All right, guys. So today's lecture is going to be on chapter 8, which is the neoplastic disease, and let's see, the first thing is the terminology description of some of these tumors, the way they're named, it's difficult to follow. It's not a great nomenclature. They use the word neo, to mean new, plasm is growth. A cancer is defined as any type of malignant growth. The problem with that definition is we need to define malignancy. All right? Malignancy refers to a cancer or growth of cells that invades the surrounding tissue. Okay? So cancer, when we talk about cancer in general, we're going to talk about malignant tumors, all right, tumors that invade the surrounding tissue. Benign tumors, not malignant, benign, they grow by expansion. They continue to grow and grow. They'll get bigger, and they put pressure on the surrounding tissue and may compress it. But it doesn't invade. By invade, what I mean is it sends out roots, sends out tendrils into the surrounding tissue. Okay? So that's the very definition of cancer. There's unrestrained growth and spread. The cells do not respond to the normal control mechanisms that regulate cell growth and differentiation. Okay? The cells just keep on proliferating. They crowd each other. A lot of cells have, within them, a sense of neighborhood, in that they do not increase beyond a certain point. They don't grow so many that they put pressure on surrounding cells. With cancer cells, that's not true. The other thing on cancer cells is they really serve no useful purpose. The reason for that is the cancer cells, because they proliferate at an unusual rate, they have lost some of the normal cellular characteristics. So we say they've lost differentiation. They are not well differentiated. Okay? Some of the functions, for example, in the pulmonary tree, we have the pulmonary tree is lined by cells that have cilia on the top of them. Those are the soft little hairs, hair-like projections. They move mucus and debris up out of the lungs. All right? Cancer of these cells, these cells would then maybe still be in the same location, but they would lose the cilia. They would lose their characteristic appearance, and we call that loss of differentiation. If they lose all their characteristics, they become what we call anaplastic, all right? Anaplastic means you can't tell what type of tissue they came from because these cells have lost all their characteristics. Okay?
Now, benign refers to a growth rate, okay, that is slow. They grow by expansion, they tend to remain localized and they're well differentiated. Malignant, they grow rapid, all right, and they tend to grow by infiltrating, as I mentioned before, and parts of their tumor can break off and then can go into the bloodstream or the lymphatic system and spread to distant sites. Okay? And the cells are going to be poorly differentiated.

Now, they use the word -- the suffix oma to name a tumor, and the prefix is going to be the type of tissue of origin. For example, adenoma, all right, that means tumor, and adeno refers to glandular epithelium. Angio prefers to blood vessels, Chondroma refers to cartilage. All right? Polyps or papilloma refers to benign tumor or stalk growing up from an epithelial surface.

Malignant tumors can start from a single cell, okay? And what happens, we postulate, is their genome, their genetic information is damaged, all right, and we'll go into ways that it can be damaged and what happens. And as a result they develop a clone of identical cells, and if unchecked they eventually develop into a distinct tumor. They have behavior that's different from that of the normal cells they're derived from, and they do not respond to normal growth regulatory signals. They just keep on proliferating. Okay?

Now, they can secrete growth factors. These growth factors will stimulate their own growth, all right, allows these tumors to keep on growing at the expense of normal cells. They can produce enzymes that break down normal cells and tissue barriers. Therefore, they're able to invade surrounding tissues, they're able to invade lymphatic channels, they can spread throughout the body. Tumor cells may not wear out, okay, like normal cells. They become almost immortal in that they don't die and they keep on proliferating.

All right. So tumors can be classified several ways. One way is the term sarcoma. Sarcoma rises, it's a tumor that arises from connective tissue. All right, such as fat, bone, cartilage and muscle.

Now, what sarcoma have we already talked about in previous lectures? Don't let me down, guys. What's -- what?

>> STUDENT: [Inaudible].
>> PROFESSOR: What is it?
>> STUDENT: [Inaudible].
>> PROFESSOR: Kaposi's sarcoma. Right. Absolutely. Right. What is that? Well, it's a tumor, small nodules of the connective tissue, all right? And what's the causative agent with Kaposi's sarcoma?

>> STUDENT: [Inaudible].
>> PROFESSOR: Good. HIV. Absolutely. Guaranteed it will
be on the final. Okay? HIV, kaposi's sarcoma.
All right. So sarcomas are cancers that arise from
connective tissue. The connective tissues include fat,
bone, cartilage and muscle. Okay? Leukemia are neoplasms
of the blood cells. What blood cells? Really not the red
blood cells. It's the white blood cells. Okay? And
usually don't form solid tumors. Instead, they proliferate,
and then they -- within the bone marrow, and then they spill
out of the bone marrow into the bloodstream, and they also
will crowd out normal blood-forming cells. All right? So
the neoplastic cells spill over into the bloodstream, so you
have large numbers of abnormal cells. All right?
Tumors are named and classified according to their cells and
tissue of origin. All right. Tumor nomenclature is not
completely uniform, but you can generalize. All right?
You do have exceptions. Lymphoid tumors. We'll get into
that. The skin tumors that arise from pigment-producing
cells within the epidermis are exceptions, for example. I
told you the oma means a benign tumor. Melanoma is the
cancer of the melanocytes, which is a very bad tumor, and
that can have a very bad prognosis, because a melanoma can
be very bad in appearance, but it's already broken off and
metastasized to other sites. Okay?
Let's see, there are certain tumors -- types of tumors
composed of primitive cells, basic cells in little kids,
even children, and these are blastomas, and the prefix is
going to be the tumor of origin. All right. So the
blastomas are tumors of primitive cells seen in young
children.
So okay, here's a basically a little summary of the
terminology. Polyp or papilloma refers to a benign tumor
that projects from the surface, okay, an epithelium, polyps
are common in the colon. They project from the surface of
the lining of the colon. Oma we tend to say is a benign
tumor. Carcinoma is a malignant tumor rising from
epithelium. Remember, epithelium can provide covering, can
also provide -- also is involved in glands. Okay? The
glandular secretions come from epithelium. Okay? So the
epithelial tumors that -- tumors that arise from the
epithelium are called carcinomas, but not if they arise from
endothelial or mesothelial cells. Endothelial cells are the
flat squamous cells that line capillaries, okay, in the
vascular tree.
Sarcoma, malignant tumor of any primary tissue other than
the surface glandular parenchyma, epithelium. Neoplasm of
blood cells refers to leukemia.
Here's your prefixes. Adeno refers to gland. All right?
One of the tumors of the breast is an adenofibroma. Angio
refers to blood vessels. Chondro refers to cartilage.
Fibro is fibrous tissue. Hemangio refers to blood vessels
and hemangioma is -- maybe you've seen that. Hemangioma is there's different terms for it, but it's a nodule growth of the skin made up of a lot of capillaries, and if it bleeds, it can bleed a lot, okay, that's a hemangioma. Lymphangioma is a collection of lymph vessels. Lipoma is a fat tumor. Myo refers to muscle, neuro, of course, is nerve and osteo is bone.

Now, lymphoma, all right, lymphoma is a neoplasm of the lymphoid tissue, and usually malignant. Even though earlier in the lecture I mentioned the word suffix tends to denote a benign tumor.

Two classifications for lymphoma is Hodgkin's and non-Hodgkin's. Okay?

Now, they can infiltrate lymph nodes. The prognosis can vary. Low-grade lymphomas have a favorable prognosis. Intermediate grades may not do as well, and they can basically the types of cells that give rise to these tumors are the T-cells, the B-cells, the NK cells, all lymphocytes. Hodgkin's. Hodgkin's is associated with a histological appearance of a Reed-Sternberg cell. It's a large cell with double nuclei. Okay? Mirror images of the nuclei, and that is what we call pathognomonic for Hodgkin's. Pathognomonic means when you see it, there's only one diagnosis. Pathognomonic is P-a-t-h-o-g-n-o-m-o-n-i-c.

All right. And that's a Reed-Sternberg cell is pathognomonic for Hodgkin's. Not Hodgkin's lymphoma. All the lymphomas that aren't Hodgkin's are called non-Hodgkin's lymphoma.

Skin tumors. All right. The basic skin cells are keratinocytes. They produce keratin. They are squamous cells in the melanocytes. Melanocytes produce the pigment. Benign tumors will be the nevus or birthmark. They're common pigmented cells derived from melanin producing cells and then you got the malignant melanoma, all right. That's a malignant tumor of the melanocytes. The keratinocytes, as I mentioned, are the squamous cells. You can get keratoses. Maybe you've seen them, maybe you know somebody who has them. They can be precursors to basal cell carcinoma, squamous cell carcinoma. They're treated by freezing. They're going to be on the sun exposed area of the skin and they're going to be small and red. A little larger than red dots. They can have a little bit of a peeling type of surface. All right, and the concern is that if they're left alone, they may go into develop basal cell carcinoma and squamous cell carcinoma.

Basal cell carcinoma, the good thing about that is it rarely metastasizes. It's really slow-growing. The squamous cell is a little more aggressive than the basal cell, and it can metastasize. Both types typically are cured by surgical excision.
All right. Teratoma. The term teratoma refers to a tumor that contains different cell types. I know this sounds confusing. The most common type of teratoma is the one found in the reproductive area, basically of the ovary. Teratoma of the ovary is most the time benign, and has several different skin or tissue types. By tissue types, we're talking it can have hair. It can have epithelium such as skin, and contain sweat glands, sebaceous glands. You can find bone. I had one patient, very interesting, who was treated for hyperthyroidism by an endocrinologist, and her hyperthyroidism did not respond to medication. So he was a very good endocrinologist, and in doing her workup, he also got an ultrasound of the pelvis. The ultrasound of the pelvis showed that she had a cyst on her ovary consistent with a teratoma. He sent her to me for a consult. I agreed and credit that it was a teratoma. Another name for a teratoma is a dermoid. Maybe you've heard that term before. It needed to be removed.

So we did remove the teratoma and within the teratoma was functioning thyroid tissue. So once we took out that teratoma, then her thyroid problems were cured. She was no longer hyperthyroid.

So this is a tumor, all right, teratoma, and it arises from cells that can differentiate and debone muscle glands, epithelium, brain, tissue and hair.

When you think of it, the egg, okay, from the ovary, is fertilized by sperm and it gives rise to an embryo and then a human being, right? So from that one cell, we get all the different types of cells in our body. So a teratoma is a tumor that has several different types of tissue. It has mixed components. They tend to be benign, okay, but they can be malignant.

All right. So primitive cell tumors. The word for primitive cell tumors, the suffix, is going to be a blast or blastoma. Okay?

So we use the word blast to be immature or young. Blastoma is going to be an immature tumor. And the prefix is the tissue of origin. All right? And so, like a retinoblastoma is a tumor of the eye within the eye. The liver is a renal blastoma, also known as a Whelms tumor. And these tumors are aggressive. They don't have a good prognosis. They need to be treated aggressively to have a good prognosis.

Here's a picture of the teratoma. You see the hair. The oily secretions on the inside of the cyst, and let's see here. Yep, here you see where this arrow is, this is -- this nodule right here may be bone. I don't know what they found here, but may be bone. This is the hair within the cyst. This is the edge of the cyst. This is the inside. It always usually has a very yellowy appearance, due to the sebaceous secretions, and this is the outside of the cyst.
All right. So leukemias. Okay. Leukemias are a neoplasm of the hematopoietic tissue. Leukemic cells basically usually infiltrate the bone marrow and lymphoid tissues and spill over into the bloodstream. The cells can be mostly mature or extremely primitive. The mature leukemias we call chronic. The immature cells, usually very aggressive, we call them acute. All right?

So with leukemia, you have over-production of white blood cells that spills over into the peripheral blood so these people have a very high white blood count. Aleukemic leukemia, not that common, is a condition in which white cells don't spill into the bloodstream, but they're confined within the bone marrow.

Leukemias are classified by the white blood cell type. What type of white blood cells are there? Well, we talked about the lymphocyte all right, so there's acute and chronic lymphocytic leukemia. All right? We've talked about the polys. Polys are granular cells. They have granules in the cytoplasm, and there are two other cell types that have granules in the cytoplasm. One are eosinophils. One are basophils. So they form under the category of granulocytic leukemia. So you have an acute and a chronic granulocytic leukemia. The last type is acute and chronic monocytic leukemia. Okay? You have monocytes, you have lymphocytes and then you have granulocytes, and any of them can be acute or chronic.

All right. Here we go. Here is a granulocyte. It's probably a basophil. But it's a granulocyte here. See all the granules in the cytoplasm. My question to you guys, what type of cell is this?

>> STUDENT: [Inaudible].

>> PROFESSOR: What type -- who said poly? Thank you. It is a poly. Why is it called a poly? Poly stands for polymorphonuclear leukocytes. And so when you look at this cell, here's the nucleus, here, here, here and here, so it looks like it's got a nucleus here and a nucleus here and a nucleus there so it has several different shapes. But what you see is basically there is the nucleus here, and then you have this attenuated band but then it connects. You glob a glob of nucleus here and then an attenuated band, so it gives the appearance of more than one nucleus, but they really are all the same. Same thing over here. Here's a poly. Okay? See, and here's that little attenuated band that connects the nucleus, so it's not several nucleus -- or nuclei in one cell.

All right. So anyway, the manifestations are caused by impairment of the bone marrow function. The leukemic cells crowd out normal producing cells. Now, what are the normal cells in the bone marrow besides producing white blood
cells? We also have the cells that produce red blood cells. And also, we have cells that produce platelets. So people have leukemia. They may be anemic, right, because they're crowding out normal red blood cell producing cells. They also may have low platelets. We call that when they have low platelets, thrombocytopenia. Penia means low, low count. A thrombocyte is a platelet. Thrombocytopenia is low platelets. So these people present with infections, and the reason why they get an infection is because they have a low number of healthy white cells. They have a high number of unhealthy, abnormal white cells. Little children, when they develop leukemia, how do they present? They very often present with recurring sore throats. Okay? Why? Because of the lowered white count.

All right. So, leukemia then, all right, can spread to other organs. The spleen, the liver, other lymph nodes. Megaly refers -- that suffix refers to enlargement, enlarge. So splenomegaly is going to be enlarged spleen, hepatomegaly is enlarged liver.

In chronic leukemia, evolution of the disease proceeds at a relatively slow rate, and the acute leukemias, they're much more aggressive. All right?

Now, the term myelodysplasia or pre leukemia refers to patients who may be anemic, they may have low white blood cell count, they may have some degree of thrombocytopenia, okay? They don't have leukemia, but when they present with these three factors, we wonder if they are pre-leukemic, if they're going to develop it. When I was in practice, I did have this one patient. She was 76 years old, long-time patient of mine for about ten years, and the last three years I was in practice, she was diagnosed with myelodysplasia. No signs really of leukemia. She did have a low blood count, you know, just borderline low. She wasn't really symptomatic, but the concern was that she might develop the leukemia.

All right. So we do have that syndrome also that exists. Multiple myeloma, very interesting. All right? Multiple myeloma is a neoplasm and it develops from plasm cells. What are plasma cells? Guys, what are plasma cells?

>> STUDENT: [Inaudible].

>> PROFESSOR: What?

>> STUDENT: [Inaudible].

>> PROFESSOR: Right. Great. They form antibodies. Okay? So the antibody-forming cells, what type of cells are they? When they -- before they -- once they're sensitized, they produce antibodies and they are called plasma cells. What are they before they're sensitized? What do we call them? B lymphocytes. Okay? So multiple myeloma, B lymphocytes, cancer cells. They lose some of their regulatory ability,
therefore, what do they produce? They produce a lot of antibody, unhealthy antibodies, not normal. But they produce it. It goes into the bloodstream. Then, high levels of that in the bloodstream, it spills over into the urine. So these people with multiple myeloma have antibody chains in the urine. When you have antibody chains in the urine, we call that Bence-Jones protein. Bence-Jones protein, and we say it's pathognomonic for multiple myeloma. If someone has Bence-Jones protein in the urine, there's nothing else that can cause multiple myeloma. Okay? So it resembles leukemia, but the cell proliferation is confined to the bone marrow and usually doesn't cause organ infiltration.

The number of plasma cells kind of stays within the bone, doesn't spill over into the circulation. But what it does is it invades the bone, it can weaken the bone so these people get spontaneous fractures, pain and disability. Here, you see a skull. In this, you'll see some of the lesions here. It's not that dramatic. But you can see right here these are what we call, like, punched-out lesions, okay, of the skull. This is all -- this is consistent with multiple myeloma. Okay? You got a big punched-out lesion here. All right.

Tumor blood supply. What happens is the tumors run the risk of outgrowing blood supply. They divide blood supply from the surrounding tissues. The malignant tumors frequently they can produce a protein that encourages new blood vessels to proliferate in the adjacent normal tissues and as a result supply them the demands of the growing tumor. We call that the angiogenesis factor. Malignant tumors can outgrow their blood supply. All right. If they outgrow it, then that area that outgrows it, necrosis, okay, sometimes they have oozing from the area of necrosis. A little bit of bleeding from the area of necrosis.

All right. Let's see. Often in small blood vessels with necrosis are exposed in the ulcerated base of the tumor, and therefore they can ooze blood. For example, cancer of the colon. The colon basically has, you know, the ascending portion, which is the right part of the colon. Goes up the right side of the abdomen, goes across, we call the transverse colon and then it goes down the descending portion. Cancer of the colon, if it's in the ascending portion, the right portion of the colon, what happens is they tend to present with anemia. Why? Because the cancer outgrows the blood supply and they have a little bit of oozing from the necrotic site. So as a result, they lose blood. Colon cancer of the left colon, the descending colon, tends to present with obstruction.

All right. Now, another type of cancer is in situ carcinoma. All right. Remember, carcinoma refers to
epithelial tissue, and we have the condition in situ. What that is is full thickness cancer, full thickness of the epithelial layer, all abnormal cells. However, it does not invade beyond the basement membrane. Okay? It does not invade beyond the basement membrane, so the epithelium is abnormal totally but it has not invaded. Most common site for that is of the cervix and then also the breast. All right. The breast tends to be ductal carcinoma in situ. Someone tells you they've had cancer of the cervix or cancer removed from their cervix, what they're probably talking about is in situ carcinoma. In situ carcinoma can last for years before it becomes invasive. If you have true cancer of the cervix, the only treatment for that is radiation and removal of the cervix and the uterus and the tubes and ovaries. Okay?

So someone said, you know, someone tells you they had cancer removed from their cervix, they're talking about in situ. Which they just remove the section that has the cancer. The in situ cancer, and these type of patients do really well. They're really not hardly any risks for a recurrence. All right. So what are some pre cancerous conditions? I mentioned the actinic keratoses briefly. All right. In reference to basal cell, squamous cell carcinoma. Small, crusty, scaly patches on sun-exposed skin. Very often they're treated with freezing and they go away. Lentigo maligna refers to melanin-producing cells. All right? And it's like freckle proliferation. Leukoplakia is thickened white epithelium, usually develops from irritation and the irritation can be from chemicals such as tobacco, tobacco tars, chewing tobacco or snuff. All right. All within the inside cheek. Okay? For -- you can get leukoplakia in other areas, too, but when I'm using the example of the mucus membranes within the mouth. Okay?

Leukoplakia can give rise to squamous cell carcinomas of the oral cavity. Okay. What are some of the etiologic factors for neoplastic disease? You can have viruss that can cause cancer. We've talked about Kaposi's sarcoma. This is the mothers. Gene chromosomal abnormality. Failure of the immunologic defense mechanisms. Ha ready. Okay. All of these, these four factors can cause cancer. Now, for viruses, what are some of the viruses that can cause cancers in humans? All right. Leukemia lymphoma may be associated with AIDS virus, okay, Kaposi's sarcoma, as we mentioned before. AIDS virus and associated with the human herpes virus AIDS. What about hepatitis B? All right. The concern about hepatitis B is if you get hepatitis B, 10% of people who get it become carriers. They're chronic active carriers of the virus. About 10% of these people go on to get cancer of the liver. Hepatitis C, all right, much
higher incidence of liver cancer in people with hepatitis C. Once again, that's a virus. Epstein-Barr virus, causes mono, has also been associated with nasopharyngeal virus, cancer of the nose. All right. Next one, gene and chromosomal abnormalities. All right? There's three large groups of genes that play an important role in regulating cell functions. Mutations in these genes can be associated with tumor formation. We're talking about the proto-oncogenes, tumor suppressor genes and the DNA repair genes.

