>> Prof. Steve Langjahr: February 27, 2017, lecture six actually. This is the second lecture in unit two. Remember we spoke of the structure, the organization of the skeleton, gave you some terms that are used to describe various morphological features on any given bone. But today we're going to look at the science of bone per se, osteology. In other words the histology and the development of bone tissue. So let's begin with just a casual observation of any given bone. This is an actual femur from our lab, and you'll have a chance to look at it. In fact I'll pass it around. But regardless of what you call it, what are some features of any bone? First as we examine especially a section of bone we find that despite appearances it's not, it's not solid at all. There is a very hard exterior which is known by the name cortical. The word cortical means bark. B A R K, just like the bark of a tree trunk. Another name for cortical bone is compact bone because to the naked eye it looks solid. But actually it's not. Is a piece of wood solid? Yes. But if you really look at it, it's porous enough, and we'll have a closer look at cortical bone in a moment. Basically cortical bone is what you're seeing under the microscope and is made up of dense concentric layers or units, somewhat overlapping units known as osteons, also known as harversian systems. So certainly the name suggests that this bone is hard, it's firm, and the function of cortical bone is simply to sustain weight that is to support the body, and you find it where support or physical protection from trauma is necessary. So here's the femur actually magnified just as I gestured to this. And this bone at the surface is what? Cortical bone. Now despite what it seems like on the outside, when you cut through a bone you find that much of the interior is very delicate, very lacy, very much open. And this is called spongy bone. But the proper name is cancellous bone. This is essential an open lattice work of conflicting or overlapping struts, also known as trabeculae, which basically serve as internal scaffolding. You know what a scaffold is? Sometimes when a building is being cleaned or built there are these super structures on the outside where workers might stand. So compare this to scaffolding because it's providing support with one notable advantage. What is the advantage or purpose of cancellous bone? It provides strength. It provides support without what? Weight. It reduces the overall weight of the bone. In fact you might argue, you might think that the bone would be stronger if it was entirely cortical bone, if it were solid all the way through. The truth is it wouldn't be stronger, but it would be way what? Heavier. And that's a disadvantage not only in terms of lugging it around, but bone has to be maintained. Bone has to be fed. Remember, bone is living tissue. So the body conserves weight by reducing weight, and especially evident by the presence of cancellous bone. In fact there are some areas of the bone where there is no bone at all. Complete emptiness. And those spaces, typically along the shaft or longitudinal channel within a bone, those spaces are called medullary canals. The word medulla means core, C O R E. And even though I've said that core is empty, in life it's not. It's not air filled, but at least in humans it's filled with this yellow compound which later we'll describe as yellow marrow, but already you expect it to be based on the color alone, what? Fat. And so yellow marrow is fat. Now we'll make it clear later this fat is not the property of bone, and it's certainly not particularly even

important to the bone. There is plenty of fat around the body. And so in fact in birds this space is entirely empty. Why would it be a disadvantage to a bird to have anything in their medullary canal? Weight. So medullary canals often are filled with adipose which later we'll describe as yellow bone marrow, quite different from red bone marrow in terms of its histology as well as its function. So moving from that slide, here we see a close up view. This is not compact bone, this must be cancellous bone. And these beams or struts are called trabeculae, again very similar to that game that you might have played as a kid, you know, where you pull out the pieces and, remember, Jenga. And so certainly the integrity of these trabeculae determine the strength. And we spoke of a disease where these beams become less numerous. What's the name of that pathological change? Osteoporosis, which leads to bone fractures, especially in elderly folks. So aside from that, as we look at this bone or any bone, at the surface you'll find some visible openings that are macroscopic. That means able to be seen with your own eyes. And these actually provide entry or exit for vessels, blood vessels, major or substantially large vessels. These are called nutrient foramina. In other words holes for veins or arteries. And that reminds us of a fact that we've made clear in the past, are bones vascular? Yes. If you break them will they bleed? Yes. And that's because of the numerous blood vessels that enter or leave any given bone at any given spot through these holes. Although this is a dried bone, if it were a fresh bone we'd be able to also dissect off the surface a membrane. Now in this case I put some masking tape to represent that. But the membrane that covers many of the outer surfaces, that membrane is