# Digest PM 4-12-22

Fri, 4/22 4:22PM • 1:15:37

**SUMMARY KEYWORDS**

bolus, small intestine, peristalsis, stomach, mixing, esophagus, sphincter, large intestine, hormones, lower esophageal sphincter, motility, swallowing, digestive system, called, happen, muscle, acid, draw, occur, talk

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Okay, folks, homestretch last two weeks of lecture. So digestive system, what we see up on the screen is pretty much everything we're going to be talking about in the next two and a half lectures. So what I did yesterday is on my whiteboard, my office, I drew this, this big, I didn't want to draw it right now, because it would take forever. So we need the time. So what this picture is, it's all those words right there. It's all five processes of the digestive system. And so I'm going to refer back to this picture a number of times as we go through the details of all these processes that we need to discuss. Before we talk about these five processes, we are first going to discuss how they are all regulated, and how do we regulate these five processes. Nervous System, of course, and hormones are going to be involved in so paracrine is a term that you should be familiar with, because we talked about it at the beginning of the semester, when we do in the endocrine system in prostaglandins, I remind you. So what we're going to do first is discuss how the nervous system is going to regulate all these processes that we see here up on the page. And so let's draw this right now. Now, last semester, when we went over the autonomic nervous system, and of course, that's the division we're talking about, the autonomic not the somatic, of course, is parasympathetic and sympathetic, right? Remember that happened last semester, we went over in a lot of detail, there was a third division that we didn't talk about. And that was the enteric nervous system. That's the nervous system of the digestive system and has its own nervous system. And it's fast as as many neurons as a spinal cord does. So what I'm going to do here is I'm going to write down digestive system. And when I say system, what that's going to include digestive tract from mouth all the way down to the anus, along with accessory organs, which would include things like the liver, the gallbladder, the pancreas, those kinds of things. So everything is what the digestive system is going to be. And we'll just do that. And then I'm going to put here at the top, because it should be at the top, the enteric nervous system, near enteric nervous system is embedded within the digestive system itself, and it has neurons, and those neurons have axons that are going to synapse with the digestive system and influence it. Well, how is it going to influence it? Well, there will be stimulatory effects. And there will be inhibitory effects is going to be dictated by what needs to be done at the time, so that the digestive system works properly. Now the parasympathetic and the sympathetic are still included in this story, because it's a set that's the autonomic that's controlling things. So what I'll do over here is I'll put sympathetic now the sympathetic can have direct input into the digestive system. And it can also have input to the enteric. So not that it can it does in both cases, and what kind of input is it going to have inhibitory input? And this is something that we learned last semester, the sympathetic nervous system inhibits the digestive system, we learned that when we talked about the autonomic. And then of course,

we had the parasympathetic, remind me the sympathetic is called what the fight or flight, right? What do we call the parasympathetic, rest and digest. So let's, let's see why the parasympathetic has a huge influence on the digestive system. And it's a stimulatory influence, the sympathetic doesn't really play a very big role. Parasympathetic plays a very big role. And the enteric does play a huge role. And it's a direct effect. And it's an indirect effect when it comes to sympathetic and parasympathetic, because again, they can work through the enteric nervous system. Now, what else is going to affect the digestive system hormones, talked about hormones at the beginning of the semester, we didn't talk about all of them, we talked about maybe 20% of the hormones in your body, you're going to have to learn for more. This is a blood vessel. Of course, it is because well, blood vessels are what carry hormones to their targets. And the targets are going to be the cells, tissues and organs of the digestive system. So there are going to be four more hormones that you have to be aware of. And I'll show you where they are in just a second. So you know what I'll do right now. So this picture that we're drawing is going to include these words that we see here on the page. And then it's also going to include these four hormones. So there's two of them. And there's another two, so you're going to be responsible for those four additional hormones that are unique, because we haven't learned about him yet. Although one of them was actually in the endocrine chapter. I just didn't highlight it. And then these two paragraphs are going to affect something very specific. It's going to be in the stomach, gastric acid. We'll talk about it later. And so this will also be included in this picture. And so I'm just going to write the word hormones here. But it's those four hormones that I just showed you a second ago. So hormones released into the blood. What kind of an effect are they going to have on the digestive system? It depends on the hormone. Some of them will stimulate and some of them will inhibit

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When it comes to our paragraphs, the same story is going to hold true a paracrine, if you recall, is some molecule produced by a cell that is then released onto a cell that is adjacent to it. Remember that prostaglandins remember paracrine activity. We also did autoCrat activity that day. Well, we have other paragraphs, prostaglandins, actuate paracrine, in the body, there are many others, there's going to be two that you have to know and we'll get to it in the next lecture. They're not going to be dumped in the blood, of course, there's, they're released onto an adjacent cell. So they're going to have some effect, what kind of an effect depends on the paracrine. Some will stimulate others will inhibit. And so this is what's going to shape what the digestive system does. So these things, once again, are regulating all these five things that you see on your page. And the picture that I drew that again, I'm going to refer to a number of times. So now what, let's start talking about those five, no, you know what can't do that yet. I'm going to talk about one hormone, these other hormones that I have listed here, they're going to be scattered throughout the chapter. The reason that I put the hormones here, and what their functions are, what stimulates them, is because I didn't want to do it embedded within the chapter, because it would just kind of take away from the story. This is kind of a glossary of what these things do. And so when you see these particular hormones scattered throughout about in the chapter, when I mentioned them in lecture, this is a nice reference. Oh, yeah, that's that hormone coli, cystic kind. And what you're gonna see today, by the way, you're also gonna see secrets. And today I'm about to talk about glucose dependent insulinotropic peptide, right now, this will be the first and last time we talk about it, I'm not going to talk about it beyond this point. But these other hormones, the other three secrets in gastric Nikolay SR keinen, I will talk about them multiple times, because they have multiple functions with multiple things within the digestive system itself. But right now, let's talk about this one right here, this is

actually in the notes are in the endocrine chapter. I just skipped it at the time, because I knew I was going to talk about it now. And so let's talk about what this particular hormone does. So I'm going to draw here, just gonna say small intestine. So that's part of the digestive tract right there. And in that small intestine is going to be the foods that we ingest. And those foods will be carbs, fats, proteins, and we're going to digest those foods into their simplest forms. And one of the things we're going to get when we digest carbohydrates is glucose. So again, we're still in a small intestine, right now we're not in the blood. Amino acids, get those from breaking down protein. And then fatty acids, we're going to get those when we break down the fats. And we'll see how all this happens in the next lecture. Or maybe the lecture after that depends on how things go. So we have glucose now within the small intestine within the digestive tract, what that's going to do, and I'm going to put the word main above here, I'll just give you the punch line, what's gonna happen here is, is we are going to, and this is going to be the pancreas, we are going to cause the release of insulin from the pancreas via the beta cells. Actually, you know what I'll put beta in here. Remember, those, the beta cells are the cells that produce and release insulin. This is going to be the pancreas. Now, what caused the release of insulin earlier on in the semester, when I told you that story was what it was mainly a high blood glucose level, remember that insulin lowers your glucose levels, and of course, little cells of their high need to lower insulin is a hormone that'll do that. But what I told you at the time is, is that it's blood glucose that causes the release of insulin. And that's not a lie. I also said that amino acids in the blood and fatty acids in the blood will also cause the release of insulin. That was a picture that we drew. Well, this is what I didn't tell you at the time, is that those same three things will cause the release of insulin through glucose dependent insulinotropic peptide gap. And so what I will do here is, is I will just do one of those. So those being present within the small intestine caused the release of this hormone, which will then get dumped into the blood and make its way to those beta cells, which is going to be what the target is in causing release of insulin. So what does this mean? It means that your insulin levels go up before your blood glucose levels go up. Because this glucose is still in the small intestine, it has not made its way into the blood yet. That's what this means. Now, again, amino acids and fatty acids will cause the same thing to occur. But it is glucose that is a main stimulus just like glucose isn't a blood glucose. It's a main stimulus to insulin, just as we learned earlier on in the semester. So this right here, that picture right there

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is right there, those two fields, two lines. And that's it. We're not gonna see gap anymore. but we will certainly see the other three hormones scattered throughout the chapter as we're going to see. Now, the paragraphs, you're going to have to wait to talk about those, but we will see those. Now let's talk about the five processes. The first process is called ingestion. And if we go back to the picture over here, I have it right at the top ingestion, ingest. What does that mean? It means to chew your food and swallow your fruit. Now, when we discuss the digestive system, we're going to assume we're eating food, as opposed to drinking liquids, not that that's not going to be relevant. But you don't chew liquid. So I'm just going to be assuming that we're just eating some solid food here. So ingestion are these two things right here. Now there are fancy words for those as we're gonna see in just a second. So we are here. So ingestion, chili, and other word for chewing is mastication. Careful when you say that word, swallowing is deglutition. Same thing. We're gonna start with chewing. Why do we chew our food. It's just mechanical digestion, not chemical digestion, mechanic, we just you're just ripping it apart. And as you rip it apart, you are going to mix it with saliva, and you're going to form a bolus, and the bolus is

what you're going to swap. Now I'm sure that you've been told over the years that if you don't chew your food, it's not going to get digested. Complete nonsense. If you had the ability, like this dude, right here to swallow an entire hamburger, and we get digested just fine. If you swallow the entire state, it would get digested just fine, it would just take longer, because now it'd be up to the stomach to kind of get at all the product of the food that you've just swallowed whole. What's what chewing does is it just increases the surface area. And it just makes it easier to get at the things that need to be broken down. Plus, I'm not telling you to swallow hamburgers whole I don't want you to die. Because obviously that can cause you to choke to death. So chewing is also going to allow you not to choke to death. And so it's it's wise to chew your food. But do you have to know you don't have to chew your food. But when you do, and I'm assuming we do, we're going to mix it with saliva to form something that we can easily swallow and that's the bolus. So that is once again the function of chewing as far as we're concerned. Now what now we need to swallow it. So we have formed a bolus. Now we need to swallow the bolus. There are three phases to it. We have an oral phase, which you control. And then we have the two other phases pharyngeal, and esophageal which you don't control, that's going to be a reflex, that's going to be a reflex, mediated by the swallowing centers in your brain stem when the ball is hits the back of the throat. So what we'll do here is very simply as put together, just three columns. One column is going to be the oral phase. The next column is going to be the pharyngeal phase. And these phases, the name of the phases is indicative of where the bolus is. So the bolus is in the mouth during the oral phase. The bolus is in the pharynx during the pharyngeal phase, and the bolus is in the esophagus during the esophageal phase. And so all I'm going to do with the oral phases, and it's very simple, your tongue is just going to push the bullets to the back of the throat to the oral pharynx. And you have control over that because your tongue is skeletal muscle mainly and we have control over that. That's the somatic nervous system. Once that bolus gets to the back of the throat, you no longer have control over swallowing. It is now a reflex and autonomic reflex. And so what's going to initiate this? Well, the oral phase is initiated by thought, the pharyngeal phases initiated by the bolus being in the back of the throat, so I'll put that year initiated by started by bolus in the oral pharynx, the back of the throat

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in how did he get there, your tongue pushed it there. And then a series of events is going to occur because back there are receptors that are going to detect that bolus or Mecanim receptors. We learned about that last semester. And they have sensory axons that go to the central nervous system, specifically the swallowing center. And then the swallowing Center has motor output motor axons. We talked about this last semester, I'm just doing an autonomic reflex right now. And those motor axons are going to lead to effectors effectors that are going to allow the swallowing reflex to continue. And so four things that you need to know when it comes to the pharyngeal phase. And so we're here right now. So I just wrote this down, just talked about swallowing center, and that swallowing center is going to cause four things to occur that you need to be aware of number one, and as I talked about this, we're going to look at this picture right Here, right here, the yellow was the bolus, this is the oral face, this big muscular tongue is going to push it to the back of the throat. Oral phase is done. Now the bolus is at the back of the throat, what's going to happen? Well, I have asterisks here a few things that you need to be aware of soft palate is one thing that's going to be affected you guys don't soft palate is it can move. And what's gonna happen is it's gonna move upwards. And when it moves upwards, what it's going to do is it is going to close off the nasal cavity. Bolus does not belong your nasal cavity. And so the soft palate moving up is going to prevent the bolus from going into the nasal cavity. So that's one

thing that the swallowing center is going to cause the movement of the soft palate upwards. Number two, is going to cause the epiglottis to cover the glottis. And the glottis is the opening to what? The larynx, the airways, right? Does a bolus belong in your airways? Of course it doesn't. It belongs in the esophagus. So that's going to prevent it from entering the airways. Now, sometimes it does enter the airways, what do you do when that happens? You probably cough right? You drink something? Or you eat something and you start the coughing you say I swallowed wrong? Well, you actually did, you're correct. In that terminology, what happened is, is that you might have taken a little breath while you were while you were you know, eating and swallowing just eating a little bit too fast. The epiglottis didn't have enough time to shut off the glottis. And then that food went in there. Now one kind of receptors Did you Did you hit in the airways with an eye irritant receptors, which caused you to cough that then blown your airways. And so you're going to try to get rid of it. But under normal conditions, that's not going to happen. So number two, the glottis is covered by the epiglottis. Number three, what else has to happen? The upper esophageal sphincter has to relax. Why? Because the bolus needs to get into the esophagus. If that sphincter doesn't relax, it stuck in the back of your throat. That's not where it belongs either. Eventually it belongs in the stomach, that's where it belongs. But we need to get it to the esophagus first. So number three, the upper esophageal sphincter needs to relax and again, Where's all this stuff? It's just one after another soft palate epiglottis upper esophageal sphincter is going to relax. And there's a fourth thing you don't breathe when you swallow. So breathing is inhibited during the pharyngeal phase. Now why would that be? Well, let's think about this for a second. If you're breathing air is going in and out of the airways, right? Can the epiglottis be on top of the glottis at that point in time? No, you can't breathe with the epiglottis on top of the glottis. So if you are breathing, the glottis is going to be wide open. And we know that we can't have that. So to ensure that the epiglottis is covering the glottis, you don't breathe while you swallow. Now, you might have when you swallow it wrong. And that opened up the glottis because the epiglottis had to move in order for you to breathe. But under normal conditions, you do not breathe as you are swallowing. Now, let's say that you did again, you have the risk of aspirating things into the airways aspirate means to get something into the airways that you weren't supposed to get into the airways. I don't know if that's in the notes. I don't think it is, here's something else that could happen. Anybody here ever see, or maybe you've experienced this personally, that you might have drank something, and all of a sudden somebody made you laugh. Maybe it squirts out your nose. When you laugh. You are contracting your respiratory muscles pretty stinking hard, right? And so that's going to create a tremendous amount of pressure within the airways causing a lot of the pressure to Scott a place to go. Where is it gonna go? Well, the glottis and if that fluid is right here, and if you're breathing by the way, do you think your soft palate is gonna come back down?

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Yeah, because when you breathe, air will go through your your nasal cavity. So as you're laughing, that fluid that might be right here is gonna go right out your nose. Why? Because of the increase in pressure. And again, things that are necessary for you to breathe, are going to be in positions that allow you to breathe. And so that's why that happens. But under normal conditions, it doesn't. So know the four things that occur during again, the pharyngeal phase, and why now the bull is going to enter the esophagus. So now we're in the esophageal phase. Oh, and so I'm not going to write these down. So there's 1234 things that I want you to put down. Now, unless you can write really fast. But again, as you're studying, this is the way in my opinion you should the esophageal phase. Now the bolus is in the

esophagus so initiated by the bolus in the esophagus, and it was able to get into the esophagus because the upper esophageal sphincter was relaxed. Now there's a number of things that are going to occur with the esophageal phase. Number one The bolus is going to be in the esophagus. Well, is that where the bolus belongs at this particular point in time it does does the bolus belong in the pharynx At this point, no it did during the pharyngeal phase, but now that it's in the esophagus it belongs in the esophagus and eventually in the stomach. So in order for that bolus to remain in the esophagus and not reflux into the pharynx, what do you think has happened the upper esophageal sphincter it needs to close. And that's something that students sometimes get mixed up on exams. When I asked questions about the swallowing reflex. I'll talk about the upper esophageal sphincter and I'll ask a question about when is it open? When is it closed? And the knee jerk reaction is well of course it opens during the esophageal phase. No, it doesn't. It opens during the pharyngeal phase. As it says right here, it's relaxing, it's opening to allow the bolts to go into the esophagus, it closes during the esophageal phase to keep it in the esophagus, we don't want it back in the pharynx. It's not where it belongs, it belongs in the stomach at that particular point in time. So once the bullet passes into the esophagus, which initiates this phase, the UEs closes. And that's the reason then why the bullet has to travel down the esophagus and it's going to happen through a process called peristalsis. I'm going to talk about it show you how it happens. And then the lower esophageal sphincter is going to relax and allow the bolus to enter the stomach. And that's when swallowing is over. By the way, please know that I think most people think that when you swallow, you're done swallowing once you've got it's no longer in the back of your throat. Swallowing is not over until the boluses in your stomach. People will have swallowing issues can have swallowing issues, because for example, their lower esophageal sphincter just won't open. It's called Achalasia. I'm not sure if that I don't think it's in the notes. But there are certain conditions to where if that bowl is not in the stomach while you're trying to swallow. That's considered a swallowing issue, which is called dysphasia. If that's in the notes, fine. If it's not, don't worry about it. I don't remember I get you guys mixed up with my Grant students sometimes? I don't think it is. So again, there's a number of things that are going on with the esophageal phase, and just write them down and as you study. There you go. That's swallow. And we just took care of ingestion, right? Because ingestion is what chewing and swallowing. Now what, well, let's now go on to the next process. And the next process is motility. And so what is motility, I'll go back to my pretty picture. And I have named motility both mixing and moving. So you gotta mix the contents. And then you gotta move the contents. And you'll do it at the same time. So let's talk about both of these processes here. So mixing and moving is what motility is all about. And it's going to be done because we have muscle in the walls of the digestive tract. And so let us first talk about actually, you know what, let's just do a little overview here. So motility. So motility is again, mixing and moving, we'll do moving first. So we're going to move well, how are we going to move the contents within the digestive tract from the pharynx all the way down into the rack that well how are we going to do that? Well, there's a few things that can happen here. Number one is peristalsis. And I'm going to list those places where peristalsis is going to occur. peristalsis can occur in the pharynx. That's how the bolus is going to get into the esophagus. peristalsis happens in the esophagus, that's how the bolus is going to get to the stomach peristalsis will happen in the stomach peristalsis will happen in the small intestine and peristalsis will happen in a small section of the large intestine.

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So practically, the entire alimentary canal can move things along through peristalsis. And with peristalsis, you just kind of nudging them along relatively slowly compared to another movement that we're going to discuss in just a second. So peristalsis is something we'll talk about in a bit of detail in just a second. I'm gonna show you how it happens. Another way that we move things is called mass movement. And this is only going to occur in the large intestine and the bulk of the large intestine. And so we're not nudging things along here, we're picking up things and we're throwing them great distances, relatively speaking. And so we'll actually talk about that today as well. How about mixing? What mixes things? Well, when it comes to mixing, and I'll just list here those things that can mix the esophagus doesn't mix anything. Mixing starts at the stomach and I'm talking about Well, nevermind, stomach, your small intestine mixes and so does your large intestine. Now, the reason the mixing is occurring in each of these is different and when we talk about mixing specifically I will touch upon that mixing actually has special names that a couple of these organs mixing is called mixing in the stomach. So actually, I'll just put next to the stomach, the word mix again, mixing in the small intestine has a special name, it's called segmentation. And then mixing in the large intestine also has a special name. It's called Hot stration. And we'll revisit those words when we get to these particular organs today, and the process that makes it. And so this is an overview of what we're about to discuss. And so let's start with peristalsis. And when it comes to peristalsis, what I'm going to do here is that it could be any one of these particular organs pharynx, esophagus, stomach, small intestine, large intestine, it doesn't matter, I'm going to draw a tube, that too, can be any one of those organs right there. So this tube right here, I'm going to put some special

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things in the two, I'm going to do this to the tube. And then I'm going to do this to the tube.

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And so same thing here. So what the dotted lines are indicating is a change in the size of the tube at those two different areas. And again, this can be whatever you want it to be. We just said the esophagus, let's say it's the esophagus. And we have a bolus inside the esophagus and we want to move that bolus along to the stomach. So how are we going to do that? Well, this is the bolus right here. Again, it doesn't have to be that could be chyme. If that was the stomach of the small intestine, it could be feces, if it was a large intestine, so that can be anything you want. But whatever it is, we want to move it along, we're going to scoot it along. And what's going to allow that to happen is the muscle in the walls of the digestive tract behind the bolus and this bowl is is going to move in this direction. That's the direction at which the bullets is going to move, just kind of give that punch line. So we want to move it in that direction. Well, what's going to happen behind the bullets and this is going to be behind the bullets given the way that the bullets is moving, we are going to increase pressure behind the bullets and how are we going to do that we are going to squeeze the walls of the digestive tract. Well, how are we going to do that we have two sets of muscle, right? circular muscle longitudinal muscle, the circular muscle is going to contract. So circular muscle contracts behind the bolus. And I think that's fairly easy to visualize. It's circular muscles. So it's wrapped around that that whatever segment that is of the digestive tract, like a snake would be wrapped around your body. And if that snake can tracks what's going to happen, this thing's going to squeeze you. It's going to squeeze the tube. And so that's what this is representing here, the tube is getting squeezed, we are constricting the tube. So we're getting constriction there. I also want you to understand that this is a three dimensional structure, I'm going to

be pointing to this right here. But do know that again, it's a tube and it's happening everywhere. It's happening on this side, this side everywhere X, Y and Z. And so it's going to constrict behind the bolus, the circular muscle is going to contract in order for that to do for that to happen. And at the same time, the longitudinal muscle is also involved in this the launch to the muscles gonna relax, like that you're already talking about both of these are going to happen at the exact same time. Now longitudinal muscle, as you know, is oriented longitudinally. So my water bottle can be whatever the heck up the esophagus and my finger is longitudinal muscle. Whereas if it was circular muscle, I would have thread and I would just wrap it around my water bottle that would be circular, longitudinal oriented longitudinally. Now what happens to muscle when it gets relaxed sessions they stay relaxed. My apologies. Let's just say relax. Well, I'll just ask you what happens when muscle contracts it gets what gets short. And well muscle relaxes, it gets longer. So what do you think's going to happen to the tube if the longitudinal muscle relaxes? Tube is going to elongate keep it two analogies. Let's go to Lowe's. Let's buy some rubber tubing. We're going to take that rubber tubing and we're going to stretch it as if the longitudinal muscle was relaxing. And if you take that tubing and you stretch it what's going to happen to the inner diameter it's gonna get smaller. You stretch that to the inner diameter is going to get smaller if you stretch that to the inner diameter is gonna get smaller. I'll give you another analogy. Those little things that you would buy at the fairs are all a mess you look at and you put them on each side of your fingers. Remember those things? Right? And when you first got to put on your fingers, maybe you're four years old, and you wanted to remove you want to numb the hell off your fingers like Get this thing off my fingers what you do, you probably pulled in this direction, right? You lengthened it? Did you not wanted to do it. He squeezed your fingers. It can it made the the tube it made the little messy thing smaller. So think of the little messy thing around your finger getting longer and squeezing your fingers. Longitudinal muscle relaxing is going to cause that to occur. What happens at the exact same time in front of the bolus while we need to decrease pressure, higher pressure behind is gonna push, push the bolus, if it's a bolus, in this particular direction, if the pressures are lower here, it's just gonna be easier to push it, it's going to be easy, it's going to be easier to move it. And so what's going to happen here is the circular muscles going to relax and the longitudinal muscles going to contract. So circular muscle relaxes. And at the same time, the longitudinal muscle is going to contract. And we can go back to that little messy thing if you want.

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When muscle contrast, it gets shorter, right. So how did you finally get that thing off your fingers? You move your fingers together? And what do you do to the messy thing, you shortened it, and then shortened it, what happened to the inner diameter, the messy thing he got bigger, justice is happening over here. This and this, and this and this are all happening at the exact same time. Why? Because it's the nervous system that is coordinating this entire thing. And the nervous system knows exactly what to do. Because this, this, whatever it is, and I'm just gonna say contents. Again, it could be the bullets, it could be time, it could be feces, it could be whatever, something that we want to nudge along. And what's going to happen is is that this is going to move in this direction. And then when it does, well, now it's going to be here, I'll draw a dotted. Now it's going to be here. And now what's going to happen is now these, this region right here is behind the bolus. And so these muscles that we're doing this are going to do that instead. And this just beautiful way that occurs, it's called the peristaltic wave. And it just nudges, nudges, nudges, nudges. And so what's going to happen over here is we're not going to constrict, we're going to dilate. so that it's easier to move it forward. So that's peristalsis. Now what,

well, now we'll do mixing, and again, mixing and moving mass movement, we'll get to it, when we get to it that is a more violent contraction and the Peristaltic contractions, were picking stuff up and throwing it with mass movement. Whereas with peristalsis, we're just gonna, let's just nudge it along, more or less. Now, what about mixing a little easier story. With this particular story, what I'm going to do is I'm going to draw three different time points. In this particular picture, we were at one time point, because everything was happening at the same time. Here, we're going to have three different time points. So I'm going to draw the two, three different times one on top of the other. And so here's our tube initially. And again, when it comes to what makes his stomach, small intestine, large intestine, so it can't be the esophagus, it can't be the pharynx, it's got to be one of these three. And so we have a tube, and we're going to have contents within the two began. So those are the contents of the two. And I'll put above that contents contents can be bolus and the stomach came in the stomach calm and the small intestine feces in large intestine. And we want to mix it now what are we mixing it with fluids within the digestive tract. And these fluids contain lots of enzymes that are going to allow us to digest what's in the context, the foods that we eat, it could be a bunch of gastric juices in the stomach that's going to help us form chi, we're mixing for a very good reason. And we need to mix or the digestive process is just not going to happen properly. So we want to mix this with the fluids within the digestive tract itself. So how are we going to do that. So now at a later time, what's going to happen is is that the tube is going to do something that we just saw with peristalsis and that is yes, we are going to squeeze the contents and when we squeezed the contents, we'll do it in such a way that it'll separate. Some of the contents will go this direction, some of the contents will go in that direction. And as we do that, we will allow the mixing of those contents because it's going to be easier because surface area is bigger now because we had two pieces as opposed to one piece. Now what's going to allow that to happen. circular muscle is going to contract and that's mainly what's going to occur so circular muscle contracts. And now what's gonna happen is we're gonna get to the original picture there at the top, it's gonna look like this again. So now the circular muscle was going to relax back to its original state. We're not going to dilate like we did over here, we're just going to go back to the original state. And now what we're going to see is I'm going to draw this lighter now. So I had the content strong, very, very dark here. Now I'm just gonna kind of draw it lighter. It's lighter, because it's been mixed with fluids, please not light enough. So I'll give you another analogy here. Come on, there we go. So we've mixed it in the mixing is occurring here. When we squeeze it, squeeze it apart, fluid gets incorporated into it, bring it back. And we've just now mixed it and now it's lightened in color. Again, that's the way that I'm depicting it.

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Let's say over to cheese house on a Sunday, and Barbara Tucci is there and she wants to make meatballs. How do you make meatballs, you get a whole bunch of hamburger, you put it in a big pan, then what do you do? You put eggs in there and bread crumbs and other stuff that Mama Bertucci puts in there. And then what do you do, you take your hands, and you start to mash it like this. And when you mash it like this, it's going to start to mix the eggs and the bread crumbs and Parmesan cheese and all the other goodies that Bommarito he puts in those meatballs by doing this. So you squeeze, you release, you squeeze, you release, you squeeze, you release, you squeeze release. And as you do that you mix and this is very necessary, again, for things to happen properly within the digestive tract itself that allows for digestion, for example, in the formation of chyme in the stomach. So circular muscle contracts and then relaxes. That's how you mix. Now what I haven't mentioned splinters yet certainly in that picture, I talked about mixing and moving peristalsis would be a movement mass

movement would be a movement mixing is mixing What the hell does the sphincters have to do with anything? Well, I'm going to show you splinters have to do with movement. So I did mention and I just didn't mention sphincters specifically when it comes to movement. So what we'll do here is is we're going to draw three different tubes except I'm going to orient these vertically, and I'm going to put a sphincter in the picture. So we're going to have to Oregon so I'm going to call it Oregon one and Oregon two, that'll be Oregon one. And I'll label everything so Oregon one organ wants to be the esophagus if that's the esophagus Well, this has to be this the stomach. So organ one an organ, two or two adjacent organs, esophagus, stomach, stomach, small intestine, small intestine, large intestine. The sphincter is right here. That's the sphincter which is as you guys know, a ring of muscle. Most often in the digestive tract, a ring of smooth muscle, except the upper esophageal sphincter and the external anal sphincter, our skeletal muscle, as you guys know, but everything in between the smooth muscle but it's still a real muscle. And at this particular point in time it's closed. We are not going to get movement of whatever is an Oregon one into Oregon to whether it's bolus or chyme. And so here, the contribution of the sphincters to motility is a big fat zero. But we can change that, we can get things to move from organ one to organ to if we just relaxed the sphincter, if we just decreased the tone of the muscle, and that's what we're going to do in the next panel here. Now, if it's smooth muscle, you can greed, the contraction meaning you can contract it hard or not so hard. Whereas skeletal muscle, it either contracts or it doesn't as we learned last semester, but what we're seeing here is a decrease in tone. So it's opening, we are relaxing the sphincter. And because we have a decrease in tone, whatever is in Oregon one now can move to Oregon two and a certain amount of it and the arrow is going to indicate how much we're moving. So in this particular case, we've just increased motility, specifically, movement is what we're talking about here moving things from point A to point B. And we'll just do one more before we go on break. We can really relax the sphincter, so we can get more through. So I'll make the arrow bigger. So here we had a bigger decrease in toe. Again, more relaxation. And so because of that, motility went up, it was easier to move things from Oregon one to Oregon two. And so that's how the sprinklers behave now, when it comes to the sphincters themselves, as you know well let's take a break when we come back a little bit more about the sprinklers and how they work. Shall we continue? Now before we move forward, just a couple of other things I want to mention here. What's causing the sphincter is to relax. What causes the relaxation of the sphincters is peristalsis. So peristalsis causes sphincters to relax to have a decrease in tone. That should make sense. If we're going through peristalsis, that probably means something is there, right? And actually, that's something that I should mention as well in this particular picture. And of course, this is peristalsis. I didn't put the word peristalsis here. So let's add that to this picture. And what I also add here is is that caused by

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the presence of contents when contents or their bullets kind, whatever feces, it'll stretch the walls a little bit nervous system going to detect it. And then we had this occurring. So if sphincters are relaxed by peristalsis, what that means is, is that something's there. And that would make sense. If something's there Don't we have to move it from point A to point B, that we needed to get it from Oregon one to Oregon to? Yes, we do. And so those fingers will relax. So for example, when the bolus goes into the back of the throat during the esophageal space, your lower esophageal sphincter opens right away. Don't wait for the bulls to get near it opens right away. We'll actually see that in just a second. So that's it for the sphincters. Now what? Well now let's talk about individual organs and how motility occurs in

each of those individual organs. We'll start with the esophagus. When it comes to the esophagus, we've actually already talked a little bit about this because this is, in essence, the esophageal phase of swallowing that is, now I want you to know some numbers here, three to 10 seconds for bulls to move through the esophagus. Why such a big range? It depends on how big the bolus is. It's a whole hamburger might take a little bit longer than 10 seconds. But if it's a normal sized bolus, it's going to be anywhere from three to 10 seconds. Please know that. And by the way, when it comes to peristalsis, and getting that bolus to the stomach, does gravity help? Sure do you need gravity if you don't, you can stand on your head and the bolus is still going to make its way to the stomach. That's why astronauts when they are in the space station floating around and near zero gravity, they can eat food, and that bolus is still going to end up in their stomach because the muscles will contract in such a way obviously to get the bolus to where it needs to go from point A to point B. Now, I just mentioned this peristalsis causes relaxation of the lower esophageal sphincter I mean I just mentioned it here what relaxes the sphincters peristalsis peristalsis in the esophagus relaxation of the lower esophageal sphincter Now, you might not be familiar with the term lower esophageal sphincter is the cardiac sphincter it's the same thing. I think anatomists like the word cardiac sphincter I think physiologists more prefer lower esophageal sphincter because we have an upper esophageal sphincter. So that's the term I will use. But cardiac sphincter you can use that as well, this the same is the same thing. And what esophageal motility is going to allow is the conclusion of the swallowing reflex and obviously allowing the contents to be emptied into the stomach and the content in this case is going to be a bolus. Now, if peristalsis is not occurring, if there's nothing in the Oregon that we're talking about, in this case, the esophagus, that lower esophageal sphincter should be closed. Sometimes that doesn't happen. And so I'm going to give you a particular condition where that doesn't happen. So again, sphincter should be closed. If again, peristalsis is not occurring. So what we'll do here is withdrawal, the esophagus there's going to be our lower esophageal sphincter, I'm going to label it, and then that'll be the stomach of course. So I'm showing you abnormal right now. patho is so here's our esophagus. Now, there's nothing in the esophagus I'm not going to draw anything in the esophagus because nothing is there. And if that's the case, once again, the lower esophageal sphincter, which is what this is, right there, Tony should be very, very high. She looks like this right here. But it doesn't it looks like that or that right now. And so because of that, what can happen is you can get what's called reflux. Now, why would things go from the stomach into the esophagus? Why wouldn't they go from the esophagus into the stomach? In this particular scenario? The pressures within the stomach are much higher than the pressures in the esophagus. Why? Because the muscle in the walls of the stomach are very much bigger and stronger and When they can track things are just gonna go in this direction. And so when that happens and when you have this particular condition and so we have here, Le s is open when it should be closed

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Kinley lead to reflux you might think to yourself what do you do? It's just gonna go right back into the stomach. Yeah, probably is. But the contents of the stomach can be very acidic. The walls of the stomach they can they can withstand a pH of down to one what's the lowest pH zero pH scale Zero 14 Zero is the most acidic once incredibly acidic. Stomach can withstand that wall to the esophagus can while the esophagus can withstand a pH four. And routinely the pH in the stomach is at least down to about three ish. And so this reflux can damage the walls of the esophagus and make things very uncomfortable so can lead to reflux. And it's called gastro esophageal reflux disease gastro stomach, esophageal esophagus reflux refluxing from the stomach into the into the esophagus and is a disease.

Now, what can cause this? Well, just dysfunction of the lower esophageal sphincter. So dysfunction of the LDS it just doesn't work, right. no cure for it, by the way can be treated. And we'll talk about treatments in the next lecture. Something else that can cause it is just higher pressures within the stomach itself that just kind of force it open. And so high stomach pressure. Now, why the hell do we have high stomach pressure? Well, I'll give you an example during pregnancy, later term pregnancy, you know, not the first few months but when the baby's big, and the baby's pressing on things like, you know, organs within the abdominal cavity can cause pressures to go up, it'll go away, almost always when the pregnancy is over. Once the baby is delivered, the GERD goes away. But it is a common problem with people who are pregnant. Gerd is and so once again, the acid now in the esophagus because there's acid in the stomach, other things are going to reflux as well as the acid is going to be doing the most damage. So acid causes damage to the esophageal wall. Most often, it's not going to feel very pleasant. And so you can get what's called heartburn. If you get heartburn, you're refluxing why they call it heartburn. It's got nothing to do with your heart. But obviously the esophagus is right in the same area of your heart and it feels like your heart is burning yards not burning the walls of your esophagus. That's what's feeling the pain and that's where you that's where the pain is coming from. But here's the thing. There are some people who have dirt nilly and know it. It happens so slowly and insidiously, that they never know that they have it and GERD causes some weird stuff that happen. Like it can cause post nasal drip, for example, which is prevalent this time of the year with all the allergies, it can cause all kinds of weird things. Gerd can, things that you'd never imagine in a million years. Gerd is the cause of like metallic taste in your mouth. That could be due to GERD, for example. You don't have to know any of that. Just just know what I have up on the page here. All right. And it says oh and by the way, again that I tell you there's no cure. You know I did there's no cure, but you can treat it we've not figured out a way to put a you know, a dandy Le s in there, the one it's not working. So you just have to manage the symptoms. So anyway, that's the Safa geo motility. Now, what about the stomach? The stomach is a mixing machine. That is its main job when it comes to motility and what isn't mixing bolus with gastric juices. And those gastric juices are going to eventually form this mixture called Chi, this pea soup looking fluid that will then be emptied into the small intestine. Now, even though that is its main function mixing, we're going to spend more time on peristalsis. gastric emptying and gastric emptying is just the stomach emptying its contents into the small intestine. And that's going to happen because they're going to move the Kime which is what it is now into the small intestine. So we're going to go from Oregon one to Oregon two. So if this is the stomach and small intestine, that would be that would be gastric emptying. So let's talk about gastric emptying.

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Each peristaltic wave empties 1% of the contents of the stomach at a time. So every time the stomach muscles cause peristalsis to occur, it's going to squeeze out 1% of the Chi One at a time, and it's going to happen about three times a minute. And it's kind of written in stone. Now why that is, is something that we're not going to get into. So three peristaltic waves per minute 1% Each time, so about every 20 seconds, if there's something in your stomach, it's gonna get squeezed into the, into the small intestine. Obviously, peristalsis will then open the pyloric sphincter course, that is the sphincter in between the stomach and the small intestine, you guys know that? What else do you need to know some more numbers, two to three hours to empty a normal sized meal. So the esophagus was what three to 10 seconds to move the bullet into the stomach, right? You need to know that, how long does it take to empty the stomach, two to three hours for a normal sized meal, by the way, I have that here. Okay, if

you eat an m&m, it's not going to take three hours to empty an m&m from your stomach, it's not going to take very long at all. So we're talking about a normal sized meal here. There's a range though two to three hours. Why is there such a range, part of it is due to what you've eaten. If you eat more carb type meals is going to empty a little bit fast doesn't mean that the carbs go to the front of the line, you get empty first, this is we're talking about a whole. But if you eat, let's say 80% carbs in a meal that will empty faster than if you ate 80% fat in the meal Fasken empty slower. And I'm going to explain to you why in just a little bit. Highly, highly regulated as gastric emptying. So that's the next thing we're going to do. So we're going to talk about what stimulates it and what inhibits it and spend most of our time on what inhibits gastric emptying when I say inhibit doesn't stop it. It just slows it down. And for very good reasons. Before we get into what we're going to talk about most and that is inhibition. Let's talk about stimulation. So what was stimulated gastric emptying, why would you empty a little bit more from the stomach per peristaltic way? Well, if there's more stuff in the stomach, simple math. So if there's 100 milliliters of climb in the stomach 1% of 100 is what one mil if you have 200 milliliters of time in the stomach was 100 was 1% of 202. So you're just going to empty more time if there's just more time in the stomach plus if it's more fluid. And so what does that mean? Well, the stomach's job is to take the bullet and mix it with gastric juices and make it fluid. That chunky. If it's more fluid, it's just easier to move. That's all. So that's what stimulates now what's going to inhibit, we're going to draw this. So I'm going to draw the stomach in the small intestine that so stomach will be up here. I'm going to make the small intestine nice and long here, specifically the duodenum, but there's really no need to really label that I'm just going to put small intestine I'll label stomach here. So there's the stomach and this is going to be the small intestine. And I'm not going to draw the sphincter but we know what sphincter it is pyloric sphincter. So let's start emptying the stomach. Again, we're emptying chi. And we're going to assume that we just say, a normal sized meal with all the food groups in it's got carbs got fats got protein. And so now the kind gets emptied into the small intestine. And in that kind, let's say this lipid, so you had some fat in your meal, which is good. That's good for you, as we learned last semester, many fats are. So now there is lipid in the small intestine, that's going to affect the stomach's contraction. Peristaltic contractions how the lipid is going to be sensed in the small intestine, there's receptors that will sense it. They're called chemo receptors. We learned about them last semester. And what those chemo receptors will do will then cause the release of your come some hormones now I'm going to abbreviate it I want to write those stinking thing out call the sister kind and call the sister chitin is one of the four hormones that you need to learn and it's right in the beginning of the chapter. And in there, it'll tell you that fat in the small intestine stimulate us release is going to get released into the blood, whereas its target the smooth muscle of the stomach. That's the target of CCK, which was released in response to fat in the small intestine. And so what is it going to do? It's going to inhibit the heck out of that muscle and slow down gastric emptying. So what I'm showing you here is inhibition of gastric emptying, which is in essence peristalsis. So inhibition of the emptying of the stomach.

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Now why? Why would it be that fat in your small intestine slows down the emptying of the chyme into the small intestine. This is the reason that is difficult to digest. And it's difficult to absorb. It's the most difficult thing to digest. It's the most difficult thing to absorb. And when we talk about digestion, small intestine is where most digestion occurs. So if you have a product, it's really hard to digest and really hard to absorb. You don't want to dump it into the small intestine too quickly. You want it to trickle in. So You give the small intestine time to digest it. And so why is it that this is going to be inhibited the way

that it is? Gives the small intestine time to digest and absorb the lipid. If you dump it into the small intestine too quickly, you're going to have problems. Okay? We don't want problems. We want things to work properly, we want to digest everything properly absorb everything properly. So lipid has, and I put three negative signs there. Let's go to the nuts. Lipids in the duodenum, most potent inhibitor of gastric emptying. That's the reason. Let's go back over here. Why is it that lipids empty the slowest I just gave you the reason because they're the hardest thing to digest. And they're the hardest thing to absorb. That's the reason cards are easy to digest and absorb by the way. So if you eat a heavy card meal, it'll just empty quicker, because it doesn't have an inhibitory effect, like fat does. What else next on the list, chi with a pH below two and it's going to put acid certainly, there's going to be acid in that kind because what is a lot of acid in the stomach. And so the acid is going to be sensed by some receptors. And once again caused the release of CCK and the another hormone that you need to be familiar with secretin you can say sekret 10. Both of those pronunciations are correct, which will have an inhibitory effect on the stomach. Now I'm only going to draw one negative sign now you might think to yourself, Well, Dr. rscc Day has three negative signs, why would CCK and secrets and only have one negative sign? The reason is because the lipid causes way more CCK to be released. That's the reason not as much CCK will be released in response to acid and secrets and just doesn't have the same type of inhibitory effect on the muscle. That's the reason. Now why is it that acid would cause gastric emptying to slow this is the reason so this is the fat story right here. This is the acid story. It gives the small intestine This is about acid time to neutralize the acid two reasons for that, number one, the walls of the small intestine just like the walls of the esophagus cannot withstand an acidic environment it'll get damaged. Reason number two that we need to neutralize the acid. The enzymes in the small intestine and there are a ton of them as we're going to see won't work. The enzymes in the small intestine need a fairly neutral environment to work. So if we don't neutralize this acid walls will get damaged ulcers. Oh, I didn't mention that over here, did I? So acid causes damage to the esophageal walls, ulcers in column peptic ulcers Same thing here. You'll damage the walls of the esophagus causing ulcers in the esophagus not just again the sock or I'm sorry in the small intestine. Now, how is it that we are neutralizing the acid we are mixing it with a base called bicarbonate. And the bicarbonate is going to be coming from the gallbladder originally it was in the liver and also from the pancreas. We'll see it in the next lecture at least I think we will. So what's gonna happen is that gets neutralized. So that's two things a third thing hypertonic we know what that means? don't or can't be and it's got to be one of the other you can't have kind that's too concentrated hypertonic or not concentrated enough hypotonic it's gotta be one or the other. But regardless, we don't want hypertonic we don't want hypotonic what do we want? What do you think isotonic? That's what we want. We don't want osmotic gradients. So this is a reflex that occurs. And so that reflex will feed back and also inhibit the stomach. Now why what's the reasoning behind this? Because it's going to once again give the small intestine time to do something different. So it gives the small intestine

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time to make chi Kime isotonic. Now how is it going to do that? Well, it's going to mix it with fluids that are isotonic that will eventually bring it out to an isotonic or bring it down to an isotonic. State that is, so that picture right there are these words right here and you notice right here, boom. All right. Now let's move forward. plus your small intestine motility. Small Intestine mixes just not as much as the stomach does, but it does an awful lot of mixing. And it's the most prevalent of the motility, small intestine. So there's a lot of mixing has to because we need to mix, the kind which came from the stomach with a

bunch of enzymes, for example, that will allow for digestion. So very important peristalsis. So in more numbers, four to six hours to move the kind to the small intestine, three to 10 seconds for the bolus to move to the esophagus two to three hours for kind of move through the stomach into the small intestine, four to six hours for kind to move through the small intestine into the large intestine, please know those values. Now. Let's go back to segmentation. And again, that's the name for mixing. Right? I already introduced that to you right there. Small Intestine mixing is called segmentation has a special name. And so now let us take these words and draw it. So we're going to concentrate on motility again, except this is mixing in the picture that we just drew a second ago with the stomach. Oops, that was about peristalsis gastric emptying. And so let me put that here in parentheses just to make sure that you understand the gastric emptying is about peristalsis. Our story now with small intestine, I'm going to concentrate on mixing segmentation. So in this particular case, this is going to be stimulation, segmentation, not inhibition of gastric emptying, you'll see why. So I'm going to draw the small intestine. One big tube. And so here's the small intestine. I'll label it. Oops, spell it right. There's going to be a number of things. And we're going to repeat a couple of them that we just saw in the last picture, the first one we'll do and this is distinct. Because what I'm going to do here is I'm gonna do this. Now, this is not the the smooth muscle causing dilation. That's not what this is, as we saw here, so I don't want you to think that it's this it's not, that's just stretch, we're gonna call it distinction, not dilation. distension is if something is present there. And what will be present there, it's kind, where did it come from came from the stomach. And so now there is kind in the small intestine, the small intestine needs to do something about it. Well, in that kind could be fat and protein, and carbs and acid. So what the distension is going to do is feed back onto the small intestine and stimulate segmentation. We're going to mix mix, mix that kind with what some enzymes, we're going to mix mix, mix that kind with what, bicarbonate to neutralize the acid that's coming from the stomach. So just the mere distension of the small intestine, you could put rocks in there. And the same thing would happen. If you stretch the walls, there's going to be an autonomic reflex that causes this to occur. What else is going to stimulate motility? Well, one that we just saw a second ago, acid, acid in the small intestine, again, let's make sure that we understand this is in the small intestine. What's the acid going to do? Same thing. It's going to stimulate segmentation. And so this first one over here distension why are we mixing the answer to and I'm just going to put take care of the contents of the Chi. What does that mean? Well, it can mean a lot of things it could be mix it with enzymes, you mix it with bicarbonate to neutralize the acid, although I have acid as a separate story here. Well, we're here acid. The mixing is going to help neutralize the kind because we're going to mix it with bicarbonate. So we mix it with bicarb. We'll talk about bicarbonate later when we get to the liver, gallbladder and pancreas. So we have acid there. What else hypertonic hypotonic kind. We saw that in the last picture as well. hypertonic it's gotta be hypertonic or hypotonic.

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Same thing. It's going to feed back and it's going to stimulate Well, what's that going to do? What's the increase in mixing going to do? It's going to help the small intestine create an isotonic chi because it's going to mix it with isotonic fluid that is in the small intestine. So that's the reason for that. And then last but not least, protein digested products, I'll just put protein that too is going to feed back in increased segmentation increase mixing. Now, why would this be? Well, we haven't talked about it yet. But the enzymes that are responsible for digesting protein, many of which come from the pancreas are for lack of a better word, nasty enzymes. And when those enzymes come, they want to start to eat things. And so we want them to eat, we want them to digest the products within the time. And it's going to be

protein products, because that's what's going to signal them to come on down. If we don't mix properly, those enzymes might be sitting on the walls of the small intestine, and they'll just start to eat the walls of the small intestine. Obviously, that's not a good thing. And so why is it that we're increasing mixing when we have protein present within the small intestine is going to help mix protein digesting enzymes we'll see them so that it doesn't eat, eat the small intestine wall itself. Now, we're point something out here. We have a couple of things that are similar in both of these pictures, acid in the small intestine hypertonic or hypotonic, calm and small intestine acid in the small intestine hypertonic or hypertonic Kine in the small intestine, but we have two very different stories. acid in the small intestine inhibits motility. hypertonic or hypotonic came in a small intestine inhibits motility Well, acid in the small intestine stimulates motility. hypertonic or hypotonic kind stimulates motility well, how can it be both? Because we're talking about two different organs, and we're talking about two different processes. So on the exam, don't go on autopilot. Say, oh, yeah, I remember that one picture that I drew 100,000 times learning this material. Acid stimulates motility? Well, that's so fast, because if that question is asking something about acid and gastric emptying, peristalsis well as actually inhibition. So the same products that are in the same exact place, will either inhibit gastric motility or stimulate segmentation. And as for the same reason, why are we slowing down the acid? Why are we slowing down the hypertonic and hypotonic coming into the small intestine to get the small intestine time to neutralize the acid to make hypertonic or hypotonic? Time isotonic? Well how small intestine do that? segmentation. So the two processes work beautifully together, slowly the emptying is going to help the small intestine do these things so that everything works, right. So again, make sure that when you read the questions on the exam, careful what you read, and understand both of these processes here. So anyway, that's small intestinal motility. Now, large intestine. Let's talk about mixing. We already saw this word Hall stration. When we see it, when I first introduced mixing right here, so large intestine mixing hot stration. Very different reason on by the way, let me just point out just to make sure you understand mixing in the stomach is to make chi mixing in the small intestine, a gazillion different reasons that we just talked about, neutralize the acid mix enzymes with the chyme, make hypertonic or hypotonic, thyme, isotonic, those kinds of things. Ha stration mixing in the large intestine is to make feces. That's why we're mixing in the small or the large intestine. So the large intestine is going to receive products that weren't digested or absorbed. And well, that's waste material. And so how striations is going to aid the formation of feces, which is going to contain a number of different things, including bilirubin, right, the conjugated bilirubin, that's what makes your feces Brown. That's going to be in your feces. We already learned that in the bloodshed. Now,

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let's talk about peristalsis. Not a whole lot of peristalsis going on in the large intestine, just the ascending colon. That's going to kind of nudge the feces along. But once we get to the transverse area, not that the transverse colon can't go through what's called mass movement, but it's going to be a bit more prevalent once we get to the transverse colon. So let's draw a mass movement and once mass movement, it's another moving. Let's go back here just to remind you, so we have peristalsis. We have mass movement. And as I mentioned before, peristalsis we're kind of nudging things along mass movement. We're picking it up up, and we're throwing at relatively long distances. And so with mass movement, I'll just draw the colon or the large intestine very, very easily like this. And I'll label everything. So here's our ascending colon. There's a transverse colon, of course. And this is the descending colon, so named because that's the direction in which the PCs are going to move up,

across, and then down. So let's say we have feces right here, I don't have to put contents that is feces, that is the contents. And with mass movement, they're gonna go from here to there, for example, might not be quite that far, but could be. There's your mass movement. I'll be dramatic about things. Violent contraction of the muscle. peristalsis is contracting, certainly, and relaxing to move things along. This, again, much more violent, that's why things are going to be moving great distances, it doesn't happen very often, though, for the average person just a few times a day. Three times a day, give or take. doesn't have to happen that many times. Why? Well, because things are moving a great distance. And is that a great distance to move? Right, the large intestine is how many feet four, ish, somewhere in there. By the way, on cadavers, those those like, like, the organs themselves are going to be a little bit longer. Because the muscle has relaxed at that point. Like in the body, they're a little bit shorter than what they stated when it comes into a lot of the books that you read. Anyway. Let's look at this reflex right here, the gas or what's gastric, stomach, colon, large intestine, so it's going to be a reflex that involves both of those organs. So I'm gonna show it to you. So we'll draw that. So I'm gonna draw the stomach here, there's going to be a very simplified picture. So I'll just draw this stomach is like a circle up here. I'll label it. So that's the stomach. Nope, can't draw out there. I can't label it there actually, only a little inside. So that's the stomach, then this will be the small intestine. That's gonna be the whole small intestine, duodenum, alium, jejunum, all of it, I'll label it. And then that'll be the large intestine, which is bigger. So there's our large intestine. Small Intestine. So let the story begin. The story is going to begin when you've eaten enough food. Again, not an m&m, but an entire meal to cause the distension of the stomach. So that's going to be number one. So number one, distension. It's not dilation, it's stretch distension. So distension of the stomach. Now, there are a wall or I'm sorry, there are receptors in the walls of the stomach, are what are called McCanna receptors. And they detect stretch. And when they do number two, they're going to be stimulated and there are axons that come from them. And so number two, we are going to stimulate the mechanical receptors in the walls of the stomach. Number three we are going to stimulate the parasympathetic nervous system and as a result of that, what's going to happen is number four, you're going to cause contraction of the large intestine gonna cause mass movement. So number four as a result of stimulating the parasympathetic nervous system this is just another autonomic reflex for mass movement occurs well why in the hell would something going on way over here in the stomach? feet feet and feet feet feet away cause this to occur in the large intestine. Can we think about this logically? Eventually, now why do we descend the stomach there's stuff in the stomach food and stomach

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is some of that food gonna be waste? Eventually you think? Of course it is. So eventually this food is in the stomach is going to be feces in the large intestine. And so what's happening here is we are literally making room for more feces. It's an anticipation of this food have actually be in feces. So why does this occur occur? This is the gastro khaolak reflex. What's the why to it? The answer is to make more room for feces, you got to get the old feces out of there. You got to get it into the rectum so we can excrete it so that we can form some more that will eventually be coming because while you're eating food Alright folks, we're done. I'll see you on Thursday.

# Digest PM 4-14-22

Fri, 4/22 4:22PM • 1:14:08

**SUMMARY KEYWORDS**

small intestine, saliva, enzymes, draw, pancreas, digest, acid, called, stomach, produced, parasympathetic nervous system, stimulate, salivary glands, receptor, inhibit, parasympathetic, gallbladder, lactose, lumen, isotonic

00:02

All right, it's about that time. So last lecture, what did we do? We talked about ingestion, we finished up motility in the large intestine. Now what we're going to do is we're going to skip down here. And we're going to talk about elimination. Although there is some motility involved in elimination, it's gonna be a reflex that we draw. And that reflex is called the rectal sphincter Eric reflex. So let's do that. And to do that, we're gonna have to draw the brainstem of the spinal cord. So let's do that really quick. label everything, it's only the sacral region that's going to be involved in the story. And in that sacral region, I should have given myself a little bit more room in that sacral region is going to be the rectal sting, Tarik reflex center. And those are going to be neurons that are going to be controlling this reflex for be involved in a reflex, I should say. And we also going to need to put the rectum in this picture, and that will be this. It's going to receive feces, I'll just put the word rectum here. Here's our reflex center, right, they're just a bunch of neurons. And let the story begin. Story is going to begin when we fill the rectum with feces. So number one feces, in about 30% fullness, we're going to start this particular reflex, and what's going to happen is is we are going to distend the rectum. And as distending well, because there's more feces in it now. So number two distension. Now, when the walls of the rectum receptors in these receptors are going to, they're going to detect that it has distended and those receptors will have a ferentz sensory axons that go out to the reflex center. And so that'll be number three. And what it's going to do is it's going to stimulate those particular neurons there. And now these neurons know there's feces in the rectum, we need to do something about it. So we have, and this is a parasympathetic nervous system. So there's a long preganglionic neuron short postganglionic neuron. So I'm going to put parasympathetic nervous system here. And this is going to be number four, we're going to stimulate the parasympathetic output. And what is that going to do? Well, the target is going to be the smooth muscle of the wall and the target is going to be the smooth muscle of the sphincter, specifically the internal internal sphincter. So number five, is that 512 Yep, five, this is what's going to happen as a result of the parasympathetic nervous systems stimulation. So number five, we are going to contract the rectal wall. And what that's going to do is give the urge to go. So when you feel like you have to go it's because the rectum is being squeezed and what squeezing the rectum the parasympathetic nervous system. Also number five, we are going to relax the internal anal sphincter, which is made of smooth muscle of course. Now when we decide to excrete the feces is going to be up to the individual because the external little sphincter is made of what skeletal muscle which is under voluntary control. That's the somatic nervous system. And that's the next part of the notes. So this right here, what we've drawn is direct eliciting Tarik reflex. The enteric nervous system is also involved as a

local reflex, but doesn't really play a big role, which is why I focused on the parasympathetic nervous system. So that right there is this right there. And then this again, we decide when that's going to happen. All right.

04:03
Next on the list, and what is next on the list next on lists is going to be digestion, which is what we're going to spend the most time on. And when we talk about digestion, what we're doing is we are catabolism the things that we eat, and we have to we have to take carbs, digest them into monosaccharides. We have to take proteins and digest them and the tripeptides died peptides and amino acids and we have to take the fats that we eat and digest them into fatty acids, cholesterol ions, couple of other things that I might mention, I don't remember if I do or not. Why do we have to do that. We need these molecules to be small enough so that we can absorb them, which means we're going to take them from the lumen of the digestive tract and into these epithelial cells and then eventually they're going to make their way into the blood. There are no transporters for disaccharide are polysaccharides or proteins that are bigger than four amino acids or triglycerides. There are no transporters for those. So we need to digest these things. We eat. And so we're going to talk about that now. And the way that we're going to approach this is, we're going to start at the mouth and just move our way through the digestive tract and see what happens at each of these different regions. And we're going to talk about the accessory organs too, as you're going to see. So let's start at the mouth. When it comes to the mouth, we're going to have some mechanical digestion. And this is something that we've already talked about when we were talking about what the role is, of your teeth and your tongue and such. And we did that back when we were talking about saliva way in the beginning, for forming the bolus, so we're going to just rip the food apart, we're going to rip the food apart. And that's mechanical, digestive, so we are not breaking it down chemically, we're just making the food smaller to increase the surface area to make it easier to break it down chemically. Your salivary glands are going to be involved in some digestion, but very little. And so now we get to talk about the salivary glands, which is an accessory organ. By the way, let me point something out to you. So back in this picture, I have listed a number of things that are not part of the digestive tract that I call accessory organs. So we just covered three of them teeth, tongue, hard palate, and now a fourth salivary glands. So we're going to get to the others too, in this lecture. And these things are going to help us with digestion, they do other things. But that's going to be our angle when it comes to these accessory organs. So what we're going to do now is we're going to talk about some spit, I'm going to show you how it's produced. Now, saliva itself has two components to it a watery aqueous component. And it's got an organic component, the organic component is going to contain a couple of enzymes and a couple of other things that I'm going to mention. So what I'm going to do is announce this is already on pilot, by the way, the things that you don't have to know I've already put on pilot the omissions. I'm going to show you how we make saliva. And so it's going to be a couple of things. So it's going to be these words minus the ones that we have crossed out plus these words. In order to tell this story, we need a blood supply, you'll see why. So this is going to be a capillary up here. The capillary of course contains blood liquid part of blood, this plasma. So I'm gonna write the word plasma in this tube. And so we have plasma there. Now I'm going to draw salivary gland doesn't matter which one, just generically, a salivary gland kind of looks like an old fashioned light bulb. So there's a salivary gland. And we have to draw another blood vessel over here capillary, you'll see why doesn't have to be that big, though. So let the story begin. And the story is going to begin with the process of filtration. And you guys know what that is, of course you do, we are

going to squeeze some plasma out of that capillary right there through the process of filtration. Kind of like the glomerulus does kind of like every other capillary in the body does almost every other capillary.

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So we're squeezing the plasma out. When you squeeze plasma out of a blood vessel, you can't call plasma anymore. You gotta call it whatever it is within that compartment, either interstitial fluid, or in this particular case, saliva. Your spit used to be plasma. That's how it started off. So now we have saliva within the salivary gland, that saliva at this particular point in time, is isotonic. So it's got an osmolarity of around 300 ish. It also has a pH that's close to the plasma, which is going to be somewhere around 7.4. Now, why do I point those two things out, because they're going to change as the saliva makes its way through the salivary gland, it's going to get modified. Now in the notes, I have some detail, that's just not important for us to know. And by the way, this is the lumen. That's where the saliva is going to end up that's inside your mouth right there. And so the salivary gland is going to secrete it into the lumen of the mouth. Before it does that, though, again, we're going to modify the saliva. And we're going to modify it in a way that's going to create a hypotonic saliva. So what I'm going to write over here is the word saliva. And I will put underneath it no longer isotonic but hypotonic, we are going to remove some solutes from it. Now we're obviously going to add some solute to it, but we're going to remove more solute than we add. And so we're going to dilute it down. And I'll tell you why. Now there's one solute, I do want you to know about next bicarbonate. What we're going to do during the modification process is that's why I have this blood vessel over here is we're going to add some bicarb to the saliva, which is a base. I'm going to assume you guys probably knew that. So the pH is going to go up. So it's no longer going to be a ph 7.4. It's going to be closer to like 7.8 or so. Hands on the flow of the saliva. But we don't have to get that detail. All I'm gonna say is, now it's Alkalyn. It has a higher pH. Now, why, why are we going through the trouble of doing this? It's the saliva is attempt to number one when it comes to being hypotonic. And I'm going to point to this. Why are we making that saliva? hypotonic? Well, what kind of fluid Do you think we want in the digestive tract? isotonic, right, and the last section we talked about that when we were talking about motility, and how hypertonic and hypotonic came in a small intestine inhibits gastric emptying, we talked about hypertonic hypertonic time in the small intestine, increasing segmentation in the small intestine. And why that was is to try to get that isotype. So that's what we want. So then why the hell is saliva being made? hypotonic? Well, let's think about this for a second. What is saliva combining with as we eat? solutes, right. If you add solutes to a fluid, what happens to the fluid, it becomes more concentrated. So if the saliva was isotonic to begin with, and you add solutes to it, it is by default going to be hypertonic. Right? That's not what we want. If it's dilute to begin with, and we add solutes to it, is it going to be as concentrated? No, it's not. So this is the salivary glands attempt to make a fluid that is dilute because it knows well, it doesn't know. But we're going to add solutes do it because we're going to eat food. Is it going to work? No, it's not going to work. Because the foods that we eat are, you know, very concentrated. So what we swallow is going to be hypertonic. But this is the salivary glands attempt to try to keep it isotonic. So if you look at your notes, I'm gonna show you where this is. So if I can find it. Great. Maybe it's not the notes. All right. Oh, wait. Finally, quiz components slide is hypotonic. You know what's not in the note? So I'm going to write it down. So I'm just going to tell you, I just didn't tell you. So what we're doing here is is that the sword will be will be closer to isotonic. When mixed with food. Again, it's not going to work. But again, it's given it the old college try. Now, why do we have to have the saliva alkaline. And I think I do mention

that one in the notes, but I'll write it down anyway. It attempts to neutralize acids in the in the mouth. Or the acids come from bacteria produce those acids attempts to neutralize acid in

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the Lumina, the mouth. That is good bacteria, actually there's bad bacteria. It's the bad bacteria that are really making more of the acid. And so why do we need to neutralize that acid because that acid eats the enamel of our teeth? Is it going to work? No. That's why we have to brush our teeth at least a couple of times a day to wash away the bacteria. That's why we floss to get rid of things that the bacteria will feed on. That's why we might use mouthwash it kills the bacteria. So if we just relied on our saliva to kill all the bacteria, the the acid in the mouth, we're going to have cavities and no time. That's why we have to practice good oral hygiene. But again, it's giving it the old college try in trying to neutralize those acids that are present in our mouth naturally. So this is what we call the aqueous component, the watery component, there's an organic component to enzyme enzymes, some antibacterials and some mucus. Let's just get the mucus out of the way. We know that mucus is going to be added to all the organs that we're discussing here because we have mucous membrane so mucus will be produced and added antibacterials will be produced and added. So the bacteria in our mouth is being combated by things that we make naturally. Is it going to work? No, but at least the saliva is trying to the salivary glands are trying. IGA is an antibody. There's five antibodies IGA is one that's associated with mucous membranes. And then we have also something called lysozyme. That's also an antibacterial that's also in your eye or your the fluids of your eye, by the way. So those are organic components, the mucus, let me be consistent with this, the mucous and the antibacterials. And then a couple of enzymes. One of them is amylase. And so let's add amylase here. Now it's specifically called salivary amylase. I think it's fairly self explanatory as to why it's called salivary amylase. We're going to see another amylase today produced by the pancreas. amylase. His job is to digest starch. And if you recall from last semesters second lecture, maybe we talked about different kinds of carbohydrates. Polysaccharides being one of them. And starch is a polysaccharide, along with glycogen and fiber. And so this enzyme is responsible for digesting starch, but it's going to do very little digestion of starch. If this enzyme didn't exist, it would not make one little bit of difference because the enzyme produced by the pancreas, pancreatic amylase, that's what's going to take care of everything. When it comes to starch. Then we have one more enzyme here. It's called lingual lipase. We're going to see lipase two more times today. lipase, whether it is the one produced by your salivary glands, or your cheek cells, or your stomach, or your pancreas, digest triglycerides. This particular enzyme, like salivary amylase, doesn't play much of a role at all. So if this didn't exist, it wouldn't make one bit of difference to the digestion of triglyceride. So digests triglycerides. Here's another thing to guys, you're going to see that as we go from Oregon to Oregon to Oregon, and accessory Oregon to accessory Oregon to accessory Oregon, we're going to be throwing all kinds of stuff that you guys, enzyme here enzyme, their bile, here, bile there, this is involved, this is important. So it might get a little overwhelming as I'm throwing all this stuff at you. But I'm going to put it all together. And yet, I'm going to do that by showing you how carbs are digested, you're going to see how all these enzymes work together to digest carbs, and then fat and then protein. Alright, so don't get too panicky when you see all these different enzymes all over the place. The story is going to come together, I promise. But I need to kind of set the table up before I feed you more food here. So this picture right here, these words minus the things that we crossed out, of course, and then that right there. Now what? Well that select the saliva is actually very highly regulated. So the amount that's produced and how fast it's flowing, is

going to be highly regulated by your autonomic nervous system. The parasympathetic is going to play the biggest role. So that's why I'm going to put the word parasympathetic right here at the top. Now if you remember from last semester when we were talking about the autonomic nervous system,

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we always said if this does one thing at this division of the autonomic stimulates the other one inhibits it the sympathetic stimulates the parasympathetic inhibits at the parasympathetic stimulates, the sympathetic inhibits, but I gave you one exception to that rule. And that one exception again, we already learned this, so it's in a synapse somewhere in your brain. salivary glands are stimulated by both the parasympathetic and the sympathetic nervous system. But the parasympathetic plays by far the bigger role, which is why it's only going to be included in this story. So I'm going to draw another salivary gland, which is producing the saliva that we just discussed. And that saliva is going to be dumped into obviously the lumen of the stomach or the mouth. Now, our parasympathetic nervous system will be releasing acetylcholine onto the muscarinic receptors that is going to stimulate to salivary glands. But there are things that can influence the parasympathetic nervous system, there are things that can stimulate the parasympathetic. And there are things that can inhibit, those things that stimulate the parasympathetic nervous system will be those things that make the parasympathetic stimulate the salivary glands more, which means more saliva is going to get produced. Now what kinds of things can stimulate the parasympathetic to once again, stimulate the salivary glands, I have a number of them listed in your notes. So think about food. Put this all as one think about food, see food, smell food. Any of those, you'll already start to produce more saliva, in anticipation of doing what? eating the food. What else. Anything in the mouth, no food in the mouth. So now the food is in your mouth. Now I have food there, you can stick rocks in your mouth. And you will make more saliva because it's putting pressure on the tongue. And so that will cause more production of saliva because it's going to be stimulating the parasympathetic nervous system. What else when you feel nauseated? Now when you feel nauseated, what do you run the risk of doing? vomiting, right the contents of your stomach. I'm going to assume that we know your stomach makes acid if you didn't know that you're going to in just a few minutes and I'm going to show you how it's done. So if you run the risk of vomiting, the contents of your stomach that have acid what you vomit it ends up in your mouth, right? Acid. So why do you think you're going to produce more saliva? To try to neutralize the acid coming from the stomach, do you think it's going to work, there's no chance in hell it's going to work. And so it's still salivary glands are giving it the old college try, it's still going to stimulate more saliva to be produced because that saliva has bicarbonate to neutralize the acid. And then to go along with that, GERD, gastro esophageal reflux disease actually had that right here. And if you remember, that's reflux of stomach contents into the esophagus, and now the acid is here where it doesn't belong. And that's going to signal the parasympathetic. Hey, listen, there's acid in the esophagus, it might be making its way into the mouth. Let's make some more saliva. So we can, again, try to neutralize that acid. So GERD is another thing that will cause more saliva to be produced, to things that inhibit. And when we talk about inhibit, it's not a direct effect. It's an indirect effect through the pairs of everything works through the parasympathetic nervous system. That's why again, I have it right there at the top. So these things that I listen to stimulate the two that I'm going to listen inhibit, do not have direct effects on the salivary gland. Just the parasympathetic does one thing that inhibits his dehydration, when you're dehydrated, your mouth is dry, right? Your parasympathetic is making that happen, why? desperately trying to get to drink fluids, that's the reason it wants you to feel thirsty. Why, so you can drink fluids to

get the fluid levels back up, and they're where they're supposed to be. So dehydration is one. Sleep is another one I had listed. Why sleep because you don't need that much saliva while you're sleeping. Now, when I say inhibit, by the way, now if you're severely dehydrated, you can have the driest mouth in the world. So you can have you know, almost stopped the production of saliva. But typically, you're just kind of slowing it down. Just like with sleep, you're not stopping saliva production while you're sleeping. I'm gonna assume everybody's probably slobbered on their pillow or slobbered on a desk during a lecture, you're still making saliva when you sleep, right? Just not quite as much. If you did, there'd be more of a puddle on the on the desk as you fell asleep during a boring lecture. All right, certainly not gonna lie. So this right here

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is this. That's what we just drew, done with the saliva for now, for now. Now let's go on to the stomach. Nothing is going on in the esophagus, when it comes to producing any type of fluid that's going to digest the esophagus just kind of through way to get us from the mouth, into the stomach, when it comes to the things that we eat and drink. I think I've already mentioned this to you guys, there's very little digestion in the stomach very little. And really, if digestion did happen in the stomach, would make a bit of difference. Your small intestine will take care of it. But we're going to talk about it anyway. So let's talk about those things in your stomach that are going to help with digestion and a couple of other things as well. Mucus being one an intrinsic factor being another which is something you should be familiar with. Because we've already talked about it. Here's the stomach. We're gonna draw a couple of cells here. This is not going to be to scale. This is going to be a cheap cell down here. You guys know what chief sales are. So that's the chief sale. Over here. I'm gonna draw parietal cell. You guys know what that is as well. You're going to spend a little bit more time on the parietal cell after we get through this little discussion. Let's talk about the chief cell first. What does it produce couple of enzymes. Gastric lipase, so there's lipase again, lipase has the same exact job this lipase does as the lipase produced by your salivary glands, and that is digest triglycerides. But like the lingual lipase, that's produced by your salivary glands. This lipase has very little to do with the digestion of triglycerides. If this enzyme didn't work, or didn't exist, it would make a bit of difference. The lipase in the pancreas, that's the one. The chief cells are also going to produce an enzyme that's going to help us digest protein. Now, when it produces this enzyme, it produces it in its inactive form this enzymes called pepsin, but in its inactive form, we call it pepsinogen. And I will put underneath the word pepsinogen. Inactive. Now why would that be? Well, these protein digesting enzymes, these proteases can be pretty nasty. And it's a thing that cells that produce these particular proteases due to kind of its self preservation. If for whatever reason this chief self produced pro He said it couldn't get it out, couldn't secrete it, it would start to eat the cell that cell cells don't want to be eaten. So they produce it in an inactive form pepsinogen. And so at this particular point in time, it does not have the ability to digest protein, but it's going to, because it's going to be turned into something that is active, and when it is, we actually call it pepsin. So how are we going to activate it, and again, this digests protein. Notice that we didn't have any protein digestion in the mouth, because there are no enzymes produced by your salivary glands that are going to digest protein, protein first gets digested in your stomach. And pepsin is what we call a protease. So when you see the word protease, it's going to be an enzyme that digests protein, Ace enzyme Protein Protein ace. Now how are we going to activate pepsin, we activate pepsin when the pH is less than three, which is pretty stinking acidic, and the pH in the stomach can be as low as one by the way. And that can happen and the stomach walls are able to survive that because the

stomach makes an awful lot of mucus and that wall is very well protected and it's very thick, is well, the esophagus in the small intestine, not so much. So let's go back to the parietal cell, the parietal cells are going to be producing acid. And it's that acid, that's going to get our pH to where it needs to be so that pepsin can work. And that's one of the jobs of gastric acid. That is to activate pepsin acid will also kill bacteria that you might have ingested and swallowed, by the way you go ciliary escalator, remember that? All that mucus is trapping, you know, a whole bunch of crap that we're that we're breathing in the air, and then what do we do with it, we swallow it, and it'll end up in the stomach. Plus, it'll rip apart and break down connective tissue in the meats that we eat if we eat meat. So acid is produced, what else is produced, this has nothing to do with digestion. intrinsic factor. Now, we already talked about intrinsic factor this semester, in the blood chapter, we were talking about anemias. Remember what anemia was, that was a P. Er, pernicious anemia, which is a lack of one vitamin

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B 12, right. This particular protein made by your stomach is necessary to absorb

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B 12.

27:43
spell that right to absorb B 12. Now, the absorption of B 12 actually happens in the ilium. But you need this particular protein in order for that to happen. Vitamin B 12 is even put it there in the helium so you don't absorb B 12 with a stone gates down the line, the very, very disappointed the small intestine. So these are the things that you need to be aware of that are being produced by cells of the stomach that are part of the discussion now, and so I'll show you where this stuff is. That's right here. So we just do this. That's what we just drew. Now what now we're going to concentrate on the parietal cells in acid. So now we're going to draw freital. So there's a couple of things we're crossing off here. And it's just how this acid is produced. This is actually a chemical reaction. We'll talk about the acid base chapter just not right now. So what are we going to do now we are going to draw a parietal cells, I'm going to take a protocell out of that wall. Let's stick it right here. So here's our parietal cell, I'll label it in our orient ourselves as well. This is the lumen of the stomach. So right here that I'm scrolling through, is all this that's where the that's where the crime is. All right. Now there is a pump on this side of the parietal cell facing the lumen. It's a transporter that is going to transport the acid into the lumen. And it has a special name, it's called the proton pump. So I'll point to it. So this is the third pump that we've learned about. Over the last two semesters. The first pump was a sodium potassium pump. The second pump was a calcium populate about that in a muscle chapter. And now this third pop and actually had all three pumps listed in chapter three when we were talking about transport. So this proton pump is going to Den secrete the acid into the lumen of the stomach and it's highly regulated. And there are a number of things that are going to regulate this transporter. There's actually five things that I'm going to show him to you now. So we're going to put some receptors on the other side of the parietal cell, the blood side of the parietal cell. So here's the receptor. And that receptor. Let me move the word parietal cell over just a little bit. That receptor is a muscarinic receptor. If you remember from last semester in the autonomic chapter muscarinic receptors bind what neurotransmitter Can you tell me? Cetyl choline beautiful. So that would be the parasympathetic nervous system, releasing acetylcholine onto that muscarinic receptor.

30:29
I'll put parietal cell over here. And what is that going to do as you don't want I'll use some color.

30:38
green for go it's going to be stimulatory. So when acetylcholine binds to the, to the muscarinic receptors, stuff will happen inside that cell and stimulate that pump to transport more acid into the stomach. So all your parasympathetic nervous system stimulates acid secretion. What else so that's one thing. Four more to go. Here's another receptor. This is a gastro No, you know what, let's do histamine. First, this is a histamine receptor. Now, this is not the same histamine that's involved or histamine receptor that's involved in allergic reactions. It's a different receptor. Okay, so what's called an h2 receptor. This is obviously going to bind histamine. And histamine is going to stimulate that particular receptor. And when it does, what's going to happen as a result, stuff will happen inside the cell. That will also stimulate that pump. Next one on the list, and by the way, histamine is a paracrine. I'm going to go back to the beginning of the notes that I'm going to show you where all this stuff is. This is another receptor, it's called the gastrin receptor. gastrin is a hormone, it's one of the four hormones that you need to know there's two paragraphs you need to know histamine being one of them. And what's going to stimulate the gastrin receptor is well, gastrin. And so what's going to happen when gastrin binds to that particular receptor, once again, we are going to stimulate that pump. Number four, another receptor. This is another paracrine receptor, this one's somatostatin. The somatostatin receptor is actually going to be inhibitory. So I'll draw that and read so mad Oh, Stan, the paracrine will bind to it, of course, that'll have an inhibitory effect on the pump. And so it's going to be the interplay between those four receptors, that's going to dictate in part how much gastric acid is going to be secreted into the lumen of the stomach, there's one more thing involved. And that is another hormone. And that hormone is separate. And now, this particular hormone is not going to have a direct effect on the proton pump, it's going to have an indirect effect. It's going to have an indirect effect on gas, you know what I'm gonna use some color again, you use red, because it's gonna have an inhibitory effect on gastric, and if you inhibit gastrin, well, then you will indirectly inhibit the pump itself. So the two s's inhibit, and the other three are going to stimulate. And again, in the end, there's going to be a certain amount of acid that's going to be secreted into the lumen. That would be deemed normal, but it's also going to be dictated by conditions that we're going to be talking about. There's three different phases. Now, let's talk about a couple of other things here before we take a break. Let's talk about a few drugs. These are probably drugs that you've heard of, and I'm going to use the common names for them. Tagamet is one. So you know what I'll do it in blue is to do something different here. So Tagamet Pepcid is another one. Zantac used to be in the same family but Zantac, no longer is around because there was some other ingredient in Zantac that causes cancer. So again, it's been pulled off the shelves that was about dental no sometime during COVID. I don't remember when. But these two by him right a Meyer if you want to when you take them inhibit this particular receptor, and then that's going to decrease the amount of acid that is secreted into the lumen of the stomach. So that is an indirect effect on the pump, but we can directly affect the pump with what are called proton pump inhibitors. And I have a couple of those listed in the notes. One of them is Nexium, by the way, these are all these all used to be prescription now you can get them over the counter. Nexium and Prilosec I believe are the two that I have in the notes. Those will directly inhibit the pump

34:50
and those are called proton pump inhibitors. PPIs Now, why would we want to take these particular? Yes? Specific for the limited? Offer? How do they specifically target those sounds?

35:10
When you say specific for the lumen? I'm not sure what you mean by that. There's other proton pumps and other cells. Oh, I see what you're saying. Oh, yeah. Yeah. Yeah. I mean, would they have other? Would there be a side effect or other effect? Yeah, there's no, there's no magic bullet. Yeah, go systemic. And we'll just start to inhibit those as well. Yep. Good question. So, why would we take these kinds of drugs? Tagamet pepsin, Nexium, Prilosec, why do you think we're decreasing? Obviously, we're decreasing the amount of acid and aluminum? What condition would we? What might we had? Gert? So let's go back. So with GERD, what's happening, reflux. And the problem with GERD is the acid that makes its way into the esophagus. Well, if you don't have as much acid, well, then you're not going to have anywhere near as much damage to the esophageal walls, you might not have much damage at all, depending on the dose that you take at these particular drugs is going to dictate how much acid is within the lumen of the stomach itself. And so decreasing. You know what, I'll do it in blue, decreasing acid secretion?

36:25
Actually, why decrease acid secretion?

36:31
I mean, there are other reasons as well. But this is the most common one. People who have heartburn, for example, will often take these drugs on a daily basis. The answer to that, for example, treat not cure, it's not a cure. Good. Now, do you see a potential problem here? And I use the word problem, liberally? We don't have as much acid in the lumen of the stomach, do you see a potential problem? You're not going to activate pepsin, which means you're not going to digest protein in the stomach. All right. Is that a problem? No. It's not a problem at all. Again, as I said, very little digestion in the stomach. If the stomach doesn't digest protein, it doesn't mean a damn thing because your small intestine will take care of it all. That's where most of the action is happening in the small intestine. I mean, is it ideal for pepsin ought to be activated in the stomach and it's not ideal. But really, in the end, it doesn't matter. But keep it in mind. Okay, what do we do now we take a break, when we come back, we're going to talk about the three phases of gastric acid secretion.

37:47
Okay, let's finish up for tonight. Now, the next thing we're to talk about, are these three phases of acid secretion in the stomach, you're always producing a small amount of acid and secreting a small amount of acid. This story is about when we produce a lot more and why would we do that? Because food's coming. And so we have these three phases. The first one is going to be the cephalic phase. And so what I'm going to do here is I put this together some time ago, so I'm just going to stick with it and not draw. The SIFAT phase is all about seafood, think about food, place food, the food's not in the stomach, yet. It's everywhere, but before it gets into the stomach. So these kinds of things that we have listed here, will stimulate your parasympathetics in an indirect way, but in the end, the parasympathetics are going to be stimulated. And when you stimulate your parasympathetic nervous

system, let's go back to a picture that we just drew. Oh, you know what I forgot to put some in here. Let me add this to this story. My apologies, your parasympathetics Not only will directly stimulate the pump, but will indirectly stimulate the pond because your parasympathetics will have a stimulatory effect on histamine. And a stimulatory effect on gastrin forgot to put that in the story. So the parasympathetic has both a direct effect on the pump and an indirect effect through these other two, the paracrine histamine and the hormone gastrin. Now, this will make more sense when we look at this. So your parasympathetic, you're going to be activated and when they are, well, they're going to activate these muscarinic receptors cause more histamine and gastrin to be released, more gastrin, histamine will have a bigger punch to the proton bomb. So we're gonna get a big stimulation of proton or inside proton secretion, H plus secretion, acid secretion. So if you look at this, and the way I've drawn it, this arrow is pointing to it's supposed to point to directly gastric acid being secreted. So the arrow going to this word gastric acid would be this arrow right here. And then these two arrows right here, this one and this one, which are from these two right here. So this one here This arrow here is pointing to gastric and histamine, you see that? All right, so this picture is just another way of drawing what's going on up here and right here. Now, in the notes, it says 30% of the gastric acid secretion, that's a big increase. 30% increase is even bigger in the next phase. And the next phase is the gastric phase. Now the food is in the stomach. So now you've swallowed. And when you do that, and again, we're talking about a normal sized meal. Again, not just an m&m, but a normal sized meal to where we will actually get distension to the stomach. That is where we start the story distension of the stomach because of who's in the stomach. Now, you got Mikayla receptors in the stomach, they will signal the parasympathetic nervous system. Story is the same. It's the same exact story as it was here parasympathetic does its thing. Parasympathetic does its thing parasympathetic does its thing. And we stimulate more gastric acid secretion, but look at the number. It's 60%. Why is it twice the cephalic phase because the stretch of the stomach, the distension of the stomach is a bigger stimulus to the parasympathetic nervous system. So a lot more gastric acid is going to be released into the lumen at this particular phase. Now what well, stomach is going to start to empty the kind which used to be the food that you were smelling and tasting and thinking about. So we're going to have an intestinal fates, the intestinal phase is just in the very beginning, when the when that time first gets released, emptied into the small intestine, this is only going to involve one hormone and that hormone is going to be gastrin. So when the Chi enters the small intestine is going to stimulate gastrin. And that gives us our last 10%. That makes 100. So what do you need to know cephalic phase and what initiates it and what the percentage is when it comes to gastric acid secretion. And same thing, gastric phase, stretch in the stomach, 60% intestinal phase, caiman and small intestine gastrin. Causing again, a little bit more gastric acid to be produced, or I'm sorry, released into the stomach. That's that. Now what,

42:23

now we go to the small intestine. More stuff, you get the cross out the stuff that we'll cross out on the screen here, it's all anatomy, you guys learned it, I have it in the notes just to remind you of how in the small intestine, there's a huge increase the surface area right like 600 fold increase the surface area, given the way that it's put together, we are going to concentrate though on the villi. So I'm gonna draw a picture here. Your small intestine makes some enzymes, those enzymes aren't floating around the other enzymes that we've talked about, and we'll talk about are actually in the chyme are actually in the saliva itself. These enzymes that I'm going to show you here are not floating around. They're stocking on the microvilli. And so what I'm going to do is, I'm going to draw this right here. And in order to do

that, I'm going to draw a bill is one single villas you guys know the villas is? So here's our villas. I'll label it. I see give it two dimensions. And so that's the wall of the villas right there. And that wall is made up of individual cells. So there's a cell, there's a cell, there's a cell, there's a cell, there's a cell, these are all cells. Now, why did I draw them all wavy like that? Those are the micro villi. You guys know what those are? So micro villi. That whole thing is one villas. And so now what are we going to do? We're going to take one, and this is an epithelial cell. That's one single epithelial cell that makes up the walls of the Belli, or the villas Yeah, belie. So let's just magnify one of these. Let's take that one. And let's make it bigger. So let's draw that cell right there. So here's our micro belie, which increases our surface area. Now, all of those microvilli are enzymes and they're stuck. They're what enzymes that right there is going to be a protease, it's like pepsin, the job of that particular enzyme is to digest protein. And then we're going to have three others there's actually more than three, but we're only going to learn three. We're going to have one here, I'll make it red. By the way, this red button has nothing to do with any of this stuff in the stomach story. I just wanted to make these different colors, and green. Again, they're produced by those epithelial cells and then they just get stuck on the micro villi themselves. Those are called disaccharide asis. And their job is to dice digest disaccharide ads. Now what are the disaccharide? Well, if you don't remember, I'll just mention them to you maltose, sucrose, lactose, they're all carbohydrates. And so these are specific disaccharide for specific, or disaccharide Asus, I should say for a specific disaccharide. And so we'll name them. So the red one, I'll just say that that is mall, tastes maltase His job is to digest maltose. I'm going to show you that today. And then the blue one, we'll call that one su Crace. The job of sucrose is to digest sucrose. And then the green one. That's lactase, the job of lactase is today digest lactose, which is another disaccharide. And so, for right now, that's all we need to know more on this at a later time. Now your small intestine oops, oops, again, also has a couple of things. Mucus, of course. And then another type of enzyme that's that's produced throughout called lysosomal enzyme. What this enzymes job is, is to keep the kind soupy, we don't want the kind to get too chunky, because it's going to be a little bit more difficult to move it along. And so this particular enzyme, again, does just that maintain some fluidity of the car. That's that for the small intestine for now. Now, the large intestine now, books will tell you websites will tell you that there is no digestion in the large intestine. And that's kind of true. We as human beings do not digest anything in the large intestine, we don't have anything there to digest anything. But we do have our friends, Flora, the good bacteria, which is actually throughout the entire digestive tract. Even in the mouth, you have Flora stuff very much of the most of the floors in your large intestine, it's not even close. And this is something that we talked about last semester, if you remember, I think it was the second or third lecture of last semester. When we were talking about carbohydrates. Actually, it might have been the first lecture on the remember.

47:16

Soluble fiber, which is fiber that we ingest, at least hopefully we're ingesting it is digested by the good bacteria in our gut, specifically the large intestine. And when it digests that soluble fiber, we get something wonderful. And that is a short chain fatty acid, actually three of them. And those are the three, if you remember, it's a saturated fatty acid. Remember, when we had this discussion last semester, there was a second lecture have fantastic anti inflammatory properties. It's another reason to get the fiber in your diet, especially that soluble fiber. And so now we have these anti inflammatory molecules within the large intestine. And that's good, because it allows you to see diminish inflammation, which can then decrease the risk of developing cancer, like colon cancer, for example.

Also, these will be absorbed into the blood. And so now you have these short chain fatty acids circulating in your cardiovascular system, decreasing inflammation within your cardiovascular system, which is also obviously fantastic. So know that there is digestion in the large intestine know that we don't do it, but are very good friends to flora. They do it for us only if we give them the food that they want. And they really liked that soluble fiber. So that's that. Now what, now let's talk about some more accessory organs. The three that we have left are the liver, the gallbladder and the pancreas, I'm going to take the liver and the gallbladder gonna talk about them together. Now, your liver does a whole bunch of stuff, it's got at least a couple of dozen jobs. This is one of them. And that is to produce bio, by the leader of it a day, give or take. There's a bunch of stuff in bio, I've obviously crossed off most of it, there's really only two things I need you to be focused on when it comes to the contents of bile, and that is bicarbonate, and bile acids, bile salts, and so we'll do this. So I'm going to draw the liver here. Oops. And I'll label it. So that's going to be the liver. And it'll be here. I'll just put the word bile and then I'm going to put the two things that you need to be aware of bile acids. salts, so bile acids, bile salts, and bicarbonate. Again, there's a bunch of other stuff in bile, but these are the two things that you need to be aware of. Now, what's the big deal with these two? And what is their function? So the bile acids, the bile salts are going to help us digest fat, we're going to actually do something called emulsify fat. I'll show you what that is in the next lecture. So they emulsify fat digest. So this is going to help with digestion, specifically a fat and then the bicarb. That's present. Think we already know what this one does, it's going to neutralize the acid. And we know how important it is to neutralize acid, we neutralize the acid in the small intestine is going to protect the walls. It's a small intestine, of course. And it's going to allow the enzymes to work in the small intestine, there's going to be a mess of, we've already seen a couple of these enzymes aren't going to work if we have an acidic environment and all the insights from the pancreas are going to come along and they won't work unless we have a neutral environment. So the acid needs to be neutralized. Now, your liver is constantly producing bile it produces more in the morning, not quite as much as the day goes on. But it's constantly producing bile. And it's going to deliver that bile. And I know this is not anatomically correct, but I don't care.

50:58
There's our gallbladder.

51:00
And that Bile is just going to trickle into the gallbladder. I'm just going to put the word bile here and what is going on, what's it doing there, it's being concentrated and stored in the gallbladder. And eventually, it's going to make its way to the small intestine, of course. And that's the story that we're going to tell in just a little bit. So this is what you have to know about bile. This is what you have to know when it comes to their functions. And we're going to repeat the functions in just a little bit. Now, before we move forward with the pancreas, other functions of the liver, the one I just told you is it produces bile. Then we have a list of others. Now I told you that the liver has over two dozen functions, and I only have maybe half of those up on the screen. The reason I picked these is because you've learned about pretty much every single one of these already. We learned about Blanco Genesis last semester, we learned about glycogenolysis last semester that last semester, that last semester, that last semester, that one last semester, that one this semester phagocytized is bacteria. That was the blood chapter, that big picture we drew about bilirubin and stuff. That's that one right there. DM and Ace amino acids last semester converts ammonia into urea last semester, all in the first chapter, by the way. So that's

the reason I picked these. Because they're already in your noggin, they're in there someplace, you just have to tease them out at some point. Now there's a couple in here that have yet to be mentioned that will be mentioned later on in this particular chapter. And that would be removed nutrients from the blood add nutrients to the blood. Those are the only two that we have yet to learn now and store some other vitamins. Although I told you about beet 12 Earlier on when it came to Pernicious anemia when we were doing the blood. So on the exam, I promise you it will be a vomit question as I like to call them straightforward as hell no critical thinking if you know what's going on with the liver, you can't get it wrong. It's easy money. Why if I'm a question is, here's a question vomit out the answer back me you don't even really have to think about it. That's what I mean by a vomit question. So that's that for the for the liver. Now what about the pancreas more stuff that we get to cross out. So the pancreas, like the salivary glands produce a fluid that has an aqueous component and an organic component. We're not going to spend the kind of time we did with the pancreatic aqueous component as we did with the saliva, not important. This is what you're going to have to know when it comes to the pancreas. So here's our pancreas. You're paying for pancreas is going to produce a bunch of enzymes and also bicarbonate. And bicarbonate is going to have the exact same role that the bicarbonate and bile has neutralize acid and this bicarbonate and all these things that are in the pancreas will eventually make their way into the small intestine. Of course they will. Now what about all the enzymes, pancreatic amylase. Now we've already seen amylase amylase is produced by your salivary glands is called salivary amylase. And as I told you, doesn't do much of anything. This is the one that's going to do the bulk of the work when it comes to digesting starch. What else pancreatic lipase we've seen light paste twice already. This is our third time the first time we saw it lingual lipase, my bear salivary glands. Chief cells make lipase, those two don't do much of anything. This is the one that's going to do the bulk of the work when it comes to digesting triglycerides. We're also going to have those that digest fat or another fat I should say cholesterol esterase. That's obviously going to digest a form of cholesterol. And then we're going to have a number of pancreatic enzymes are going to digest protein. And I had the names listed in your notes but what I'm going to do is I'm going to give you a little bit of a break I'm just gonna say pancreatic proteases. There's three of them that I have listed in your notes. trypsin, como trypsin, carboxy peptidase. There's actually a couple more. And so that is here. So what we're just writing down here is this. What you don't have to know are the specific proteases. I know something is too terribly important. All right. Now what are we going to do? Now what we're going to do is we're going to combine the pancreas, the liver and the gallbladder along with the small intestine with a little story. I'm going to show you how these enzymes and how the bicarb and how the release of these particular fluids from the gallbladder and the pancreas are going to help us do what we need to do when time is presented to the small intestine. CCK is going to be in this story secretin is going to be in this story, these enzymes are going to be in this story bio is going to be in this story. And this story is not really written in your notes in any one place. It's written in a number of different places. So

56:00

what are we about to draw, we're about to draw these words right here. That's going to be part of the story. This is going to be part of the story, these two lines here released, stimulated by secrets and CCK potentiate the effect and release stimulated by Colusa Sakai and so it's all kind of scattered, I don't know if that's one page or two pages, or how many pages it is. But we're gonna put some stuff together. Now, just as I said, we're gonna start to do throwing all this stuff at you. But we're going to put it all in one little story or little stories, I should say. So let's do this. So I'm going to draw the liver, the

gallbladder, the pancreas and the small intestine. So here's our liver. And again, bile being produced by that liver constantly. And so I will once again put the word bile there and our bile acids

56:54
and bile salts. They work together. bicarb, of course.

57:05
And we need to put our gallbladder in the story.

57:10
And so let's do that. And so here's our gallbladder. I'll just yeah, just write it up.

57:17
And now, the small intestine as well. Now, I know what I'm drawing is not anatomically correct. But the picture would look ugly as hell, if I did draw it anatomically correct. I know that two of the comes from the gallbladder is going to meet the two that comes from the pancreas at the sphincter of OD, I'm not doing that. You'll see why it just got too much stuff the right in this picture. Pancreas is going to be over here. There's the tube that's supposed to meet with the tube over there from the gallbladder, but it's just not going to. And so there's our pancreas, I will label it, we will then put in the pancreas, those pertinent things that are going to be part of it's fluid. And all I'm going to do here is write the word enzymes. So bicarbonate, and enzymes, well what enzymes, all these, all of them. pancreatic enzymes are pancreatic amylase, cholesterol is straight buttered out and added up. That's what I mean by enzymes. I wanted again, this is of course, the small intestine put that here. duodenum specifically, but I'm just going to put small intestine that's good enough. So let's story begin. Oh, and by the way, this is going to be continuously being produced and trickling into the gallbladder. So I'll just kind of put that there as well. Now, let the story begin. So you've just eaten a meal. And that meal has made its way to the stomach. And now the stomach is going to empty it into the small intestine. And what's going to be emptied into the small intestine is kind of that kind is going to have acid in it. Of course it is that kind will have fat lipid. That kind will have protein now that kind of will also have cars, but carbs don't specifically cause the stimulation of any one type of hormone. But these three things do, which is why carbs aren't in the story here. But certainly they're going to be taken care of by enzymes in the pancreas and the small intestine, but they're really not part of this specific story. So number one, those are now present in the small intestine. They got there because his stomach emptied them into the small intestine, they're part of the con. Now that acid is going to be sensed by chemo receptors. And it's going to cause the release and this is going to be number two of a hormone and that hormone is secret. And this is a hormone that was in the beginning of the chapter. We just mentioned secret 10 A little bit ago in the gastric acid story All right. So that's one thing that's going to happen. All three of these will cause the release of another hormone. And it's called the sister keinen, C, C. K, that's also number two, that's going to happen simultaneously. Then what? Well, of course, these are hormones are going to be dumped into the blood, they're gonna go to their targets, target tissues, well, what are the targets, secret 10 has a target, it's going to be the pancreas, specifically bicarb is going to be what it's going to be telling the pancreas produce little bit more of that, that's going to be number three,

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there's going to be a stimulatory effect there. CCK is going to go to the pancreas as well.

1:00:48
This will be number three, it's going to be eyeing those enzymes instead, that will be number three, that is going to be a stimulatory effect. At the same time, while secretin is over there by the pancreas, it's going to help secretin. Now, when it comes to causing more bicarb to be released, I'll show you again where all this is in your notes. So that's also with three eyes, you don't like that dotted line very much, I'm gonna draw it again. Because all my pictures are so neat. So that's what that's talking about. So I'll even write the word potentiate there. I'll show you where that is in the notes. What else is going to happen? Well, secretin has another target. And that target is going to be over here. When you hear, see secret 10 I want you to go bicarb immediately. Now it certainly has a role when it comes to gastric acid, but its main role is to get bicarbonate, the small intestine to neutralize acid, that's also number three.

1:01:58
Another number three

1:02:01
is CCK. Its target is the gallbladder. And what it's going to do to the gallbladder, it's going to cause contraction. And when the gallbladder contracts are Bile is going to make its way into the small intestine. And that's going to be number four. And of course that bile has in it bile acids,

1:02:26
bile salts,

1:02:28
and it's going to have by card. Number four is also going to be getting that fluid from the pancreas into the small intestine, which also contains bicarbonate, and a whole mess of enzymes. And so what we have here is this beautiful reflex that occurs, we have something going on in the small intestine that needs to be taken care of. And once that we need to neutralize the acid, we need to digest the lipid, the protein and carbs are going to be there as well. And how are we going to do that I just showed you. And it's mediated by these two hormones. And so this is showing you how all this stuff starts to work together. And so the bile acids and salts are going to help with the digestion of the fat, the bicarb from both the bile in the pancreas will neutralize the acid and then the enzymes from the pancreas will help digest that protein. There's other things going on too, of course, because we have these proteases that are on the walls of the small intestine, they'll certainly be part of the story as well as I will include when I talk about specifically the digestion of protein. So when it comes to this picture right here, once again, where do we find it in the notes well released stimulated by Colleen sister kinda when I'm scrolling through right there that that's CCK stimulating the enzymes. That's what that is. What else release stimulated by secretin CCK potentiate. The fact so these two lines right here. It's this number three was secretin. And this dotted line was CCK. Helping again, bicarbonate pancreas, what else going back here, by releasing the liver, the gallbladder, totally sister kind of causes the gallbladder to contract. That's all this stuff over here. And there you go. Now what, let's put some stuff together. Now as I told you, I would let's talk about how cars digested then protein, we will get the proteins and I will have to

finish that in the next lecture. So let us draw that. And by the way, I also put together a little flowchart that flowchart right there that you see are all these words, and it's going to be the pictures that I'm about to draw for you guys. So you are going to eat some cards. Now what we'll do also here is is that I'm going to put some symbols, circle is going to be glucose. Let's do some colors here. And now we want to colors. A square will be fructose. Should I move that over a little bit more? Actually, I'll put it over here. That's as good glucose. A square will be fructose. These are monosaccharides, by the way, not disaccharide model. So this is what we want to get our carbs down to these three molecules so we can absorb them. So the first thing we're gonna start with in this story is starch. Starch is just a whole bunch of glucose molecules strung together. 2468 10 Good 12 We have an even number. We want to digest starch. Well, how do we do that? Well, we already know, salivary amylase produced by your salivary glands, that's going to be happening in the mouth, but very little. And then the big dog comes pancreatic amylase that's going to be happening where in the small intestine, even though the pancreas is releasing it is obviously going to make its way to the small intestine, small pancreas. Small Intestine. And so what's gonna happen, we're gonna break our boss here. Now, is this exactly the way it happens? Yeah, but no, it's not always just two glucose molecules that we're gonna get. But that's good enough for this course. And so now we have these molecules. So no longer do we have starch, we have something smaller than starch 2468 1012. Well, what are these molecules called? They're called maltose. maltose? is two glucose molecules stuck together? Well, that's still not small enough to be absorbed, we need to break those down. And what's going to do that for us? Actually, I am going to lose color. Now. What color was my maltase? It was red. So maltase, an enzyme that is stuck to the epithelial cells. So of course, this is going to happen in the small intestine is going to break those bonds.

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They're there. They're there. They're in there. And

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now what are we going to get individual glucose molecules now, we have a molecule that is small enough to be transported

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2468 10 and 12.

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That's if we starch. Now, can we eat other cards, of course we can. We can eat things, we can eat foods that have maltose in them, and not just again, starch. So I will put the word maltose. Here, I will draw maltose, which is just two glucose molecules stuck together, we already know that maltase will digest that for us. And of course, it's in the small intestine. We'll just repeat that. And the same outcome is going to happen to glucose molecules. We have our model saccharides. Well, there are other disaccharide ZZ that we ingest sucrose, table sugar, the sugars that are added to foods for example, that's a glucose molecule and a fructose molecule stuck together. We know how we're going to digest those. I believe that one was blue, sucrose. And that's also going to be in the small intestine. And what we're going to get in the end, because we're going to break the bond right there. Oops, let's do it in blue. Crushed. We're going to break that bond. We'll be here we're going to break this bond. And what

do we get a glucose molecule and a fructose molecule good. Now we can absorb them and and last but not least, let's have some milk and some cheese. We're going to ingest lactose, which is a glucose molecule and a galactose molecule. lactase is what's going to break that down for us. And of course, that's going to happen in the small intestine as well. I'll have to put it on this side. And what we're gonna get is a glucose molecule and a galactose molecule. There you go. So again, we're starting to put it together now. Now, before I let you go tonight, I'm going to tell you how lactose intolerance works. And actually, there's a bunch of people, more people are lactose intolerant than people aren't that as adults are lactose intolerant. So why would you be lactose intolerant? The problem is you don't have lactates. That's what lactose you're intolerant to lactose, because you can't digest it. So lactose intolerance. I'm going to show you how we develop the symptoms of lactose intolerance. And so our problem is, you lack lactase. Now, this is what should happen under normal conditions. If you're somebody who's not lactose intolerant. Fortunately, I'm not lactose intolerant. I get to drink my milk and eat my cheese. This is how my body works. So that is going to be the digestive tract, specifically the small intestine, but I'll just put digestive

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tract. No, I'm going to put the small intestine let's be specific.

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So there's our small intestine on those cells should be lac lactase. And it will be initially where to put the blood in his picture to shorten it up just a little bit. So here's our blood. So that's capillary Of course. So what's the story begin? So lactose. Now, what should happen to lactose? Well, it should be digested by lactase, into what glucose plus galactose. And those are mono saccharides. Those will be small enough, I'm not going to put the plus sign there, those will be small enough for us to do this. absorb them into the blood. Now, it's not exactly a direct line like that, we'll see exactly how it happens in the next lecture. So that's what should happen, we should absorb those into the blood. And as that lactose makes its way down, it gets broken down, and boom, into the blood it goes. And so as we do that, we keep the Chi isotonic, because we keep on getting rid of solute. But now we're lactose intolerant. And if we're lactose intolerant, well, we don't have that. And if we don't have that, so number one, we don't have lactase number two, we can't do that. Number three, that ain't happening. Number four. As a result of that, the lactose is going to stick around, and a lot of it. So we're going to have high levels of lactose within the digestive tract, we are going to start to concentrate the kind. And so as a result of that, number four, and by the way, this ain't happening. So we'll just make that three as well. So as a result of that, we have a hypertonic time because we can't get rid of the lactose. Well, our blood shouldn't be isotonic. So you tell me.

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Do we have a gradient?

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We have an osmotic gradient, right? Tell me which we want is going to move. It's gonna go into the digestive tract. Correct. So number six, we're gonna get water in the digestive tract. And so as a result of this, you have lots of fluid now in the digestive tract.

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And when you have lots of extra fluid in the digestive tract, well, you have yourself diarrhea. It's called asthma, you can get diarrhea for a number of different reasons. You get diarrhea because you got like some pathogen within the digestive tract, like food poisoning, for example. You can have what's called osmotic diarrhea. It's got nothing to do with the pathogen. You didn't eat poisoned food. Well, it was poisoned to you because it had lactose in it. So this will develop depending on how much you know lactose you've ingested. But certainly this is one of the reasons that these kinds of things happen when people are lactose intolerant that then again, ingest lactose. The other thing that happens as well is is that the bacteria, the flora that's within the digestive tract, the flora feed off of the lactose and produce lots of gas. So with lactose intolerant, what do you get? You get bloating, you get pain, you get diarrhea, and this is the reason. Okay. All right. Two minutes left. There's no reason to start even begin to start protein digestion. We'll do that on Tuesday. We'll finish this chapter in like a half an hour on Tuesday, and then we'll move on to some goods, some other good stuff

# Digest PM 4-19-22

Fri, 4/22 4:22PM • 31:05

**SUMMARY KEYWORDS**

epithelial cell, small intestine, digest, absorb, cholesterol esters, draw, cholesterol, monosaccharides, amino acids, talking, triglycerides, fat, micro villi, capillary, digestion, molecules, absorption, protein, bile acids, transport

00:01

Alright folks at that time. So, at the end of the last lecture, we were talking about digestion, we got done discussing carbohydrate digestion. So we're gonna take our cards, we're gonna digest them down into monosaccharides. That's the last thing that we did. Now we're going to do protein. So let us digest protein. Now, when it comes to protein, we're going to digest protein into try peptides, die peptides and amino acids. So, let us take this and just draw a very, very simple picture of protein digestion. Now, protein digestion is going to start to stomach carb digestion, if you remember, starch starts in the mouth continues on then, in the small intestine does not happen in the stomach, that's carbohydrate digestion, protein digestion is going to start in the stomach and then continue on in the small intestine. So the circle is going to represent an amino acid. And I'll just going to draw a very simple protein, just going to string some amino acids together here. And above it, I'll just write the word protein. And so let's start digesting the protein. Now what's going to digest the protein well in the stomach protein is going to be digested by a protease pepsin. And so let's write pepsin here. And let's put in parenthesis stomach that is at the pH is less than y three, so it's got to be an acidic environment, the stomach for pepsin to work. And then we have micro villi proteases. I'm going to remind you what that is. And that's a picture that we drew in the last lecture. And that would be this one right here. So these enzymes right here, the proteases, that are on the micro villi, within the small intestine, these are going to grab the proteins as they float on by dissect them. And of course, that's going to be happening in the small intestine. And then we have other proteases from the pancreas. So pancreatic proteases. Again, all of these are proteases. And, of course, they're going to be delivered to the small intestine. So protein digested in the stomach protein digested in small intestine most happening in the small intestine. And so what's going to happen to this protein, we're going to start to chop it up. So we'll chop it up right there, and they're in there, let's die, or let's digest, they're there, they're in there. And so they're gonna get in the end, we're gonna get molecules that are three amino acids big. And some molecules that are two amino acids, big couple of them. And then some individual amino acids, those that are three amino acids big we call a tri peptide. And that's good enough in di peptides here. That's good enough. And of course, we have our amino acids. And there you go. That's that. So again, what do you need to know know what specific enzymes are doing the digestion and know exactly where the digestion is taking place, just as we saw with carbohydrates. Now let's move on to fat. This is going to be a more complicated story. That's the hardest thing to digest. We already know that talked about it when we were discussing gastric emptying. I don't know maybe two lectures ago, one lecture ago, I don't even remember. And so let's go to the words on again, here's a little flowchart of what we just drew. And now, fat digestion. Fat

digestion is going to start in the mouth with lingual lipase move on to the stomach with gastric lipase, but most of the digestion, of course, will be happening in the small intestine, this is where most of the action is going to take place. So what I'm going to do first is the digestion of triglycerides by themselves and cholesterol esters by themselves. And so let's do that. So I'm going to draw a triglyceride. And if you recall, a triglyceride is a glycerol molecule with three fatty acids attached to it. So here's the glycerol. And then we have these three little fatty acid tails. And we're going to digest this and we're going to digest it with lipase. And we have lipase made by your salivary glands. That's lingual lipase. And so next to that I will put mouth and then your chief cells make a lipase. That's gastric lipase. Chief cells are cells of the stomach, so I put stomach next to that. And then finally, the lipase, that's going to be doing most of the work, pancreatic lipase, which of course is going to be delivered to the small intestine. And what is it going to do to the triglyceride, we're going to chop it off there. I'm going to chop it off there. And what we're going to get in the end is a mono glyceride glycerol molecule with just one free fat or a free fatty or I'm sorry, a fatty acid attached to it. So that's a mono glyceride

05:00

And then a couple of free fatty acids, three fatty acids, and they're called free if you recall, because well, they are not attached to any other molecule. So that's the digestion of triglycerides. Now let's talk about cholesterol, specifically cholesterol ester. And so I'll write that down here. Now, I've never used the term cholesterol Ester in the past, when we did talk about cholesterol last semester, I just called it cholesterol. But what a cholesterol ester is, is a cholesterol molecule with a fatty acid attached to it. So glycerol is not the only molecule that has fatty acids attached to it. So this cholesterol, and we're going to digest this now this is only going to be digested in one place as far as we know. And that's with cholesterol Ester ace. And, of course, that is a pancreatic enzyme, which means it's going to be delivered to the small intestine. So this digestion is going to happen in the small intestine. And what are we going to get in the end, we're going to get an individual cholesterol molecule and a free fatty acid. And so we just digested that. Now, a more complicated store. So what I have up on the screen, again, are these individual molecules, what I'm going to do here is, I'm going to call this animal fat. And when I say animal fat, I'm talking about fat that would be on a steak, a pork chop, chicken, that kind of thing. Now, what that animal fat is made up of, as far as we're concerned, is going to be a bunch of triglycerides, and a bunch of cholesterol esters, just a ball of fat is what this is going to be. And so what I'll do here is I'm just going to draw some triglycerides and some cholesterol esters just as we just drew them a second ago. And there's just a bunch of them. Now, you might be thinking to yourself, well, all that's going to happen here is that our lipases and our cholesterol esterase, they're just going to come in here and you're just going to digest all of this, this triglyceride and cholesterol Ester until we have once again monoglycerides, three fatty acids and cholesterol molecules, well, that's not the way that it's going to work. You got to imagine this is a three dimensional structure, it is a ball of fat. Now on the outer edge of that sphere of fat, these enzymes can probably squeak their way in there and do a little bit of digesting but how are we going to get to the inner core of the fat. Now, that would take for ever, for these particular enzymes to just kind of sneak their way crawl their way through. Because of that, because it's so inefficient, that's not the way that it's going to work. What we have to do first is emulsify this, that's a term that I've already talked about. And we're going to emulsify it with bile acids, bile salts. And so this was one of the two components I needed, you guys to be aware of that is part of bile among a bunch of other things that we crossed off. And so we are going to do something called emulsify that fat. And so in the end, what's going to happen is the bile acids and the bile salts, and I'm

going to just have them be this green little thing over here. So that is a bile acid, a bile salt. What these bile acids and salts are going to do is they're going to rip this animal for fat apart and isolate the cholesterol esters and the triglycerides so that they're all by themselves. And so in the end, what it's going to look like is it I'm not going to draw them all, I'm just going to draw one triglyceride. And it's going to be a whole bunch of bile acids, bile salts surrounding it, they went in there, and like little Purana just ripped apart that animal fat. So we had these individual molecules that we can now digest. And so I have to also draw a cholesterol Ester over here. And that's going to be surrounded by these bile acids, these bile salts now I'll show you a more colorful picture that's on pilot. And this is a digestive process. And I told you when we were talking about bile, that bile acids and salts are involved in the digestion of fat. Now emulsification also happens in the stomach by other things that we're not going to talk about. What you need to know is emulsification and as far as we are concerned small intestine. So I'll put that here because as we know the bile is going to be delivered to the small intestine from the gallbladder. So I'll show you a more colorful picture that's on pilot

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and that would be this. So there's our big old ball of fat, a bunch of triglycerides and cholesterol esters and now what happens is these bile salts come in and just Like to rip it apart into smaller and smaller and smaller pieces until we get down to what we call fat droplets. And the fat droplet is these fat droplet fat droplets, individual triglycerides, individual cholesterol esters. Now what's going to happen? Well, now the cholesterol esterase is going to come in and start to digest this. pancreatic lipase is going to come in and digest triglycerides. And now we've just digested fat into its simplest form. So that's the digestion of fat. The words that go along with that are of course, here. And then I do a little flowchart here. Another way of looking at it. So three different ways picture I drew the flowchart and the words that we have on the page. We just digest. And by the way, why are we doing this, let's make sure that we understand why we digesting the things that we ingest, the reason that we have to digest the things that we ingest is because we need them in their simplest forms carbs, monosaccharides, glucose, fructose, galactose, proteins, simplest forms tripeptides, died peptides and amino acids, that's tripeptides. And di peptides are not the simplest forms, but they're small enough to get the job done. And then fats, fatty acids, monoglycerides, cholesterol, they need to be small, why? Because we need to be able to absorb them. And that's the last thing we're going to talk about. So now we're on our fifth process, we've done four, we're on our fifth one, and that last one absorption and what is absorption, this is the lumen of the digestive tract absorption is the epithelial cells that make up the walls of the digestive tract are going to transport the monosaccharides tripeptides, peptides, amino acids, fatty acids, boundless rice cholesterol, and then eventually they're going to make their way either directly into the blood or indirectly into the blood. So let's talk about absorption. And we're going to tackle it the same way. We'll start with monosaccharides. So let's absorb monosaccharides from the lumen. And we're going to concentrate on the small intestine because that's where most absorption is going to occur. Of course, we already knew that. And so now what we're going to do is this. So before we move forward, absorption of nutrients, very little absorption in the stomach, I've already told you guys that most absorption is going to be happening in the small intestine surface area, the small intestine is humongous, it's 600 fold bigger than its than it would be if it didn't have that architecture that it does. And that architecture is what the circular folds to belie the micro villi, you're not going to have to know for my exam, you certainly needed to know it for lecture receiver. I just added it just to remind you the surface area in the small intestine is immense. And so let us move forward and let's talk about the

absorption of monosaccharides. So let's draw this. So we're going to do is this I'm going to draw villas. And I'm going to draw up the vessels within the villas as well. So this is going to be a villas. And I'm going to put the epithelial cells that line the wall of that villas, and we know that those epithelial cells have micro villi. So that's why I've drawn it wavy like that. That's the microbial I have drawn a picture like this already when we were talking about the enzymes. And so there's a cell, there's a cell, there's one, there's one, there's one, so we have a whole bunch of cells here. And then what we also have are vessels within the villas itself. We have blood vessels, capillaries. So there's our capillary. And then we have a lacteal. So I'll label it. So there's our lacteal. There, of course, is our capillary. I'm going to go back to the first picture that I showed you guys in this particular chapter. So capillary lacteal. There's our villas. To go back to this picture right here. When I drew my office, this one right here, there's a capillary within the villas, there's a lacteal within the villas, and that's where I'm going to draw, I'm just going to draw them one on top of another, you're not really like that structurally, but it just makes the picture a little bit prettier when I do. And so let's absorb cards. Now, the way I'm going to approach this is that this way, let's take this area right here. Oops, we'll do it right. So we're gonna magnify this, and just a little area of it. So I'm just going to take one of the epithelial cells and magnify it. So let's take that up at the ileal cell right there. And let's put some vessels there as well. So Let's magnify.

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So here's the micro villi. So micro villi, that epithelial cell. And then we are going to have, again, like I said, I'm going to put one on top of another there's the capillary. And there's the lacteal. So that's going to be the way that we're oriented and of course This is the lumen over here. This is where the chyme is, this is where all the stuff that we just ate and digested into the simplest form, that's where it's located. And it's going to be absorbed over here at the apical membrane, right here at the apical membrane. And so let's draw one of these on the next page. And let's see how we're going to absorb monosaccharides. It's very simple. So here's that epithelial cell again. And we're going to keep this simple. I know in your notes that I have secondary active transport facilitated diffusion as transport processes that are involved here. Don't worry about it. What I draw here up on the screen is what you're going to have to know. So let us put a transporter here. Now, there's actually more than one transporter that's involved in this particular story, we're not going to worry about it. So that is a transporter, and his job is going to be to transport the monoglycerides, or I'm sorry, the monosaccharides to the lumen. That's what this is right here. In this direction, so glucose, fructose, and galactose, which are monosaccharides. Each of those processes, right, there is absorption, and I will right absorb. So we just absorbed them. And it's just transport process that removes them from the lumen and takes them inside the epithelial cell. From there, there's going to be another transport process where now these molecules will end up in the blood good, because now we can use them. We don't do this, we might as well have not eaten them. They're going right through us. So now we have the glucose. We had the fructose, and we have the galactose in our blood. And where is it going to go? Or where are they going to go? I should say they're gonna go to the liver first. The liver will actually convert all the galactose into glucose, most of the fructose into glucose deliverable, use what it wants, and then dump it right back into the blood so that the body gets the rest. So liver gets first dibs on these things. So there you go. That's as simple as it is, again, a little bit more complicated, because there are other transporters. We're not getting into it. What about protein? Well, it's gonna look to say, so our protein digested products, amino acids, type peptides, tripeptides, there are actually four different transporters involved in this particular story, we're just going to turn it into one transporter. So there's

our epithelial cell, we're just going to put one transporter here. And then we're going to put our blood vessel in our lacteal. And so let's absorb once again, here's the lumen. And we're going to have amino acids. And we're going to have dye peptides and try peptides. Oops, draw it the same way I did the other one. So amino acids transported into the epithelial cell. Dye peptides transported into the epithelial cell in the tripeptides as well. That is a teeny bit more detail in the notes where I talk about how most of the DI peptides in the tri peptides, once they make their way into the epithelial cell are actually then digested into amino acids most of them are now I don't care, you still gotta have some type peptides and die peptides about 10% of what we absorbed here on by the way, absorb absorb absorb, variable important that we do. Because if we don't, again, you might as well not be needing to stop. And then these are going to go directly into the blood. And that's going to be an important point to remember. So amino acids, I'm just going to call them a die peptides just die p and then tripeptides stripy directly into the blood monosaccharides directly into the blood. And of course, then off to the liver, they go deliver gets first dibs, it takes what it wants in the body gets the rest. The story is going to be different with fat in the story is more complicated with fat and it's more difficult with fat, that is the fat digested products monoglycerides three fatty acids and cholesterol. And so let's let's see what this story is about. The lacteal is now going to come into play. So we're going to draw our epithelial cell again, we're gonna talk a little bit bigger because we're going to have some action going on within this epithelial cell. So here's our epithelial cell, you're going to draw a bit bigger