All right. These tend to occur in pairs. The proto-oncogene is a gene that encourages cell growth, healthy cell growth. Okay? The concern comes from the fact that it can mutate, or a chromosome can break and it can be translocated. If it mutates, then what happens is it stimulates or causes excessive growth. When you have excessive growth, not regulated growth, but excessive growth, then you run the risk of that growth causing cancer, becoming cancerous. Okay?

So as a result, the proto-oncogene, if it mutates, we say it is oncogenic, it is cancer-causing. The tumor suppressor genes are genes that occur in pairs, all right, and they suppress tumor development. If one of them mutates, as long as you still have the other gene, you have protection. All right. Same thing with the DNA repair genes. If one mutates, as long as you have the other one, you still have protection and repair ability of DNA. All right. So these slides explain it. The proto-oncogenes are normal growth genes in the human. All right. If it becomes oncogenic through a mutation or translocation, then it's an abnormally functioning gene that stimulates cell growth excessively, leading to unrestricted cell proliferation.

Tumor suppressor genes, these normally suppress, all right, the cell proliferation, loss of function by a mutation can lead to unrestrained cell growth. Because they exist in pairs, you have to lose both of the suppressor genes for a cell to malfunction. DNA repair genes, same thing. They regulate the process that monitor and repair any errors in DNA duplication. That can occur from radiation, chemicals or other environmental agents. As long as you have one of them, you still will have the protection.

Failure of immunologic defenses, cancers may arise from multiple genetic insults to the genome, all right? Our genetic information. And as a result, it might activate an oncogene, and loss of the tumor suppressor genes. Followed by additional random genetic changes, may develop into a tumor.

What happens is the mutant cells produce cell proteins that
are not present in the normal cell. Therefore, our immune system should pick these proteins up as foreign, okay, but they don't. All right? If these abnormal proteins are recognized as abnormal by the immune system or destroyed, that's great. However, the immune system maybe does not pick them up as abnormal cells. Okay? Now, heredity, very interesting. Certain cancers can run in families. Do you know of any cancers that can run in families?

>> STUDENT: Breast cancer.

>> PROFESSOR: Breast cancer, right. The statistics on breast cancer, we will discuss it more in greater detail in another chapter, but for this lecture, we're going to say statistics on breast cancer, one out of nine women. Very scary statistic, because there's more than nine women in this class. One out of nine women, in their lifetime, will get cancer of the breast. Okay? It can run in families. When it runs in families, what's the real concern? The real concern is the first degree relative. Okay? So it's the mother. All right? It's not the grandmother. The grandmother is the second degree relative. First degree relative would be the mother or the sister. A sister would be a second degree relative. What's the problem with heredity? The problem with heredity breast cancer, one of them is the gene BRCA 1 and the gene BRCA 2. BRCA 1 and 2. >> STUDENT: We're doing a study with that in our research, and I know for the grandmother or the aunt or any other family, that as long as if they're -- they had breast cancer before they were 50, then that's when we were taking it into consideration.

>> PROFESSOR: So before they were 50. So basically that's like pre menopausal, as opposed to menopausal. Right. So the BRCA-1 gene and BRCA-2 gene. If women have this, it's a mutation, all right, it can be passed on. If they have it, the quoted statistic is 80% chance of getting breast cancer. And they now have a blood test for the BRCA gene. So if you carry the BRCA gene, okay, the mutation, BRCA-1 or BRCA-2, those two genes are located in different chromosomes. Risk of breast cancer is 80%. All right? With the BRCA-1 mutation, the risk of ovarian cancer is 20 to 40%. Okay? With the BRCA-2, risk of ovarian cancer is 10 to 20%. So the BRCA mutation increases the risk of breast cancer and also ovarian cancer. All right. BRCA-1, 80% risk of breast cancer, and ovarian cancer is 20 to 40%. BRCA-2 is 80% chance of breast cancer, and 10 to 20% chance of ovarian cancer. Okay. All right. Inheritance of certain genetic. Okay. The Philadelphia chromosome, what that is is a translocated chromosome, and it was discovered in the city of Philadelphia. That's why it's called the Philadelphia chromosome, and it's associated with chronic granulocytic
leukemia. Okay?
Now, so diagnosis of tumors. We'll go over -- there's early warning signals in another chapter. There's laboratory procedures. What are some of the laboratory tests you can have? A colonoscopy, Pap smear for women, examination of the esophagus and stomach.
Abnormal slides, okay, basically slides refers to getting cells, obtaining cells. A Pap smear is where a cotton tip applicator is rubbed on the cervix, and the reason for that is the cells, either shed cells or squamous cells then on the cotton tip applicator and then it can be rubbed on a glass slide. Glass slide gets sprayed so the cells dry flat. It then gets sent to the lab and it's read by a cytologist, or cotton tip applicator gets put in a small vial with liquid in it. That goes to the lab. They then spin it down, the liquid down, get the cells, stain them, and the cytologist looks at them with the idea that all the cells are going to be normal, not that they're going to be abnormal. We use the term dysplasia for abnormal cells. So that's the hope. That cytologic diagnosis, smears, also and aspirations. One of the best ways to deal with any type of palpable growth or lump is a fine needle aspiration. Fine needle aspiration, we'll talk more about it with breast cancer, but a fine needle aspiration is where it's a very thin needle, it goes into the lump, they attach a syringe, they back draw on the syringe, get a look at some of the cells that are in the lump. They then take it out, and then they put the cells on a glass slide, it goes to the pathologist, he looks at it and he says all the tissues are normal, or all the cells are not normal, I need a bigger specimen, or there's cancer. All right. One of the best ways of dealing with any type of lump. You also have frozen section slides. That's done usually in the operating room or in the hospital. Specimen is sent down for a diagnosis. They freeze it. They can stain it, and then by freezing it, they can slice it very thinly and examine it with the microscope.
Okay. This is normal -- anybody want to -- okay. Forget it. It says it on the top. It's stratified squamous epithelium. Okay? Stratified squamous. Here's the top of the epithelium. This is the bottom. Right here is the germinal layer of cells. The germinal cells then keep producing cells and cells and as they keep producing cells -- wrong slide -- they keep going up and up. As these cells move up, they change in characteristic. Number one, notice how pale the nucleus becomes? Very pale staining. Why? It's no longer active. A lot of these cells are flattened. They lose their nucleus, okay? This is the normal progression for stratified squamous epithelium. Our skin is stratified squamous.
Right here, this area right here, is the basement membrane. Below this is connective tissue. So as long as the epithelial abnormalities stay above the epithelium, we can say maybe they have cancer in situ, but it's not invasive because it didn't invade the basement membrane.

The next slide, what do we have here? First of all, look at the nuclei. The nuclei are dark staining. Do they stop at the -- along the germinal layer, the bottom layer? No. These nuclei up at the top are very big, very active. That's cancer. This is full thickness, abnormal epithelium. This is carcinoma in situ. The good thing is that these cells have not invaded beyond the epithelial basement membrane. These are connective cell tissues here. The basement membrane is still intact.

All right. So diagnosis of tumors. Very interesting. There are certain tumors that produce a protein, all right? One protein is CEA, stands for carcinoembryonic antigen. That's produced by the colon cancers. Another tumor that is the ovarian -- in ovarian cancer can produce a protein called CA-125. The significance of these is that if they're present, if, they can be used to follow the patient's response to chemotherapy or radiation.