called the periosteum, which is literally what it means. Peri means around, ostium bone. The periosteum is a membrane made of dense irregular connective tissue. And it's the source of cells, bone cells that will be activated or recruited whenever a bone is broken or whenever weight, that is the body weight is increased and the bone has to increase in strength. So the periosteum is the source of bone cells useful in repair and useful in growth or changes in the bone overall. So I'll send this around because you really should look at it. Again to repeat, the outer layer design for support is called the cortical bone. Deep to that we have this bone which is airy and spongy, that's called cancellous bone. In some parts of the bone there is no bone at all, an empty space called the medullary canal, which is often not empty but filled with fat, and the name of that fat is yellow bone marrow. Then perforating the surface of even the cortical bone nutrient foramina. And finally the membrane that surrounds much but not all of the outer surface, that's called the periosteum. So in this view, which is a scanning electron micrograph, we can see the cortical bone here, and this which is sort of overlapping struts of bone tissue, these are called trabeculae. Obviously this is cancellous bone. Here's a femur at the surface, just like, just like the one that's moving around the room. And so what are these holes that are visible to the naked eye even in the outer cortical bone? Nutrient foramina. In life blood vessels would enter and leave through those holes. Let's go on. Here's the level of anatomy that you're more familiar with, and that's the microanatomy. You've studied this and it's easy to correlate I think with this diagram. We said that cortical bone was made up of overlapping units referred to as osteons.

And here an osteon is illustrated, and in fact you've got a picture of one or at least a diagram of one right in front of you there. Here's an osteon, another, another, another, another. And here is a segment of one which has been blown up and enlarged. Perhaps this image is a little more familiar, again showing an osteon. And the osteon is essentially made up of a central canal which is more commonly called the Haversian canal. This is microscopic. In the lab it looked empty because it is empty, but in life what occupies this space, what fills the Haversian canals are blood vessels, microscopic blood vessels, and also nerve fibers, sensory nerve fibers. With that said, once again if a bone breaks does it bleed? Does it hurt? Yes. Again because of injury not just to the vessels but to the nerve supply as well. So the Haversian canal, aka center canal, is where you'll find blood vessels. I'm sure this image is instantly recognized, and so this is what? Haversian canal. Now where are the actual bone cells? In a specimen like this the answer is they're gone. But at least we know where they were. Where did they inhabit, what was the space that they previously occupied? These black empty gaps here, that's the location, the previous location of these bone cells called osteocytes. They are, by definition, mature bone cells and their function is not to produce the matrix but what? Maintain it. That's an important word to emphasize. You may live in a home. Did you build that home or do you maintain it? You might have built it but chances are you just what? Maintain it. Maintaining the matrix means maintaining the integrity of the chemistry of bone matrix. And, of course, where is the matrix? The matrix is everything sort of beige here, which is naturally by definition the interior cellular material. So the osteocytes are trapped literally in the space that is devoid of matrix. And the name of that space is a lacuna. How do you pronounce the plural version? Lacunae. Lacunae plural, lacuna singular. Actually the word means little lake, little lake, L A K E, because it is a kind of lake even though it's not filled with water. Now I notice that there are visible cracks in the matrix which connect lacunae to lacunae, and eventually these cracks meet and open up into this large space, which is the Haversian canal. These cracks are actually called canaliculi, which means little canal. And they are essentially designed to allow the bone cell, the osteo what? Cytes, to reach out in these cracks. Again we don't want to think of bone cells as just your typical round cell. Bone cells have long extensions which reach into the canaliculi. And what are these bone cells reaching for? What is here in the center canal which these cells need to survive? Blood, but specifically oxygen and nutrients. So these bone cells literally reach out in order to obtain through diffusion oxygen and nutrients available in the blood vessels that are found in the Haversian canals. Notice that these lacunae are organized in circular rings. And notice that the matrix then is layered. That is here's layer one, layer two, layer three. The word for these layers of matrix, lamella plural, excuse me, lamella singular, lamellae what? Plural, rings of intercellular matrix. Referring to this drawing that you see here, we see the lakes. So this is one lamella, two lamella, three lamella, four lamella up five. Why not six? Why not seven? Why not eight? Why not nine? Why not ten? Why are the number of lamellae usually restricted to four or five? If we get further out, now these cells are way too far from what? The blood

supply available, and simply then could not survive. So notice that any osteon, any one of these units typically is made of no more than five lamellae. This is a scanning electron micrograph of a lake. What's the name of these lakes, these pits, these spots? There it is right there, what, the lacunae. So OS, that stands for osteocytes. BM is not bowel movement, that's bone, not bone marrow, bone matrix. And LA, LA is the space, LA is the lacuna. And notice that the bone cell is reaching out through CA. What's CA? C are the canaliculi. So this is a real close up view, something you'll never see in our lab, but it is an osteocyte. Osteocytes don't make the matrix, they what? Maintain the matrix. Which brings us down to a chemical description of that matrix. We mentioned earlier in the course that the matrix is hard. And it's hard by virtue of the calcium and phosphorus salts that it contains. Calcium and phosphate salts are essentially the inorganic component of matrix. And in terms of ratio these salts produce or maintain about 65% of the chemistry. But the other ingredient is something very organic. A protein that you've known for a time. What's the name of this protein? Collagen. Now collagen is strong but it's not rigid. Collagen is F word, flexible. And notice that the collagen provides at least one third of the chemistry of matrix. So it's all too tempting to think of bones as hard, just like this tabletop. But an actual fresh bone, if you had one in your hand is hard, but yet you can also bend it a bit believe it or not. And that bendability is made possible by the presence of collagen. You can do something fun for the kids at home. If you don't have kids, I don't know, round them up from the neighborhood. But if you have a turkey you can strip off all the meat or just eat it, and a turkey leg is basically a femur. I should say a tibia. And you can put it in vinegar for a couple of weeks. Vinegar is a weak acid, yes. And that's going to dissolve which of these components? It's going to dissolve all of the calcium and phosphate, leaving you with just collagen. So as I pull this out of the vinegar it certainly resembles a turkey leg. But notice that it's very what? Very bendable, because there's no calcium here, it's all now 100% what? Collagen. Now this is a very dramatic representation, but it certainly proves what we've said. Bones are not, but actually capable of a little bit of. So, with that said, if you reduce the supply or ratio of calcium, as with rickets, as with osteomalacia, the bones don't become brittle, they become very what? Bendable. Hence what? Bowlegged, as in rickets or osteomalacia. On the other hand if the collagen disappears then now the bone is much more prone to fracture because now it is brittle. Remember the collagen provides a degree of flexibility. So this ratio of two thirds inorganic to one third organic is very important as it dictates the physical properties of bone. If someone asks you the physical properties of bone, bless you, would you say rigid? Well you could say rigid but you'd also have to say with a little bit of give, a little bit of flexibility. Alright. This is the formula for what's called hydroxyl appetite, which is the inorganic compound made mostly of calcium and phosphate. And of course in order to produce this you have to have a diet which is rich enough in what? And if your diet is not rich in this then the bone is not going to become brittle, it's going to become soft, pliable, as with osteomalacia or rickets. So let's move on. We've looked at the micro and the macro. Now the question is what causes bones to develop

at all? There certainly are no bones in an early embryo. So the question is where do bones come from? The process of making bone is called ossification. And there are two means of ossification which you can read about which we'll describe and we'll expect you to be able to explain in your own words. The first of those two methods is called intra, what is it, intramembranous. Intra means within. Membranous, membrane. So in literal terms this is hardening that is ossification from within an existing membrane or sac. In other words hardening within a fibrous connective tissue container. So I've used just this blue marker to outline an arbitrary shape. As it turns out intramembranous ossification is not common and limited to what areas or examples? Only found, only used to form parts of the skull and the what? Clavicle. It's also used any time any bone breaks, it's used to repair fractures. What we're about to do now is give you a step by step chronological outline of the events that take place in this process of intramembranous ossification. The first presumes and involves the existence of this sac. This sac is made of fibrous connective tissue. The first stage is the precursor cells, called osteoblasts, not osteocytes. These osteoblasts actually migrate, that is move into the interior of this rather empty or spacious sac. So those black dots represent what? Osteoblasts. Are they accompanied by other structures? Yes. These cells are accompanied by blood vessels which grow also into this interior and of course provide oxygen and nutrients to these multiplying cells. To repeat, these cells are not osteocytes, they are osteoblasts, and they are supported by the penetration of these blood vessels at the same time. Their infiltration is rather uniform. That is they do not congregate or concentrate in any one area. But they produce random centers of bone growth. And those centers are called ossification centers. At once these cells begin to secrete a protein, a protein C word, collagen. And that collagen fills some of the void, some of the empty space. And so using a different color I'll put some squiggly lines in there, green. So that represents the production, the secretion, the addition of what protein? Collagen. Is the bone hard at this point? Is collagen hard? No, it's very what? So this is not really bone matrix, it's a precursor of matrix. It's called osteoid. And it provides a loose fill, F I L L, to much of the empty space. What has to be added to make this hard? Collagen itself is not hard. So the next step is the addition of, the deposition of calcium salts which fill the voids and form a lattice work of crisscrossing beams. What do we call those tiny crisscrossing beams which we've seen in the images so far today? Trabeculae, which creates a kind of bone, a very airy and open bone. What do you call that kind of bone which is very airy and open? Spongy. So the interior is not compact bone, but rather spongy or cancellous bone, which remains airy but ultimately will be filled with a different kind of tissue, red bone marrow. And as you know, red bone marrow, aka myeloid connective tissue, is the source for blood cells. Now this increased deposition of trabeculae, which occurs from the inside out, will essentially move toward the surface. In other words the trabeculae will become very dense on the inside surface of this membrane. And so think about it. If we take a hashtag like this and keep adding more and more and more eventually that open appearance will be filled in. And so what starts out as cancellous bone will eventually become very hard, very compact along

the inner surface, which provides that cortical bone at the surface. So item D, increased density of trabeculae at the surface creates that thin outer layer of what? Compact bone. And that traps, because when matrix surrounds a cell that cell is no longer mobile, and so these cells that were called osteo, B word, blasts, are now called osteocytes. The difference between an osteoblast and an osteocyte is just a matter of timing and age. To repeat, which cells produce the matrix, osteoblast. What cells maintain that matrix, osteocytes. And so we show this continuum here. Where do osteoblasts come from? They come from osteoprogenitor cells. What do osteoblasts do? Make the matrix. And once that matrix surrounds an osteoblast that osteoblast is no longer mobile and so it changes its name. It might have been an osteoblast yesterday but today it's an osteocyte, and its job is not to make matrix but maintain the matrix. So this describes the origin of bones created this way. Especially bones of the skull. But are your skull bones the same size now as they were on your birth? Did they expand, did they grow? Of course. So there has to be provisions for growth. And so it turns out that this shape, this early bone that might have indeed been your parietal bone or others of your cranium, obviously has to expand and get bigger as your brain enlarges. And so this membranous sac becomes the periosteum. But periodically, that is almost continually through childhood, there is remodeling, expansion and growth of these bones, especially in the skull. And using an analogy, if you're going to add on to your house, what do you have to do before you can begin remodeling or adding on to your home? You have to demolition. Yeah, you have to demolish. And so the cells that demolish and make possible remodeling are a whole different group of bone cells. These are called osteo what? Clast. Now an osteoclast as shown here on the screen is not related to an osteoblast, not related to an osteocyte. It's a totally separate cell. Osteoclasts contain a lot of lysosomes. And their job is not to make matrix, not to maintain matrix, but to actually dissolve matrix. In other words to break down bone. And the purpose of their function is to allow for remodeling and expansion. So to put a fine point on this, osteoblasts come from osteoprogenitor cells. Osteoblasts make the matrix. They become later osteocytes which maintain the matrix, and continuously there are the presence of osteoclasts which what? Dissolve the matrix. Now this ratio of osteoclasts to osteocytes is pretty important. In fact it's a rigorously controlled ratio. What if the number of osteocytes were to fall and therefore the ratio of osteoclasts to osteocytes was tipped? Would you like to have more of these than those? That would lead to bone destruction. In fact that is the cause of osteoporosis. So this balance between osteocytes and the dismantling function of osteoclasts, pretty important. Moving on. The second kind of bone formation, much more common, occurs mainly in the appendicular skeleton. It's called endochondral. Endo means in or within. Chondral refers to cartilage, right. So literally what does endochondral sound like? We're making bone from within an existing piece of cartilage. Now it's tempting to misunderstand that. And very often I'll read students say I get it, cartilage turns into bone. No, cartilage does not turn into bone. Cartilage is replaced by bone. Is that a big difference? So maybe in this analogy, maybe you've seen petrified wood in a forest, right. It's stone actually.

Did the wood turn into stone? No, the wood was replaced by minerals and became rock-like. So that's an important distinction. Essentially we're replacing cartilage and ossifying it from within. And the name of that process is endochondral. Essentially all bones except the skull and clavicle are formed this way. And they begin then with a piece of cartilage represented by this blue outline. A miniature version of the bone to be. And so what is this tissue? What is that bone to be? What is the histology of that? One hundred percent what? One hundred percent high in cartilage. Is there a membrane around it? Yes. That membrane is called the perichondrium. And so this perichondrium is, to begin with, invaded by BV, blood vessels. Remember, what have we said all along about cartilage? Avascular. Now it's becoming V word. So what was avascular is now becoming vascular. So let's show some vascularity which enters or essentially perforates through the perichondrium at a number of locations. Step B, invasion of a perichondrium. By what? Transforms it into the periosteum. This membrane used to be called what? That membrane used to be called the perichondrium, now it's changed its name, it's now called the periosteum representing the bone to be. Is it a bone yet? No, hardly. But notice that the vascularity occurs at two ends and typically also in the center of this long bone. So the cartilage model is invaded by blood vessels accompanied by the cells that you would expect. What cells are the four runners of osteocytes? Osteoblasts. So once again these black dots are not osteocytes, they are osteoblasts. And they essentially congregate here, here and here. What do you call a congregation of osteoblasts wherever they occur? Congregation of osteoblasts is called an ossification what? Center. And how many ossification centers do there appear to be in a bone, a long bone such as this? We have one, two, three. Actually two, these at the ends and that at the center. Those ossification centers at the end are called epiphyses and the one in the middle is called the diaphysis. I saw a hand go up. Ossification centers, this is going to sound sort of disrespectful, but ossification centers are centers for ossification. Meaning centers where ossification is occurring. So what's happening here? What's happening to this cartilage? It's not being converted to bone, it's being R word, replaced by bone. So what is an ossification center, it's just a bunch of these cells, what are they? Osteoblasts, which are producing bone matrix, hence these areas are becoming hard, right. Hence the name ossification center. These at the ends are called epiphyses. That's plural, epiphysis is singular. And of course that's a strange word or maybe deserves some definition. Physis, physis means growth, epi means upon. So literally epiphysis means growth upon, or in this case at the ends, at the ends. So this is an, this is an epiphysis and that's a epiphysis. This in the middle is called a diaphysis, a word meaning between in this case. So this is the diaphysis, this is the epiphysis. Keep in mind that these are ossification centers. But what tissue is found between the diaphysis and the epiphysis for a long time, what is this tissue, what was it to begin with and what is it still? Cartilage. What cells are there? Condroblasts. And so these areas are not hard, they are still very S O F T, right. Moving on, item D. These cells, the black dots, what were they? Osteoblasts. They're going to produce and secrete the bone which will essentially be laid down in overlapping beams or struts. So

using just a crude impression, these struts are the T word. What are they? Trabeculae, which create what type of bone in this area? Not solid but cancellous bone, good. So item D, calcification of the cartilage creates these trabecular beams which essentially generates at these sites spongy bone. And this causes the death of the cells that previously occupied this space. What were the cells that were here in the beginning? Chondroblasts, chondrocytes. Why are they killed? Well because they're simply overrun by this, by this spongy bone. Now the surface osteoblasts which continue incidentally to move toward the surface, so I'm showing these arrows. The osteoblasts are moving toward the surface. And as they reach the surface, which is capped by this membrane. What's the membrane? They basically pile up. And so what remains, what remains spongy bone in the interior becomes rather thick and hard at the surface. And what do we call that bone? We call it compact bone, which provides a layer of strength only just under this membrane. Now the breakdown of some of the matrix will occur. And now remember that breakdown is by these cells called osteoclast. And that will especially occur in this area previously described as the diaphysis. In other words this area becomes hollowed out, hollowed out. What are the cells that create that hollow space? They're the osteoclast. And what is the name of that space which is generated by their activity? The name of this empty space was called the medullary cavity. And later it will be filled with adipose, filled with what? Yellow bone marrow. Now here's the important part of all this. The diaphysis is advancing toward the E word, what? Epiphysis. And what's in the way? What tissue is being destroyed as the diaphysis marches on toward each of the two epiphysis? Cartilage. And is this cartilage being killed off as the diaphysis moves in that direction? Exactly. So what's happening to the thickness of this cartilage as the advance occurs? It gets thinner and thinner and thinner. So length wise growth occurs as the diaphysis moves in this direction. In other words this bone, which might have been this size, is now what? That size. And it's not because the bone is growing but because this piece of cartilage remember is still alive, and the name of that cartilage zone is called the epiphyseal cartilage plate. What kind of cartilage? Hyaline. An area of cartilage between the D and the E, what? Between the diaphysis and the epiphysis. So here in this enlargement we show the epiphysis, we show the diaphysis. The epiphysis is of course now largely bone. The epiphyseal cartilage plate is and remains what kind of cartilage? Hyaline cartilage. Is it growing? Yes. So in a very crude analogy let my body represent the epiphyseal cartilage plate. What's behind me there? Diaphysis. What's that way? Epiphysis. Now I'm the epiphyseal what? Cartilage plate. Am I adding more cartilage? So cartilage is being added here, but what's happening to this cartilage? It's going away. New cartilage going away. New cartilage going away. What's happening to the length of the bone? It's elongating. What's happening to my body, which of these processes alternately will prevail, which is faster, ossification or new cartilage growth? Ossification. And so eventually what happens to the epiphyseal cartilage plate? It gets thinner, thinner, and then disappears altogether. When it disappears altogether the diaphysis has now reached and fused with what? Reached and fused with the epiphysis. What happened to the cartilage? Gone. What does

that signal, what does that represent, what has now been completed? Bone growth. So elongation of the bone will stop when the cartilage disappears. In other words when the diaphysis consumes all of this cartilage and meets with and joins up with the what? Epiphysis. How long does that take? A few weeks, a few months or many years? Many years. So this process which leads ultimately to the elimination of the epiphyseal cartilage plate is ongoing from birth, but it's certainly active in childhood. And then it undergoes a big acceleration, a big acceleration at puberty. And that's because of the added effects of what hormones? What hormones hit the fan at puberty? Testosterone and of course in women the estrogen. This accelerates bone growth and also hastens the end of bone growth. So that's understandable, which gender tends to have an earlier growth spurt? I'm really asking you the question which gender enters puberty first. And therefore females tend to have that growth spurt early in middle school let's say, but they lose their epiphyseal plate sooner, right. So the latecomers, the guys, eventually will surpass that. You've seen that. I always refer to my son's junior high school prom. He's down here and his date's up here like this. But of course in high school the reverse is the effect. So to answer the question which gender enters this adolescent growth spurt first, females because of a surge of estrogen. Now don't forget that both genders have pituitary growth hormone from the get go. Growth hormone is there even at birth and continues to be an important factor. But ultimately it's the sex hormones that finish the job. And with that said, many misguided teenagers, men, I should say boys, will take testosterone. Am I making this up? And if you inject yourself with testosterone you might be muscular and get a lot of dates, but you're actually accelerating the closure of this plate, and therefore literally bringing to a halt what? And you're going to be in what, than you otherwise would, so take that. Now here are two hands and the bones are the same. But which of these are adult hands and which are not. Notice gap, gap, what is that? That's not a broken bone, although it does represent two bones later to become one, right. What is that zone there? There it is there too. It's called, here it is, epiphyseal what? Cartilage plate. Common discussion, common description the growth plate, but we call it what? Epiphyseal cartilage plate. What kind of cartilage is it? Hyaline. Is it still growing? Yes. Will it eventually disappear? Yes. And when it does, what does that mean? End of growth. Before this is done, that is before growth, longitudinal growth is finished, where is the bone the weakest? Where is the bone the weakest? Right at the epiphyseal cartilage plate. So are these areas subject to fracture? They are. In fact here's a look at a femur that's split right there. Apparently the individual died for another reason. But what used to connect this? Why did it split there? That was filled with cartilage, epiphyseal cartilage. Dorothy. That's a good question it's not, I didn't, thank you for correcting me. It doesn't actually break, but it is a weak spot and often what happens is the bony plates just basically slide away, you know, like this. More lateral. Well they are connected but, but they're weak at that spot. They're not locked together. Which is an interesting story because I reached this point one semester years ago and a student said OMG. They didn't have that expression back then but it was something like that. She said when

I was a kid I fell on my thumbs and I broke my thumb and the doctor said that I separated that bone at the growth plate, the epiphyseal cartilage plate. So what? Well she held up her thumbs, and I got a photo of it, and there it is. Now which one did she break? That one. Now it's perfectly healed, don't get me wrong, her thumbs work fine. But why do we know the injury occurred here? That stopped growing, and therefore she had thumbs which are a little bit smaller. Now that's no big deal when it's thumbs. But what if it's legs, then it's a big deal. So let's move on. Longitudinal growth stops when the diaphysis meets with and fuses with the epiphysis. And for men that tends to be later because they start puberty later and finish later as a rule. Wrapping this up quick. There are marrow cavities. Yep, we've got to do this. We'll take three more minutes of your time. Red marrow is found in the spaces between the trabeculae. Red marrow is what color? Good guess. And what is red marrow made of? It's made of myeloid connective tissue. Again this is a very common misstatement. Some people think that bone makes blood. Bone does not make blood. What makes blood is myeloid connective tissue, just happens to be inside the bone. And by blood I mean erythrocytes and leukocytes. You've heard of bone marrow transplants, right. So a needle is put through a bone, which is painful, and they suck out this material which is pasty and semi-liquid. Why would somebody need a bone marrow transplant? It's not for the red cells, it's for the white cells. If you need red cells just get a transfusion. Now the other tissue which is residing in bone, but certainly not in any way bone at all, is yellow marrow. Guess what color? Good. And yellow marrow is 100% adipose which has no supreme function. I mean after all what is the function of adipose wherever it is, stores calories. Again we want to make this point, is there yellow marrow in the medullary canals of lone bones? Yes. Is that the property of that bone? No. And so if you are starving, will that marrow disappear, will the yellow marrow be converted to energy for the sake of the whole body? Absolutely. So it's not really the property of the bone. But final remark, can you break a bone? And if you break a femur what marrow is going to spill out from the interior from the medullary canal? And that is F A T, right. So now you've got fat in that wound which is bleeding profusely, right. Can some of that fat get into your blood? And that can actually go to your brain and cause death. It's called a fat embolism, a fat embolism. So yellow marrow has that potential complication. Thank you for that. Have a great day. Isn't that a great picture? There it is.