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microvilli no transporters. In this particular case. You're going to tell me the transport process up to this point I haven't asked you or to know what the transporter is and what the transport process is. It's in the notes but you don't have to know it for this I want you to so we have our digested products. digested fat products and what are they monoglycerides cholesterol, and some free fatty acids. Now, I'm only drawing one of each, but certainly there's a whole mess of them. So we have these products. Now, these are fats, these are not water soluble. In the fluid in the chyme ms, mostly the fluid is water. So what that means is that these have to be transported around monosaccharides that were in the lumen, those are water soluble, they don't have to be carried amino acids, tripeptides de peptides, they're water soluble, don't have to be carried around. These have to be carried around. And what's going to carry him around is our old friends, the bile acids and the bile salts. And so what does this mean? It means that bile acids and bile salts have a role as we saw with digestion. They also have a role with absorption. They're not done doing their job until this job is done. And what they're doing is is that they are carrying these molecules to the apical membrane, this particular epithelial cell. And what's going to happen then is this. Transport again, this is absorbed of these products into the epithelial cell. So the model glyceride is going to make its way in here. The cholesterol is going to make its way in here and the free fatty acids, they're going to make their way in here. I want you to know the transport process. This is the only one tell me what it is. These are small, fat soluble molecules squeezing the way through the membrane. It was the very first transport process. We talked about Chapter Three last semester. Two words. SD simple diffusion, I think I'm hearing people mumble it simple diffusion. simple diffusion is what the diffusion of a molecule through the membrane it just kind of slithers through like a snake. There's no transporters involved. And what a lot what does that gas is like oxygen and carbon dioxide, small molecules like mono glycerides, free fatty acids and cholesterol. They're small enough to sneak their way through the membrane. I want you to notice Do you see any green things in this cell? Now on by the way, this whole thing right here, the fat digestive products along with the bile salts and the bile

acids that are surrounding it. It's called a micelle. It's in your notes. Let me actually let me show you this. So what we're doing right now is this, these words, so surrounded by bile salts, bile acids and bile salts in the small intestine to form micelles water, preventing acids, monoglycerides and cholesterol. So that line that I have outlined right there, it's this. So again, those bile salts was bile acids, a very important job with once again, absorption. And now those products there it is simple diffusion. It's right there. And now these products had been absorbed. Now these products are within this epithelial cell. Well, now what's going to happen, though, let me put my blood vessel over here my capillary. And when we put my lacteal over here, this is what's going to happen. Those molecules, right, they're going to be combined. The mono glycerides are going to be combined with free fatty acids. And we're going to make a tribalists, triglyceride again, then we're going to make a cholesterol ester and a whole bunch of them. We put them a little closer together. Now you might ask yourself What the hell doctor are. That seems like a complete waste of time. That's how the molecule started when we talked about fat digestion. Well, what did we do? Well, we started with triglycerides, and we digested into mono glycerides and free fatty acids. We took cholesterol Ester digested and got into cholesterol free fatty acids, we took this big ol piece of animal fat, ripped it apart. We emulsified it to give us the triglycerides and cholesterol esters so that we could digest them down into mono glycerides free fatty acids and cholesterol. Why the hell did we do that? If they're just going to be triglycerides and cholesterol esters, again,

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what they're inside the epithelial cells tell me they're too big. Triglycerides cannot be absorbed. They're too big cholesterol Ester cannot be absorbed. They're too big. So we need to digest them first so that we can get them inside the cell. So we can absorb them into the cell and then reconstitute them into the triglycerides in the cholesterol esters, then what's going to happen? I'll do a different color here is they are going to be combined within the cell itself. A couple of other things are going to be added that we're not going to worry about. And in the end, what we're going to have is what's called a Kailo micron. And you've already seen this word I might not have pointed it out, I don't remember if I did or not. But let's go back to this picture over here. And I'm going to show you where the word Carlow micron is. It's right there. So we absorbed these products here, fatty acids, one of the slides cholesterol, this is the epithelial cell that I've been talking about. We create a Kailo micron within the epithelial cell and then boom, it's going to make its way into the lacteal. So now the lacteal comes into play. So this is gonna get transported not into the capillary, but into the lacteal. And by the way, Kailo micron is in the same family as LDL and HDL. It is a LiPo protein. It's the lightest of the lipoproteins because it's made up the most fat, the Kyla MICRA and also have some protein that's there. I again, I'm just not talking about it. There's other things that are kind of microns made up, not just those two products right there. You don't have to worry about it. Now, if the Kailo micron, so I'm going to write it in blue, if the Kyla microns in the lacteal. Well, it's not in the circulation. Well, how are we going to get it into the circulation? You tell me? You guys learn this with lecture, see it in the beginning of the semester, at least sometime? How do we get from there to there? What duct? Is it? The thoracic duct, right? So that lymph is going there via the thoracic duct. And now the Kailo micron is in the circulation. Good for us. Now we can use it. And so there you go. So that's why we need to like 11 By the way, why can't it just go directly into the capillary like these protein digestive products and the monosaccharides is too big. It won't fit through the walls. So that's why we need the lacteal. So it can get into eventually the circulation. So there you go. A more complicated process. That's harder to absorb. That's harder to digest something that I'd already

discussed when we were talking about motility gastric motility specifically. Now what couple of last things and then that's it, we will be done. And so we'll go back over here really quick. Okay, so we just did small intestine absorption, large intestine, what did we absorb it the large intestine, we absorb a B vitamin, short chain fatty acids we talked about that they get digested right into her the soluble fiber gets digested into the short chain fatty acids. We have already talked about this. Vitamin K, we talked about that in the blood chapter, if you remember what's making the vitamin K and the short chain fatty acids, the flora. Right, so we live symbiotically with that flora. We do stuff for it, we literally feed it crap. And it feeds us, again, these kinds of things, because we're absorbing it, they're in there, they're producing them.

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Last but not least, absorption of water. A whole ton of water makes its way into your digestive tract on a daily basis, on average, about nine liters. Now, because we're drinking nine liters of water. Part of it is certainly the ingestion of fluids, but foods have water in it. And also all the fluids that were that we're making saliva model leader by a little over a leader, all those fluids that are within your digestive tract, it's mostly water. So we add it in each of the different organs. We ingest it and by the time it's all said and done, that's about how much we have on a daily basis. Almost all of this is going to get absorbed up is going to write into the blood, but not equally. A little bit of water is absorbed in the stomach. Most of its going to get absorbed in the small intestine. Of course it is we already knew that. Most absorption happens in the small intestine. Now in the past, students have several doctors etc. That doesn't agree with the lecture seabird has in her notes because she says that the large intestine is one of his main jobs to absorb water. And she's under percent right. It doesn't mean that just because the large intestines main job is to absorb water that absorb most of the water. It's not the case at all. The small intestine is again where you're going to get most absorption. So there's no discrepancy between my notes and lecture secrets notes. We're still saying correct things. We're just talking about different things. So no, these percentages and how does it happen by the way, osmotic gradients. Well, what does that mean? Well, you guys know but, like if we're talking about absorption of glucose and fructose and galactose and aluminum to the cell, well that creates an osmotic gradient waterfalls, and then water will follow them into the blood and when these things are absorbed, water will follow. When these are absorbed, water will fall, absorb nutrients water gets absorbed, absorbed nutrients, water gets absorbed. That's how it goes. All right, that is that for that chapter. Now what we're gonna do is we're gonna go into the next chapter and that's acid base. It's not fluid electrolyte balance chapter. We've done that for two semesters. We've covered that chapter over the last two semesters. Now there's some extra stuff in that chapter. It's for you to read is for you to have that chapter is not on the exam. The last exam is the digestive system, acid base balance, just those two chapters just as it says in the syllabus. Although I think in the syllabus, I did have fluid electrolyte balance is going to be discussed today. That was a typo on my part, one supposed to happen. So we're going to do acid base balance next. And that's it two chapters. We're gonna take a break though. So let's just take a break because it's a different chapter. Now. It's a little early for the break. We've only I've only been talking 31 minutes, but that's fun. So when we come back, acid base balance

# AcidBase PM 4-19-22

Fri, 4/22 4:22PM • 39:50

**SUMMARY KEYWORDS**

ph, buffers, co2, respiratory, h ions, acid, concentration, bicarb, respiratory alkalosis, bicarbonate, equation, normal, ventilation, put, range, alveolar ventilation, patient, notes, buffering, add

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Okay, folks, here we go. Last chapter, let's talk about acid base balance. Now in order to do that we have talked about pH. And we'll do this very, very briefly just the basics of pH. And so what is ph ph is the measure. So it's the measure of free keyword there, H plus concentration, such that if you have an increase in H plus concentration, plus you, you're going to have a decrease in pH. So there's an inverse relationship between the two. And if you have a decrease in H plus concentration, well, then you're going to have an increase in pH, again, an inverse relationship between the two. And so we'll just draw a very simple picture here, we're going to have two different solutions, A and B, we're going to compare them just to you know, write home about this stuff here. So this is going to be a, and there's our solution inside of that, whatever the heck that is, can be whatever you want it to be. And there's B, and so we're going to have H ions in a, we're gonna have H ions, and B, the concentration is going to be higher in a than it is in B. And so we will clearly show that and we will write it here. So we have a higher H plus concentration, which means we are going to have a lower pH because we are comparing it to B, which is in comparison, a lower H plus concentration, which means we're going to have a higher pH. Now I'll just give some values here. So for example, and this is going to be the pH value. I don't know pH equals let's go 7.02. And this one will be a pH of seven point, I don't know anyone made them up. Again, what's important is just knowing again, the relationship between H concentration and again, the PA, so the number, the pH value tells you what the concentration of H plus is, how does it do that? Well, we can do a little calculation here. So you take the pH value tend to that particular value, and you do a calculation, you plug it into a calculator, and it's going to spit out some value. So at a pH of seven, this is the concentration of H plus at a pH of eight, which is a higher pH. This is the concentration of H plus which is a smaller number, again, a lower concentration now, are we going to have to be able to do this on the exam. Now we are not, I just threw that in there. And this was actually in your notes last semester, what is important is to know the normal pH and the arterial blood. So let's just do a Ph homeostatic line here. So arterial pH, the setpoint is 7.4. And so that's what that's going to be. And then we have a normal range. And the normal range within your arterial blood is high end to normal is 7.45, low end of normal a 7.35. Now, if you go from lab to lab, it might be slightly different. But those are classic values. And those are the ones we're going to stick with. And those are the ones that I would like you to know. And that's the new notes. And it's just right here. So normal arterial pH. Now, if we go above or below those values, and we have what's called an acidosis, or alkalosis, we'll talk about that in a little bit. What keeps pH within this normal range, two things are going to keep the pH within that normal range. So what keeps pH within the normal range? We're going to talk about each individually one, we'll spend way more time on any other. So what keeps pH within the normal range? Is our

question and the answer is two things. What are those two things, number one, pH buffers. And number two, pH regulation. I should say pH buffering and pH regulation via your lungs and your kidneys. You cannot talk about pH balance in the body pH or acid base balance was in the body I should say unless you do the respiratory system first. And the urinary system as well. Because we're going to be discussing again alveolar ventilation that's going to come back alveolar ventilation, it's coming back. reabsorption is coming back secretion. It's coming back secretion and absorbed reabsorbed by the kidneys, obviously, ventilation via the lungs, those two things are going to what ultimately keep our pH where they need to be. Alright along again with the buffering. So let's talk about buffering first. Now,

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obviously, there's a whole bunch of stuff that I've crossed off here, there's three buffer systems, we're not going to get into the details of any of them, I just don't think it's a value added to the course itself, you think about chemistry course are going to just annihilate this kind of stuff. I don't think it's that important for us to understand what's happening here. So we're not going to talk about any of these individual buffer systems. And again, there's three, the only thing I need you to know is the one is the most powerful, and it's an open buffer, the co2 bicarbonate buffer system strongest buffering system, I want you to know that. So what I have circled there is what I want you to know. Now, when it comes to a buffering system, regardless of which one it is any of these three, they're going to work the same way, their job is going to be to minimize the changes in pH with an added acid or a base, what they're trying to do is keep the pH from getting beyond the normal range, that's the job of a buffer. And so I'm going to show you how that happens very, very simply. So what we're going to do is we're going to add acid, or we're going to add bass, and we're going to try to keep the pH changing very much. And so how are they going to do that? Well, let's talk about adding acid first. And they're going to do that by buffering H plus ions. So I'm going to show you what that means. We're gonna drop blood vessel here. And in that blood vessel, we are going to have H ions. And those H ions are going to be at a certain concentration, and we're going to be within our normal range. So here, our H ions, Malta going to put buffers within the blood and the buffers are going to be represented by like the blue sphere. So it'll be the three buffering systems that we just had on the screen, none of which you have to know except the one that's the most powerful. Now let's put some buffers here. So that is going to equal a buffer. All three of them. So I'm going to put buffers in here. So there's a buffer buffers here. Now, these buffers already have attached to them H ions, so they're grabbing them. So I'm going to attach some H ions to these buffers. Now, the pH value that I'm going to choose is something within our normal range. And so I'm going to pick I don't know, pH 7.38. Right, that's right there. 73574573 eighths right in there. So that's the pH of this blood right now. Now, that's based on the H ions that are freeze, let's go back to that word that I had here, free H ions, what does that mean? It means that they're not attached to anything, it means that the buffers, the H plus that are held by the buffers aren't contributing to the pH at all, because they're not free, they're attached. These on the other hand, these H ions that are free are the ones that are giving us the pH value that we have. Now what we're going to do is we're going to add acid. So we're going to add acid to the blood. Now, why in the hell would we do that? Well, it actually happens naturally through metabolism. So anaerobic and aerobic cellular respiration, produce acids, right? What kind of acids pyruvate lactate, remember that from last semester, I'm sure you biochemistry classes where they probably pounded the hell out of you with that kind of stuff. Carbon dioxide is an acid that's here, we'll be sending respiration. So we're constantly adding acid to your to

your body to your system. So I'll actually even put over here. You know what, we don't have to just answers in general, it doesn't have to be those acids, by the way. Ketones as well, or ketones acids. Yep. So ketones can be included in this. So just acids we just talked about it generically. Well, if we add acid, so what I'm going to do is I'm just going to start to add acid, which means we're going to add H plus because what is an acid do so like, we'll take a simple one, hydrochloric acid, I think everybody knows what that is. So hydrochloric acid is an acid because when it dissociates it causes the concentration of H plus to go up a chemists would say it's a proton donor, H plus and proton is the same thing. So H plus or ACL is donating H plus. That's all. So we are going to donate these H ions because we have an acid present. And so that's just what happens. And so what's happening right now is actually I'm going to draw those in a different place. I'm going to draw near the buffers. I'll put a buffer over here what the hell so let's Add some acid to the system. So we're adding H plus, as we add the H plus, well, certainly the concentration of H plus is going up. And if the concentration of H plus goes up, well, then certainly

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pH should be going down. So I'm gonna say add acid. And over here, I'm gonna have, if we don't have buffers, which is complete folly, there's buffers, but let's just say we didn't, for example. So without the buffers, what kind of a pH changes we're gonna get, I'm gonna make the number of going to completely make it up, I don't know, 7.04, it got pretty stinking acidic. It's certainly below what it's supposed to be right. So I'm three, five is a low end to normal. But in real life, we have buffers. And what those buffers are going to do is try to keep the pH in changing very much, and how are they going to do that the buffers are going to grab these ions. Now, they're not going to be free anymore. Now that I that contribute to the pH change, so they're gonna grab, or they're gonna be able to grab all of them, no, but that's okay, they'll grab enough of them to where the pH change won't be that big. So instead of going down to 7.4, maybe they'll go down to 7.3. I don't know, five, which is still considered normal. So let's go to the notes. So what if I just draw here, I drew this one line, buffers bind H plus with added acid, which is going to minimize the change in pH. That's all. So let's do this one buffers release H plus with added bass. So we'll draw a picture that looks identical to that one, at least initially, it's going to let's put our H ions here, we're going to have our buffers here as well. And again, the buffers already have each ions attached to them, doesn't mean they can't grab more, they certainly can. And when it comes to a base, a base is going to decrease H plus concentration. Because it's going to combine with the H plus. So if we did something as simple as you know, sodium hydroxide is a bass and it dissociates into let me put it over here on this side, more room, sodium hydroxide dissociates into hydroxyl ion plus the sodium, and then this right here combines with the H plus. So we get our water, so we just decreased concentration. Because this combines with that. And again, it's just a simple base, but there's a bunch of other bases. Anyway, bottom line is we're going to decrease H plus concentration, let's put our buffers here. So we'll scatter them about. And again, these, these blue things represent the three buffer systems that are in your notes, one of what you have to know. And so attached to them already are these H ions. Now we have our buffer system, or our buffer, I'm sorry, our base that was added to the system. So how am I going to represent that because what they're going to do is they're going to combine with H ions. Just gonna cross them off. So those are the ones that were combined with the base. And so we'll start out with the page 7.38 again. And what we're going to do is we're going to add a base this time, so I'll do it the same color I'll do in green. Again, very basic stuff. So we're going to add a base now, we're going to add the base without the buffers. Again, that's not real life. And so what

kind of change are we going to get? Well, let's just say it goes up to 7.74. I made it up, but certainly above 7.45. With the buffers, I'm going to say that the change is going to be 7.43, which is certainly within our normal range. Well, how did that happen? Well, this buffer, let go of that H plus ion. Now it's free, this buffer let go up that h i n now it's free. This one let go of that one. Now it's free. And so it starts to slowly increase H plus concentration to where we're supposed to be. And so this picture right here is just this line right here. Buffers release H plus with added bass, they're doing that to try to keep the concentration of H plus as constant as possible. So they're trying to minimize the change in PA so buffers keep us within our normal range. And again, here's arterial pH.

