kamath C, Baglioni P, Geen J, Okosieme OE. Can a random serum cortisol reduce the need for short Synacthen tests in acute medical admissions? *Ann Clin Biochem.* 2010;47(Pt 4):378-380. doi: 10.1258/abc.2010.010008

Venkatesh B, Mortimer RH, Couchman B, Hall J. Evaluation of random plasma cortisol and the low dose corticotropin test as indicators of secretory capacity in critically ill patients: a prospective study. *Anaesth Intensive Care.* 2005; 33:201-209.

Small Group Resident Assessment

Session:		
Facilitator (s):		
DATE:		
Small Group 3	Contributes to group discussion	
	BE/ME/EE	Comments
Resident 1		
Resident 2		
Resident 3		
Resident 4		
Resident 5		
Resident 6		
Resident 7		
Resident 8		
Resident 9		
Resident 10		
Resident 11		
Resident 12		

BE—Below Expectations
Minimal discussion during the session
No discussion on the site discussion board
Comments not contributory to discussion or distracting to discussion
Has minimal knowledge of topic as expected of PGY year

ME—Meets Expectations
Contributes to group discussion in a meaningful way
Incorporate textbook/website/podcast reading into discussion
Has knowledge of topic appropriate to level of training

EE—Exceeds Expectations
Contributes to group discussion in a meaningful way
Incorporate literature into discussion
Has advanced knowledge of topic

Small Group Evaluation

The moderator demonstrated adequate knowledge of subject.

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

The moderator's facilitation of the conference facilitated my learning.

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

The overall discussion was relevant to the stated topic(s).

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

The faculty/resident's teaching methods (slides, handouts, videos, etc.) were effective.

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

Faculty Facilitator Evaluation

1. **Preparation – was faculty well prepared?**

Needs Improvement Effective Exemplary

2. **Engaged residents - Encouraged discussion and actively participated, demonstrated enthusiasm?**

Needs Improvement Effective Exemplary

Strengths:

Areas for Improvement:

Reviewer Recommendations:

Resident Facilitator Evaluation

1. **Preparation – was the resident facilitator well prepared?**

Needs Improvement Effective Exemplary

2. **Engaged residents – Controlled and led the session and encouraged discussion, active involvement, and demonstrated enthusiasm?**

Needs Improvement Effective Exemplary

Strengths:

Areas for Improvement:

Reviewer Recommendations:

Evaluation of the Teaching materials

1. **Were the objectives appropriate for the topic?**

Needs Improvement Effective Exemplary

2. **Was the amount of material appropriate?**

Too Short Appropriate Too Long

Strengths:

Areas for Improvement:

Reviewer Recommendations: