>> November 28, 2016. We've been speaking of the endocrine system, chapter 19. And before we go forward, let's go back just a bit. We left you with the adrenal gland. We spoke of the fact that it has two distinct layers, which are functionally quite independent. And so, let's review this final page that we left you with. The adrenal cortex has three layers, which we gave definition to. The outer most layer here at the surface is called the zona glomerulosa. This area produces a group of steroid hormones referred to as mineralocorticoids. And the most important example of that is one known as aldosterone. Aldosterone targets the kidney, specifically the nephron. And promotes the retention, the reabsorption of sodium and water. The next layer, the middle layer, which is quite thick, is called the zona fasciculata. This layer is concerned with a hormone family known as glucocorticoids. These tend to support, that means help to maintain blood sugar. And they do so by promoting the breakdown of fat and/or protein reserves throughout your body. Best example of this kind of hormone is one you may already know about called cortisol. Also called hydrocortisone. And its used medically to reduce inflammation. Inflammation of the skin. Inflammation of a joint. So, it's quite valuable medicine aside from being a hormone. Then, we talked about the zona reticularis, which is right up against the adrenal medulla, as it turns out. This produces a group of steroids called androgens. The most important one here testosterone. So, we left you with this startling discovery. Do females make testosterone? Yes, they do. Where? Right here in the zona reticularis. Its importance is to maintain some of the reproductive anatomy. That is, it maintains the condition of the gonads. But much more broadly and in terms of its impact on the brain, it supports libido. Sex drive. The interest in sexual activity. So, naturally, when it comes to an endocrine gland, there are two possibilities, in terms of abnormality. If the gland is overproducing, that's called hypersecretion. If it's not producing enough, that's called hyposecretion. And these are diametrically opposed. That is, their symptoms are opposites. So, in the case of hypersecretion, hypersecretion of the adrenal cortex, that's also known as Cushing's syndrome or Cushing disease. And it usually features too much. That is, too much of these hormones. And produces, among other things, fluid retention, weight gain because of the fluid retention. And also, poor resistance to infection. The reason for that is too much cortisol, which not only is anti-inflammatory, but also suppresses the immune system. It may also lead to masculinization. But if you're a male, you certainly wouldn't be disturbed by that. But if you're a female, you might be shaving twice a day and that wouldn't be good. So, too much testosterone in a female is never very pretty. What's the opposite? The opposite of hypersecretion is hyposecretion. First described by a physician. His name was Addison. And so, therefore, in his name, Addison disease. This features fluid and mineral depletion. In other words, water loss, dehydration and sodium loss. It also leads to something called hypoglycemia, which means low blood sugar. Obviously, then, hyposecretion essentially the opposite of hypersecretion. And we mentioned that hyposecretion is easy to treat. How do you treat an under active endocrine gland? Give them the hormone, either by shot or pill or some form. And that's called HRT – hormone replacement therapy,

which is common practice in any case of hyposecretion. This table here you have on the next page is not worth much in the way of comment. But it helps focus what you should be concerned with. How should you study this system? You should be aware of the gland in question. That is the location of the gland, the name of the gland, the hormone or hormones that are produced. And the targets of those hormones. And the actions of those hormones. All of these ideas we laid out on the white board last week. So, that is what study of this system really boils down to. So, then, let's move on in no particular order. We've got a few more endocrine glands to consider. And the next one on our table or rundown here is one called the thyroid gland. Pretty familiar. You've seen it, I bet, in the lab. It's named for its proximity to the larynx. What is that dominant piece of cartilage that forms most of your larynx? Thyroid cartilage. This gland is located below the thyroid cartilage. And because of its proximity to the thyroid cartilage, it's called the thyroid gland. In the cat it's actually two distinct glands. That is, one on the left isolated from the one on the right. But in the human being, there's a kind of bridge that joins across the front of the trachea. And that bridge is called the isthmus, which is Latin for a connection. So, the gross anatomy of the thyroid is that it's shaped a bit like a butterfly. Two lobes connected by an isthmus. It's the largest purely endocrine gland and is easy to palpate. If you've ever had a physical exam and the doctor never put his or her hands on your neck, you should go back for a refund because palpating the thyroid is very important, as it can detect early cancer or disturbances of this gland. It's easy to palpate. It's richly vascular. It receives blood from the carotid. And also, from the subclavian arteries, as you can see. Therefore, it's often injured because of its proximity to the skin. It can be injured with any blow to the neck, as a result of any trauma to that area. Now, the gross anatomy is not that exciting or even that complicated. So, let's slice into this gland and look at it through a microscope. In fact, you've got an insert here, which shows the internal anatomy, which is basically a bunch of hollow spheres about a millimeter in diameter. Each of these clear, fluid filled areas is referred to as a follicle. The word follicle in Latin means a bag. And this is a nice illustration of the appearance of thyroid follicles. They're made of simple cuboidal epithelium. In fact, in the lab, early in this course, when you were looking at simple cuboidal epithelium, it very often was from this very gland. The thyroid. So, the cells are simple cuboidal epithelium. And these cells are called the follicle cells. Also known as acini, which is a Latin term for a grape, because they look like grapes situated on the inner lining of these otherwise empty bags. Now, this space is not empty. It's filled with a fluid. And that fluid contains and basically is occupied by the hormone produced by these cells, which is called thyroxine. Also, abbreviated T4. Four because it contains four iodine atoms. The function of T4 is complex. But yet, at the same time, can be simplified because it doesn't target any specific organ. Rather, it has a general effect on all cells. And overall, increases general metabolism. That means it ramps up the consumption of glucose. It ramps up the consumption of oxygen. And the production of ATP. It also helps the body generate heat. H-E-A-T. So, in short, it supports metabolism. It raises metabolic rate. Now,

the cells that are outside the follicles. For instance, these right here. These cells are outside the follicle. And as such, are called parafollicle cells. Also, called C-cells because of the hormone that they produce, which is very different and unrelated to thyroxine. The name of this hormone is called calcitonin, which is almost self-explanatory. Calci referring to calcium. Tonin means to support. And so, as we translate that name, we say fundamentally that calcitonin promotes the deposition of calcium in bone. In other words, it's a bone builder. It also increases the activity of bone building cells, which you recall are called osteoblasts. With just this information, then, you can imagine a medicinal purpose for calcitonin. For what condition would calcitonin be useful for medically speaking? Osteoporosis. Because it would be a bone builder. It would help to restore the strength and integrity of the skeleton. So, as you see, these two hormones have nothing to do with each other. You do recall from last week, we mentioned a hormone produced by the pituitary called TSH. What was that? Thyroid stimulating hormone. And obviously, it targets the thyroid. But just to be clear, it promotes the release of thyroxine. It does not have much effect on calcitonin. In fact, calcitonin is hardly or rarely an issue. What I'm saying is, when this gland dysfunctions, it has mostly to do with the follicle cells. Rarely are the parafollicle cells involved. So, with that said, what are the possible syndromes or changes that you can expect in thyroid disease? Two of them. One is called hyperfunctioning. It's also called hyperthyroidism. Basically, producing too much thyroxine. So, what? You're going to have elevated metabolic rate. Increased consumption of oxygen. Increased production of heat. The flip side is hypofunction with the opposite symptoms. Lowered metabolism. Less oxygen consumption. Less heat production. And a correspondingly slower metabolic rate. In fact, the hyper not only implies to the elevated metabolism, but these people tend to be fidgety. They tend to be restless or listless. Whereas, hypofunctioning, the folks tend to be lethargic. That is, not very energetic. Again, because of the absence of T4. How do you treat hypofunction? Give them what? Thyroxine. And you may know folks that are taking daily levels of thyroid hormone. Now, another condition that develops occasionally is an enlarged thyroid. And you might assume that that would be producing too much of this hormone. Actually, mostly, it's the lack of an element in your diet, specifically iodine. And therefore, the gland gets very large removing as little or as much iodine as may be available in your diet. And the name for that enlargement is called what? [Inaudible]. And you don't see that much in the US of A because what does the government do? Like it or not. Well, essentially, the government puts iodine in salt. And puts iodine in flour. And therefore, avoiding what? Any development of that situation. Hyperfunctioning not only produces the symptoms that we mentioned. But it also causes a lot of adipose to develop or form behind the eye. And that pushes the eyeball forward, which produces a very striking change in the face. Something called exophthalmos. And here's a picture with somebody with exophthalmos. So, unless you missed this day in anatomy class, you would say, oh this person is suffering from what? Too much thyroid hormone. The opposite is too little. And that produces a syndrome called myxedema. You might say, well what's going on here? We have an

elderly woman. But it's not just an elderly woman. We have a lot of fat deposition below the orbit. And around the face. And this is called myxedema. You can read about it in your book. And here are two females both with hypothyroidism, also called myxedema. How do you fix this? What do they have? Not hyper, but hypothyroidism. So, how do you fix this? You give them thyroxine. And, in fact, this is the before and this is the after. Before treatment. After treatment. Does this seem to work? Yeah. Pretty spectacular. So, that's proof that HRT. What's that? Hormone replacement therapy can be useful. Here's a young adult. And from the front doesn't look like anything going on here. But this is pretty suggestive of what? [Inaudible]. A goiter. Again, uncommon in this country. But very common in African countries because of inland populations away from seafood. Seafood and access to marine organisms contain a lot of iodine. So, those people tend not to have goiters. But inland African countries do. And this is a pretty major goiter, wouldn't you say? And needless to say, this would be heavy and unsightly. But it also compromises breathing a little bit. How? [Multiple speakers]. Well, what's it resting on? What is. The trachea. So, it can compress or make the airway a little bit compromised. Anyway, you can treat a goiter with what? What do you have to add back to your diet, which is missing and usually the cause for most goiters? Iodine. And so, you can take iodine as a vitamin supplement. Or you can just get salt, which is iodinated. Actually, today, everybody likes sea salt. Haven't you noticed that? Oh, sea salt. Wonderful. And actually, sea salt, guess what? Comes from the sea. And therefore, automatically it contains a lot of what? Iodine. So, if your choice is plain salt or sea salt, go with the sea salt. Next. The so called parathyroid. The word means what it says. Around the thyroid. Actually, these are small buttons, which are found on the posterior aspect of the thyroid. Hence the name, parathyroid glands. These were historically an endocrine gland, which was unknown and undiscovered for some time because they looked to be part of the thyroid. But, in fact, they're totally separate. So, they're only visible very faintly on the backside of the thyroid gland. And they number anywhere from four to six. About the size of a dime or even smaller. So, the name parathyroid reflects the location of these glands. Now, what do they do? Despite the attachment to the thyroid, they have nothing whatsoever to do with the thyroid. They're completely independent. They contain a bunch of cells that are called principle cells because they are the principle cells, meaning the most abundant. And these produce the hormone PTH, which is commonly known simply as parathyroid hormone. PTH also targets the skeleton. But look, it what? It doesn't promote calcium deposition. It actually removes calcium from what? Releases or removes calcium from bone. And so, rather than be a bone builder, this hormone tends to dismantle bone. And where is this calcium going to go? Once we remove it from the bone, it's going to go into the blood. Where it's averrable for a fetus. So, here's the question. When would it be appropriate to remove calcium from your, from your skeleton? Well, if you're pregnant, does your fetus need an unusually high concentration of calcium to build his or her skeleton? Yes. So, in simple terms. When would this hormone be useful? It would be useful in pregnancy in order to elevate blood calcium

levels for the developing fetus. Now, incidentally, it not only, therefore, increases blood calcium. But in ways that you'll learn about in physiology, it also lowers phosphate levels in the blood. And not surprisingly, it increases the activity of what? Osteoclasts. You recall, osteoclasts are those cells that break down, that dismantle bone. And so, certainly, this hormone would not be desirable in a healthy person. But would be useful in a female during pregnancy. Right now, though, you can see two hormones that are direct antagonists. What was the hormone we mentioned moments ago, which tends to promote the building of the skeleton? Calcitonin. Which one is the antagonist to that? Well, it's apparently PTH. So, just as with the muscular system, there are very often hormones that work at cross purposes. That is, have the opposite actions. There are other, other cells in the parathyroid that are not associated with this hormone. And for no good reason that I can figure out, they're called oxyphils. And no one seems to know what they do, even today. So, the best guess is that they may support the principle cells, or they may actually become principle cells at some point. Other than that, we don't know much about them. But we do know some of the devastating expectations in parathyroid disease. What are the two possibilities again? The gland is over or what? [Multiple speakers]. Under. If it's over secreting, that's called hyperfunction. And that causes very high levels of blood calcium, which is not in itself problematic usually. Although, it may precipitate. That is, it may cause high levels of calcium in the urine. And when you have high calcium in the urine, that may manifest as kidney stones. But remember, where's this calcium coming from? Yes, we know it's increasing the blood. That is, rising in the levels of the blood. But basically, it's being taken from, stolen from the skeleton. Now, would you say the skeleton becomes brittle? No. Because if you take away calcium, what's left is the collagen. And therefore, the bones become not brittle, but pliable. And this is a condition we gave name to very early in the course called osteomalacia, osteomalacia, or softening of the bones. But by far more devastating, indeed lethal, is not hyperfunction. But what? Hypofunction. Because this decreases blood calcium to dangerous levels. In fact, causes what's called neuromuscular hyperexcitability. It causes skeletal muscle to contract spontaneously. And you might not worry or think much about that. But what skeletal muscle can you not afford to have spasticity develop in? [Multiple speakers]. Diaphragm. If your diaphragm is contracting irradicably or poorly, then that's going to halt breathing. And so, this disease is fatal because of respiratory arrest. However, it's easy to treat. How do you treat hypofunctioning? Remember, if, if you're missing the hormone, you would just give the hormone. But in this case, it's even easier. It's even cheaper. Nobody receives calci, excuse me, parathyroid hormone. Because it's way too expensive. The problem here is easier to fix because hypofunctioning is basically just what? And how can you raise blood calcium? Hm. Give them calcium in the form of a pill or dairy products or so forth. So, this is something that doesn't really require HRT. But rather, just elevating the calcium levels through dietary change. Here's a slide that maybe doesn't look like much to begin with. But over here, you'll see these, these containers, which are spherical groups of cells, which are actually simple cuboidal

epithelium. So, actually, this is the thyroid gland. And these are the follicles, which we mentioned. These cells here, which are outside the follicles, those were called parafollicle cells. And this tissue here belongs to this gland, which is physically in contact with the thyroid. But yet, functionally independent. So, most of these cells are the principle cells, which are manufacturing PTH – parathyroid hormone. So, I think you can see if the thyroid gland were to be removed, there would be some devastating effects. You would miss the thyroid hormones. You would miss calcitonin. And you'd take away the source of this hormone. PTH. So, all said, the thyroid gland and the parathyroid gland are pretty important for normal health. Let's go. Let's move on to the next endocrine gland, which is actually not just an endocrine gland. In fact, we've already discuss this in the context of the digestive system. The name of the gland is the pancreas. The prefix pan means entirely or completely. And the other part of the word means flesh. So, the rough translation of this word is all flesh because it looks kind of fleshy. Whatever that means. When you see it in a gross specimen. And you have seen it already. If you haven't, I'd be surprised. You've had opportunity to locate this in your cat. And you know already where to, where to find it. It's in the curvature, the inner curvature of the duodenum. It's size and shape we've already described. It's often compared to a hammer or a kind of fish because it has a long extensive tail. So, you know about its location. You know about its gross anatomy. And you know incidentally that it is pierced by a duct, which is coming down from the gallbladder. The name of that duct is the common bile duct, which merges with the pancreatic duct, as seen, to form a nipple. You know the name of that nipple. The hepatopancreatic papilla, which secretes into the duodenum. So, to back up a bit. This structure, as is the duodenum, is located not in the abdominal cavity. It is retroperitoneal along the inner curvature of the duodenum. Now, microscopically, we've cut through here with this insert. And we discovered that this gland is not just exocrine. That is, it does more than secrete digestive enzymes. It's also an endocrine gland. So, this gland is both what? Exocrine and endocrine. Very different functions, which are totally unrelated. So, you know that there are ducts that are seen here throughout the interior of the gland. And those are called pancreatic ducts. Also, called acini. And these are exocrine producing and secreting enzymes through the hepatopancreatic papilla. And thus, make digestion of various food constituents possible. Incidentally, you know that gallstones can make their way through the common bile duct. And as such, they may at times block this nipple. And therefore, prevent the passage of these enzymes, which produces a lot of pain and discomfort, i.e. inflammation of the pancreas, which is called pancreatitis. But outside these ducts and isolated in their own little spaces are referred to as the islets of Langerhans. Obviously, a reference to somebody. And his name was Langerhans. He was a Dutch anatomist. And he discovered these and determined they're quite unrelated to the digestive role of this particular gland. There are at least three types of cells in these islets. Two that you need to know by name. First, the alpha cells. These are those producing a hormone called glucagon. And the function of glucagon is complicated, but the bottom line is that glucagon does what to blood sugar? Raises it. It does so by

targeting the liver and causing the release of glucose from the liver. By far, the most troublesome and problematic of the cells in this area, though, are the beta cells. These produce the hormone that you all know by name called insulin. Its role is to lower blood sugar, which might at first seem dangerous. And it can be. But let's be clear. What insulin is doing is escorting glucose from the blood into cells. And do all cells need glucose? Yes. So, insulin is indispensable as it provides a way for cells to acquire glucose. And therefore, survive. I think its pretty common knowledge that the most common endocrine abnormality in the world relates to these cells. That is, the underactivity of these cells. And the name of that condition naturally is diabetes. So, when it comes to pancreatic dysfunction, we're really not going to comment or consider digestive issues. And neither are the alpha cells usually at fault. The number one problem that is important medically speaking is beta cell failure, which can occur early or late in life. And when it happens, of course, it produces diabetes. Occasionally, these cells are overactive. And that would produce too much what? If the beta cells were overactive, then we'd be producing too much insulin. And the blood sugar would be very low, dangerously so. And the name for that would be hypoglycemia, which would affect the brain and cause unconsciousness. Again, this is uncommon. But at the same time, can be common. Not because of the beta cells. But because of misuse of insulin. Are there people that inject themselves with insulin routinely for their own health and survival? Yes. And can they sometimes OD? That means overdose. And what if somebody gave somebody a shot of insulin, which was way beyond what was appropriate? That would cause H-Y-P-O what? Hypoglycemia. And could be lethal, in fact. Hence, insulin is a dangerous drug if you don't know what you're doing. But still, overactivity of the beta cells not common. Underactivity of the beta cells very common. And goes by the common name diabetes. But actually, the first of two words. The second word is mellitus, which means sweet or sugary. So, the complete translation is a bit obscure. But diabetes as a word means to pass through. To pass through. And mellitus means sugary, right? So, this deserves a little explanation because if you know a thing or two about diabetes, diabetics go to the bathroom a lot, yes? That is, they have huge quantities of urine. And that urine is sweet by virtue of the presence of sugar. Afterall, when this hormone is missing, then you're going to have not low blood sugar, but high blood sugar, which will appear in the urine. And be usually the first diagnostic sign for this condition. Historically, from a medical history standpoint, you could argue that diabetes was discovered by dogs and by ants, which I know is a stretch. But observers noted that dogs and ants were licking up this urine from people that were dying in the streets. And so, they tested it. And found that sure enough it is sweet. And therefore, started to investigate the real cause of the problem, which is the absence of this hormone. Can we treat diabetes? Yes. Can we cure diabetes? No. How do we treat any underactive gland? We just give them the hormone. That can't be taken orally, because insulin is a protein, which is digested by the digestive tract. So, diabetics have to what? Inject themselves on a daily basis. Now, without getting too far into physiology, we mentioned that diabetes can be early onset or late onset. When it happens in early childhood,

it's called type one diabetes, which is due to a failure of what cells? Beta cells. Later in life, it has little to do with beta cells. Rather, instead, it has to do with obesity. And so, you know that at least one third of the people in the US of A are what? [Multiple speakers]. I'm not making this statistic up. Over a third of the US population is technically obese. And therefore, even young children have what used to be called adult onset, now simply called type two diabetes. And the treatment for that is very simple because they're not really lacking insulin. Rather, their cells are resistant to insulin. And it has to do in part with their high body weight. So, how do you treat this type of diabetes? Diet and what? Exercise, which pretty much eradicates this condition. You'll learn a lot more about diabetes naturally in phys. So, let's press ahead. To the gonads. The ovaries. And then, we'll give due respect to the testes. The ovaries, you might say, are reproductive structures. But they're also bona fide endocrine glands. At least the ovaries are protected a bit by the bones of the pelvis. And here, they're seen in their natural position and location. They're about the size of a large almond, let's say. Maybe one or two inches in length. And they're inside the pelvic cavity. Actually secured. That means attached to the uterus by way of a ligament you know already is called the ovarian ligament. You might have learned that in lab by now. Microanatomy is interesting. And we'll spend more time on this topic next Monday. But for now, it's essentially important to cut through an ovary. At least an adult ovary. Not a child's ovary. And we'll find various cells in various states of development. In a primitive and young ovary, there are thousands of these cells called follicle cells, which form a cluster known as a follicle. And these will grow on a monthly basis under the influence of a pituitary hormone with an appropriate name. What was the pituitary hormone that targets these follicles? FSH, which stands for follicle stimulating hormone. And under the influence of that hormone, on a monthly basis, usually one of these follicles will develop into one of these structures, which can be called a mature follicle. Also known as a Graafian follicle. Then, on day 15 or so of the menstrual cycle, this thing, which is a fluid filled follicle, will literally tear through the surface and out will come a single cell surrounded by many others. And that act or event that happens is called, you know, ovulation. Which some women say they can feel because there is a bit of a pain associated with it. Now, after this event, the structure, which was known as the Graafian follicle reforms into something that then changes names. And remains for at least two weeks. It's called the corpus luteum. Corpus means body. Luteum means yellow. So, guess what? It's a yellow body. And even though it's a vestige of cells that remain from the ruptured mature follicle, it assumes importance. That is, at least for two weeks, it manufactures and secretes estrogens and progesterone, which support the uterus and make it ready for possible implantation. So, the overall functions of estrogens and progesterone is to maintain the integrity, the readiness of the female reproductive system. So, backing up a little bit. What makes a young girl a young girl, is the absence of two pituitary hormones. One which we just mentioned is FSH. And the other one from the pituitary is LH. When these are secreted, then the ovaries become functional. And remain so until their depleted of these units known as follicles. And when a female's

ovaries contain no more follicles, then he, not he. Then, she would enter what stage? Stage of life, 45 thereabouts. You know it's the M word. Menopause. And it's not because of a lack of these hormones. It's for the lack of estrones and progesterone. And a complete exhaustion of these available units known as follicles. Now, that was quick. And, of course, simplistic. But we'll come back to that on Monday. In men, of course, we have testes. Testes are a similar size. However, their location quite different. They're located outside the pelvic cavity. Or they should be anyway. They're located in a sack, which is suspended below the pelvis. You know that to be the scrotum. And the reason for this is not without function. Because the scrotum is quite a bit warmer or cooler than the body temperature here? Cooler. And this promotes the production, makes possible the production of sperm. So, they're located outside the pelvic cavity within the scrotum. Microscopically, if you cut through a testicle, you'll see there are no follicles. But rather, hundreds of feet of tubes. This is a section through those tubes. And the name of those tubes, which are stuffed in here. They're called seminiferous tubules. And they produce sperm, but not constantly. That is to say, only at the onset of. Well, let me put it another way. Do eight-year-old boys have sperm? We hope not. Do they have testes? We hope so. What's missing is this hormone FSH, which promotes the activity of the seminiferous tubules. And therefore, produces sperm. That said, once this process is set in motion, do men produce sperm throughout their life? Throughout their life span. Are there men in their 80's that are impregnating women? Believe it or not. So, Google that and find out. Because I want to know. Who is the oldest dude that has fathered a child? I just want to know that. But, anyway, interesting fact, unlike females then, a male's reproductive capacity endures throughout his life. Now, outside. These are seminiferous tubules. Outside the seminiferous tubules, there are cells that are called quote, quote interstitial cells. And that's because they are in the interstitial space outside these tubes. They produce the famous or infamous hormone testosterone. But again, that's contingent on these pituitary hormones, especially this one. Which in females is called LH. But in men, if you look back to your notes, it's called ICSH. What's that stand for? I-C-S-H is basically just this. Interstitial cell stimulating hormone. Which obviously targets the interstitial cells. And promotes the production of testosterone. Testosterone, just as with estrogen, promotes the integrity of the male reproductive tract. And also, is responsible for secondary male characteristics, i.e. facial hair, deeper voice, muscle mass, etcetera. So, let's finish, as we must with the pineal, which has nothing to do or very little to do with reproduction. The pineal is so called because it resembles a pine cone. There it is, right there. You know about it already. It's attached to the posterior wall of the third ventricle. It's part of the brain. It's made of nerve tissue. So, it's position and size are familiar already. It's very tiny, weighing less than a 10th of a gram. What's it do? Well, microscopically, it's made of nerve cells, neurons, which in this location are called neuroendocrine cells. And the only significant hormone that has been traced to this gland is one that's called melatonin. Now, this hormone appears to be in part responsible for inhibiting the release of pituitary gonadotropins. Now, if you just memorize that, it's a

waste of your time. But what does it mean? Pituitary gonadotropins. What are the pituitary gonadotropins? [Knocking]. So, what does melatonin do? It stops the release of these. And when would that be appropriate and welcome? When do you want these hormones not to be secreted? During childhood. Do you want these hormones to appear in a five-year-old? Look out. No way. So, what prevents premature puberty is the presence of this pineal hormone, which is melatonin. This hormone may also be involved in something called circadian rhythms. That means normal wakefulness and sleep, sleepiness throughout the day. And so, with that said, this gland sometimes may be overactive. Very rare. But when it happens, it delays what? Delays the onset of puberty. But if this gland quits too soon. That is, if it stops producing melatonin early, then you're going to have premature puberty. In other words, these hormones are going to be released. And you're going to have, you know, seven-year-old girls menstruating, which wouldn't be particularly great. Now, finally, if you've heard of this hormone and you may, it has little to do with its function on the reproductive system. But actually, this hormone is one of the few hormones that you can actually buy without a prescription over the counter at Costco or Walgreens or whatever. And what's it promoted for? Maybe you've used it. You get a little snippet here. This was a cover story from Newsweek. But it's very often used in what therapy or strategy? To treat jet lag and all sorts of what? Sleep disorders. It is not really a sleeping pill. But it may help to reestablish what? The normal sleep/wake cycles. It's probably overblown and overused. But I mention it because you may see it on the counter at Costco or otherwise have already used it. So, it does have some therapeutic application in that way. So, that's it. We are at least through our superficial look at the endocrine system. We're going to revisit, of course, the testes and ovaries starting on Wednesday.