For example, someone comes in and they're diagnosed with a colon cancer, and they draw a blood test and the patient has a very elevated CEA level. The person then undergoes surgery. They remove the colon cancer. The CEA level goes to zero. Okay? They then get serial CEA levels every month, and after six months they start to go up. What's that mean? There's been a recurrence of the colon cancer.

Now, CEA is not a good blood test. You can't use it as a screening test. Why? Because not all colon cancers produce it. But for the ones where it does, it doesn't carry a prognostic factor but it makes it easier for follow-up. Same thing with CA-125. Some ovarian cancers produce it, some don't.

Now, PSA, anybody know what PSA is a screen for? Prostate cancer. Okay? That is -- that goes up when there's prostate cancer. Okay?

Now, let's see here. So the CEA can be produced by tumors of the GI tract. Usually the most common one is going to be the colon.

Alpha fetoprotein, I'm not going to really talk about that. That can be associated with carcinoma of the liver. Carcinoma of the liver is not that common. Colon cancer is. Acid phosphatase associated with prostate cancer. So here's a diagram trying to explain CEA. But basically you have embryonic cells and then mature, they deliver, and then later on, with cancer invasive -- invasion, transformation of the cancerous cells that become more immature. They lose their adult characteristics, they become neoplastic.
All right. Here's an example of CEA used to monitor the response. So remember, these tumor-associated antigens, they're great markers. They can be used for response to measure the response to therapy. They cannot be used for diagnosis. Okay? They cannot be used for diagnosis.

So a treatment of tumors. You can have surgery, radiation therapy, hormones, we'll talk about hormones and cancer another time. Anticancer drugs, adjuvant chemotherapy, immunotherapy, non-specific. We mentioned about these in a previous lecture on the cell mediated immunity, cytokines, interleukin, interferon. All of these can be used. Chemotherapy eliminates the cells that what? That divide frequently. Has to be fast-growing cells. If these cells grow fast, then what happens is they take up the chemotherapeutic agents and they end up dying. The problem is that there are other cells in the body, okay, other areas in the body that are fast-growing cells. Can anybody think of any?

>> STUDENT: Hair.

>> PROFESSOR: Hair is one. Right. Certain chemotherapeutic agents, people lose their hair. All right. What else? Another one is the bone marrow. Okay? We're always producing red blood cells. White blood cells and platelets, and very often, before they do a test, okay, to monitor -- to administer chemotherapy, they will measure the white blood cell count, red blood cell count and platelets. If platelets are down, or the white blood cell count is down, or the red blood cell count is down, they may not give them the chemotherapy. All right? Because what happens is these fast-growing cells divide frequently like blood cells. They also take up the chemotherapeutic agency, and it suppresses them and kills them. Okay?

All right. So normal cells can recover quickly. Side effects disappear gradually. So these are some of the symptoms of chemotherapy, anemia, with anemia you get weakness, tiredness, fatigue. You can get depression. Hair loss. Loss of appetite. All right. So the survival rates in cancer, anywhere from 4 to 95%. Best cancer to have, thyroid cancer. All right. We measure the effectiveness of treatment. One of the ways we measure is five-year survival rate. The five-year survival rate for anybody diagnosed with thyroid cancer is 95%. 95% of those people who had it five years ago are still alive today and doing well. Pancreatic cancer has just about the worst, okay? Usually people die within a year after they've been diagnosed with cancer of the pancreas. Can you guys think of anybody that was diagnosed with cancer of the pancreas in the past couple of years?
>> STUDENT:  Patrick Swayze.
All right. So cancer second to heart disease as the most common cause of death. One in every four people will develop cancer.
Lung cancer, most common in men. Breast cancer most common in women. Thank God our statistics on breast cancer is so good. We really have great results in dealing with breast cancer.
Chances for survival, significantly reduced once the tumors spread to regional lymph nodes or distant sites. When the tumor spreads like that, and we use the term metastasizes, there's metastatic lesions in other areas of the body, so it's hard to have a good prognosis with that.
The five-year survival rate does not always indicate a cure. Certain tumors can come back after five years. Ovarian and breast are two of the ones that can happen.
Recurrence rate can be failure of the body's defense mechanisms, re-activation of the tumor.