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Now, ideally, the buffer First, we'll be successful. Sometimes they're not. Sometimes we do go outside of our normal range even when we have the buffers. Now why would that happen? Because I just showed you that buffers keep pH within normal range as well, that is, if not too much acid is added or too much basis add. It depends, sometimes the buffers can become overwhelmed. And so we are now and here's our 7.45 to 7.35. range. If we are with outside that range, if we have a pH that is greater than 7.4, or five, if RPh is something less than 7.35. What that means is, is that the buffers were overwhelmed, they try. But they were unsuccessful, not because they didn't try. But because there were just too much. So you can only ask the body to do so much. And so the buffers were overwhelmed. And if that's the case, well, now our pH is going to be outside of his normal range. And bad things can happen depends on how severe This was called a disturbance is that's going to dictate again, what kind of bad things could happen. And by the way, we call this a disturbance, I'll just put the word there right off the bat. So that is an acid base disturbance. And we'll name the specific disturbances in just a little bit. Now, at this particular point, the buffers can't do anything more for us. We've run out of the buffer the buffering power within the blood itself. And so because the buffers can no longer help us, now, the lungs and the kidneys are going to come to the rescue. Or at least they're going to attempt to come to the rescue and regulate our pH back to where it's supposed to be. And so now let's talk about that. So where are we in the notes now, so we're done talking about the buffers. Now let's talk about pH regulation. And in order to start this discussion, we need to go through some values here, there's a little bit of math, we're not going to do any math on the exam, don't worry. So we already know what normal arterial blood pH is, for we know what the normal range is 735745, we know that you also have to know the normal bicarbonate concentration in the arterial blood 20 fours are sweetspot 22 to 26 is the normal range. And then co2, we already knew this one. We already know that 40 is arterial blood co2 levels. How do we know that? Remember, the oxygen gradients that we did in the respiratory chapter is that big, humongous picture, remember that. So P little a co2 40 P little a co2 40. But of course, is a normal range, there's no range, everything in about these are what would call be called arterial blood gas values. So these would be values that you would obtain all three of these values over here, if you took blood from an artery, and then send it to the lab really, really fast by me within about a half an hour, because you gotta get these values pretty quickly. By the way, it hurts. It's very unpleasant for the patient. They don't like arterial blood gases. Because to jam those arteries, it's just not a pleasant experience for them. Now, there's another value here, you don't have to know an SCR, the solubility coefficient of co2, you didn't have to note in the respiratory chapter. You don't have to note in this chapter. Now, you might notice here that I have something in red. Because last night, as I was looking at this, I saw a typo in my notes in your notes. That says H plus right there that's 100% wrong, it should say co2, please cross out each plus and puts co2 there. And I think in your notes, I

also have this in a margin equals it probably also says H plus, that is a typo, cross that off and make it co2 concentration. I don't know how that happened, but it just did, unfortunately. So anyway, something else that's important to this discussion is this chemical reaction, we hydrate carbon dioxide we get carbonic acid carbonic acid doesn't like to be carbonic acid. So quickly dissociate into h plus and bicarb. And this is a this is a chemical reaction that we will visit a number of times as we discuss acid base balance. Now, this particular chemical reaction was used not not this specific one, but is using this Henderson hostel block equation, which is sometimes written with bicarb and co2. So the hundreds in house walkways and anybody's taking a chemistry class knows that in the numerator, we have the base and the denominator we have the acid that is if we have plus log over here on this side. And so what it's telling us this, this equation is that pH is going to be equal to

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the ratio of bicarbonate to co2 and alpha co2 times P co2 Two is the concentration of co2. That's Henry's law. We'd had we saw it in the respiratory chapter. Again, I typed it wrong when I put the notes together, and I put h plus there instead of the co2. Again, everybody, hopefully everybody changed that. Now, we are not going to be using this equation for any calculations whatsoever. But let's just quickly put some numbers in here. If we put our setpoint values 24 and 40, into this equation, so put 24 here, put 40 Over here, our solubility coefficient of co2 is point o three, if you do the math, by the time it's all said and done. 7.4 is going to be your pH value, which is the setpoint value. This equation is remarkably accurate when it comes to predicting what pH is going to be based on bicarbonate concentration, and the partial pressure of co2. But we're not going to do any calculations, this is what we're going to use this particular equation to do, we're gonna look at a qualitative, so I'm going to write that equation out really quick. So pH equals PK, if you know a PK is fine, if you don't, don't worry about it, you know, a log is fine. If you don't, I don't care, bicarbonate concentration in the numerator. And then we have the solubility coefficient of co2, that is how well it dissolves in the blood, multiplied by the partial pressure of co2. So there's our Henderson Hostal block equation, we're going to tease out what we have to know. This is what we have to know from it qualitatively. So pH, I can't put an equal sign. So it's going to be proportionate to bicarbonate is in the numerator. And P co2 is in the denominator. So we're going to be changing bicarb and co2, and we're going to have some kind of change in pH. And so we just have a simple fraction here. And so now let's take this equation, let's take this chemical reaction, this one right here. And I'm also going to have one statement about what co2 is and what bicarbonate is on the next screen. So let's put our chemical reaction, co2, plus water, so we're going to hydrate co2, I'm not going to put the carbonic acid in it, it's unnecessary. Gives us h plus, plus bicarb. By the way, that's why co2 is an acid because co2 and a desired rate it gives us by Earth gives us h plus. I will also put over here our little equation. See I wrote that so stinking video gave myself no room for the equation. So co2 plus water gives us h plus plus bicarb. Now we're going to put our little sliver of equation over here pH proportional to buy card over B co2 Not equal but proportional to. And then when I'm also going to put over here is I just stated that co2 is an acid. And I'm going to assume that you guys know that bicarb is a base. If you don't well, you do now. All right, so now what are we gonna do, we're gonna change p co2, we're gonna change bicarb we're gonna make them go up, and we're gonna make them go down as we look at these three things here up on the screen. So we're going to increase p co2. And let's look at the chemical reaction. First, if we increase p co2, that means we have more co2. If we have more co2, more co2 will be hydrated. If we hydrate more co2, we're going to make more H plus. So what this is going to lead to is an increase in H plus concentration, which means

we're gonna get a decrease in pH, right? And then the opposite is going to hold true. If we have a decrease in co2, we're not going to hydrate as much co2, we're going to have less H plus. So we'll have a decrease in H plus concentration, which is going to have as a result, a higher pH. Now what about bicarb, so we're going to change bicarb, we'll make bicarb go up. So as bicarb goes up, more bicarb is going to be available to bind H plus. So we are going to bind more H plus, which means we're going to get a decrease in H plus concentration, which means we're going to get an increase in pH. And then the opposite is going to hold true. If we decrease bicarbonate concentration, we are going to bind less H plus, which means there's going to be more free age plus around which means our pH will go down. One more thing. We're going to have an increase in acid. Now here I'll actually even give examples that I talked about earlier lactate pyruvate things that are being produced during metabolism ketones.

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So again, those are acids They're going to donate a proton. So we're going to get an increase in H plus as a result of them, and a decrease in pH. But let's concentrate on the co2 in the bicarb story right now. So we just talked about these changes in pH, looking at this chemical reaction. Now let's look at the changes in pH based on this little sliver of an equation. If we have an increase in co2, that means the denominator gets bigger, right? If the denominator gets bigger, what happens to the pH value? It goes down, it's simple math, it's second grade math. That equation right there is just, I don't know four equals eight over two. So it is make the denominator bigger make that I don't know make it for now that's two, it's a smaller number. I don't think I had to actually do that. I'm pretty sure you guys know how to do second grade math. But just in case. So if the denominator gets bigger, that gets smaller denominator gets bigger, that gets smaller. If we decrease co2, that's gonna get bigger, you decrease co2, the pH goes up the numerator, whatever the heck the numerator does is what's going to happen on the other side. bicarb goes up, pH goes up by car goes down. pH goes down. Now, again, second grade math, you might think, why the hell are we going through this doctor tissue? Because believe me, when I'm asking you questions on the exam about acid base balance and stuff, second grade math, it's gone. Don't let that happen. I mean, now with you guys, you guys have been and I'm not kidding, you guys have probably been the best classes I've ever had. When it comes to academics and just the report, we don't want you guys to leave. I'm not kidding, you guys have been an awesome class anyway. So that's when you are going to use this particular equation when you have these changes in bicarb. And these changes in co2. And then I added this over here, co2 is an acid, well, what do you what happens with pH with added acid goes down less acid pH goes up, right? co2 is an acid, more acid, lower pH, less acid, higher pH bicarb, is a base base causes pH to go up when there's more of it, increase base, increase pH, decreased bass, decreased pH. So there's all kinds of ways to look at these things as they are changing as you are diagnosing a patient with a particular condition, which I'm going to make you do on the exam. We're just not there yet. We're getting the basics out of the way. All right. Now, what did we just do right there. I mean, it was actually already in the notes. So it'll be here, I just kind of went through it methodically with you guys, as we looked at the chemical reaction, that little sliver of an equation, and then again, knowing what what co2 is, and bicarb and acid and a base respectively. Now, what are we going to do? We're going to talk about the specific disturbances. Now. There's four total disturbances that I have here, but we have what are called mixed disturbances are well explain what that means later, just not right now. Let's go through the basics. So what I'm going to do here to explain this, is we're going to draw our homeostatic line again. And here it is, here's arterial pH. normal range

7352745. And I'm going to just draw this again, except I'm going to put a little bit more detail in it. So again, we're going over disturbances now and what a disturbance is, what a disturbance is, is when pH is out of range. All right, we're going to talk about the reasons that it can happen. So we're going to be on up here now there's two kinds of disturbances that we're going to be discussing. One of them is called a respiratory disturbance and other ones called a metabolic disturbance. Now, because our pH is high, it's an alkalosis. So we have either, or it doesn't have to be either it can actually be and a respiratory alkalosis. I'll explain what that is. metabolic alkalosis, would also cause pH to be too high. Now. When you hear the word respiratory, I want you to think co2. When you hear the word metabolic, I want you to think bicarb. And so I will write that over here somewhere where the hell would I write it? No, I won't. I'll just write it over here. So respiratory equals co2, metabolic equal equals bicarb. That's what I want you that's what I want the association to be.

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So a respiratory acidosis, or I'm sorry, alkalosis has to do with co2, and the pH is high. And so what that means is that a respiratory alkalosis is going to be when pH is greater than 7.45. And it's going to be due to With a low co2 level, how low below what is normal, which is 35, right. And so this will be due to a p co2 that is less than 35. If you have a respirator if you have an alkalosis, and it's due to co2 being less than 35, it is a respiratory alkalosis. All right, metabolic alkalosis, we also have a pH that is above what is considered normal. And that's 1.45. And this is a bicarb problem. And so here, you're going to have a bicarbonate concentration that is above what is normal to high end up normal is 26. So if you have a pH that's greater than 7.45, and it's due to bicarb, being above 26, it's deemed a metabolic alkalosis. Again, I'm just giving you the basics right now we'll get into the details. At a later time, just not tonight. Of course, we're going to be thorough, and we're going to do acidosis. And with an acidosis, we have a pH that's less than 7.35. And there's a respiratory acidosis. And there's a metabolic acidosis. So here's our respiratory acidosis. And metabolic acidosis. And once again, the pH is going to be less than 7.35. Less than 7.35. Now, these changes are itty bitty, right? We're looking at a 10th of the pH change here, and is considered what we call a disturbance, you have to keep pH within this very, very normal rate or narrow range. Or bad things are going to start to happen. If you have a pH of around seven, which is considered neutral, right? On the body didn't feel a patient that has a period arterial pH of seven, you should be panicked. Because something is really bad. Something bad is happening. And depending on what else is going on with that patient, it could kill them. Tell me why by the way, what is it about pH that's so detrimental? When it's outside of his normal range? What is it going to start to do denature proteins and proteins or what hormones, enzymes, structures of cells, it's all kinds of stuff. Stuffing going to work, right. And so that's why we need to keep these rain, these pH values within that very normal range or narrow range. Now, respiratory acidosis, the problem is going to be our co2 levels are too high, well, how high above what's considered normal, which is 45. bicarb is going to be lower than what's considered normal and the low end of normal is 22. And so if you have a metabolic acidosis, or Ph that I should, I should say is less than 735. And it's due to bicarb being less than 22. We have a metabolic acidosis. Now, what's causing these things. So again, disturbance disturbance. So these are the four disturbances that we're going to concentrate on. Let's talk about respiratory alkalosis first. So that's what first one we're gonna do, we're gonna see a patient that we're familiar with. So a respiratory alkalosis, as we know, decreased level of our to co2 less than 35, giving us a pH of greater than 7.45. We just drew it. And so now I have some reasons here. Now I know that in your notes, it says something different than this because I change it in your notes because it's something a bit more

clinical. And that is screwing up mechanical ventilation. Right. Okay. So let's do this. So this is respiratory alkalosis. So we're doing this one first, right at the top, and then we'll do the respiratory acidosis. So I think I don't have time for it tonight. So respiratory alkalosis. And again, we're talking about a pH is greater than 7.45.

34:16
And we're going to draw something that again, we're very familiar with conductive zone, respiratory zone, pulmonary capillary. So again, as I said, you cannot understand acid base balance unless you know the respiratory system and the urinary system.

34:36
And let's put our little gas exchange here. Oxygen in co2 out alveolar ventilation again, when it comes to a respiratory alkalosis due to hyper ventilation. And what does that mean? What that means is is that you have a high alveolar vent Leyshon, what is alveolar ventilation? Or what is ventilation, it's the amount of air going in and out of the lungs, right, specifically alveolar ventilation is the amount of air going in and out of the respiratory zone that's available for gas exchange. Now, if you have an increase in ventilation, what you're going to do is and I'll just put here, once again, an increase in alveolar ventilation, what you're going to do is you're going to breathe off more co2. What that means is you're going to get rid of more co2 from the body. So you're going to remove more co2 from the blood specifically. And so the by the time it's all said and done, if you're breathing out more co2, the co2 levels in your blood are going to be low. Now, why would we hyperventilate? So again, respiratory alkalosis due to hyperventilation I have few examples in your notes, one of which is hyperthyroidism. So hyperthyroidism, this person might have Graves disease. And as we discussed in that particular endocrine chapter, and I'm pretty sure that it was one of the things that can happen with hyperthyroidism, it doesn't mean it's going to, but it's certainly something that can happen. Too much T three and T four increases metabolic rate including that of the respiratory center, the respiratory center, if you recall, is that center in the brainstem that controls your breathing many, many things affect it. That's what increases ventilation. This is a picture that we saw back in the respiratory chapter I actually drew it myself it was a rectangle I drew over to chi respiratory center, which is a you know, something from that one game the seventh game this all pixelated fiction, and then up Pictionary, the one that's on the online, Minecraft, something like that I draw I draw like I draw like somebody would draw for Minecraft. Anyway, is that still popular? Is it really? You said? No. Everybody else saying yes. All right, doesn't matter. Let's let's continue on this discussion right here. So we're stimulating the respiratory center. That's what's causing the increase in ventilation. Now when you hear the word hyperventilate, I think you probably are envisioning somebody hyperventilating to where it's kind of become dramatic. The people with hyperthyroidism, Graves disease, they don't realize that they're hyperventilating, it's very subtle, but not so subtle that it can cause a respiratory alkalosis. Now, it just depends. Not everybody, again, with Graves disease has a respiratory alkalosis, but some can. And so we're increasing ventilation. Now this is something that's going to be a bit more dramatic, and something that you're going to be able to actually visualize it anxiety attack. Most times when people have not I want to say most times, but many times when people have anxiety attacks, they do hyperventilate, again, hyperventilation causing because we're it's stimulating the hypothalamus, hypothalamus can affect the respiratory centers, as we learned in the respiratory chapter. And so this is obviously going to be something that's short lived. Whereas when it comes to hyperthyroidism, that can be something that's

more chronic, of course. And so once the anxiety attack is over, well, the respiratory alkalosis is over. But it doesn't mean that you know, some things can happen as a result of that, like passing out, for example, because you made your pH go high. Now, this is what I used to have in the notes, I forgot to change it, but we're talking about over aggressive ventilation. So you put a patient on a ventilator, you control now how that patient breathes, you can control the settings on the ventilator, you can control that patient's tidal volume, you can control that patient's respiratory rate. And those are the things those are the two variables when it comes to ventilation. And sometimes the settings just aren't right. And that's why I mean, that's what I mean by over. I don't even remember what the word it was that I used. But sometimes the settings are just a little on the high side. And you as a clinician that you guys, you guys won't do it. But sometimes they can happen to where you over bet over ventilated patient I think is what's the words that I used. And so that creates the respiratory alkalosis. So those are three examples that I'd like you to know.

39:19

Are there others? Yeah, I mean, I have one there acute aspirin toxicity? I have another I have aspirin toxicity and another one metabolic acidosis. I think but it's chronic. I do. I'm pretty sure that was in your notes right there. We're not going to get to it tonight, obviously. And we're not going to get the respiratory acidosis. We're going to be done for the evening because there's only two minutes left. And I'm not going to start something and not finish it. So I will see you guys on Thursday for the last lecture of this semester.

# AcidBase PM 4-21-22

Fri, 4/22 4:22PM • 1:10:11

**SUMMARY KEYWORDS**

bicarb, co2, ph, compensate, kidneys, ventilation, disturbance, acidosis, respiratory, respiratory acidosis, compensation, bicarbonate, alkalosis, acid, metabolic acidosis, respiratory alkalosis, exam, lungs, problem, normal

00:00

The last one, folks, here we go. I know that there for many of you, as a final after the lab or after the lecture tonight, if you want to study, go ahead, I don't care, it's not going to bother me a bit, I'm not going to be offended by it at all. I'm just going to do my thing up here. If you want to listen, you can, it's obviously recorded. I just wanted to have this up on the screen right away respiratory co2, metabolic bicarb. Something else respiratory, lung, metabolic kidney. And I'll repeat that a number of times. So at the very end of the lecture on Tuesday, we were talking about different disturbances. And a disturbance, if you recall, we are outside of our normal pH range, we can have disturbances due to lung problems, respiratory, we can have disturbances due to the kidneys not being able to keep pH normal, that would be metabolic issues, either too high, or too low, alkalosis acidosis. And we ended by talking about respiratory alkalosis. Now we're going to do respiratory acidosis. And they've already started the picture. And there it is. So what's our problem here, again, we have a lung problem. And the problem here is going to be ventilation is too low. Let's also put our gas exchange here. So gas exchange, as we will know, oxygen in the blood co2 out of the blood into the alveoli, and then what happens as we ventilate. When we breathe out, we're going to breathe out that excess co2, again, with the respiratory acidosis, our issue is ventilation that is alveolar ventilation is too low. If that's the case, well, we breathe off less co2, as a result of that, where's the co2 going to end up in the blood. And so as a result of that, you're going to have high levels of co2 in the blood. So you're going to have a higher P co2, and if it's above 45, and causing a low pH, it's deemed a respiratory acidosis. Now, why would people have a respiratory acidosis? I have a number of examples in the notes. And so that one should be very familiar to you guys. We already actually talked about this, the pulmonary diseases that we did in the respiratory chapter, COPD, in a social lung disease, ARDS, affecting the respiratory muscles, because of Ganbare, you know, some neuropathy, ALS, causes a decrease in ventilation. And we talked about at the time that decrease in ventilation will in part increase co2 levels in the blood, we're just repeating it now. So pulmonary diseases decrease in ventilation. But one more thing for some of those pulmonary diseases like interstitial lung disease, emphysema, edits and stages, those those kinds. So some pulmonary diseases, you also have poor gas exchange. And so if you have poor gas exchange, what that means is, is that not as much co2 is going to be diffusing into the, into the lungs. And so with poor gas exchange, so not only a decrease in ventilation, but sometimes in some instances, depending on the pulmonary disease, you also have poor gas exchange. And if that's the case, less co2 out of the blood and into the alveoli. And therefore, what that will lead to, of course, is an increase in co2 in the blood and increase in co2, which is by definition, what a respiratory acidosis is. So pulmonary diseases

can be a reason that somebody has a respiratory acidosis. What else? Well, hyperthyroidism gave us a respiratory alkalosis. Hypothyroidism can give us a respiratory acidosis. You're decreasing the metabolism of the respiratory sentence to remind ourselves respiratory centers and the medulla DRG. VRG. That's controlling your breathing, decrease your metabolic rate, action potential frequency is lower, you decrease ventilation. What else last but not least some drugs. We talked about morphine last semester talked about how we can just stop your breathing if you recall. And so not just morphine, it could be heroin. It could be a bunch of fentanyl. There's a whole bunch of opioids out there. This depresses the respiratory center, just kind of like hypothyroidism does but in a very different way. And then benzodiazepines can be a another reason. Again, people are taking too many of these. And so events are that it's been labeled examples and X people take benzodiazepines typically because they have anxiety Valley and would be another example of a benzo and so benzos opiates, they depress once again the rest Vittore center, decrease your ventilation and boom,

05:02

you have yourself a respiratory acidosis. So those are respiratory problems. What about metabolic problems? Again, metabolic bicarb. Now failure the kidneys to maintain a normal bicarb level I just said that I don't know three minutes ago. So with metabolic alkalosis, we're going to have high bicarb. So metabolic alkalosis. Our issue is we have high bicarb. And that bicarb is going to be above what's normal. And the high end of normal is 26. Right? 22 to 26 is our normal bicarb. So it bicarb is high and PHSI metabolic alkalosis. Now what would cause a metabolic alkalosis? I have two examples in your notes. And I will write them down here. Vomiting is one of them. And another one is Do you remember what the other one is? Okay, ingestion of too many alcohol and drugs. So I'm going to tell you what that is. So too many alcohol and drugs. And I'll put in parentheses here an example of which you guys ever hear of Tums or Rolaids? It's just a base. People take Tom's because they say they have a sour stomach. A lot of times heartburn as well. Could just be too much acid in the stomach. And so what's gonna happen here is with vomiting, you're gonna vomit, acid, right? Because there's a lot of acid in the stomach. So you've Ahmed and not just acid but again, the contents of the stomach, which contains a lot of acid, too many alcohol and drugs, what is that going to do? What's going to buffer the acid in the stomach. That's how they work. Now, again, too many if you take a couple of times, this ain't happening. But if you take two times every hour for three or four days, then it's going to happen. So we're talking chronic here. Same thing with vomiting. In both of these, it's got to be chronic. You know, maybe after my fifth exam on Tuesday night, you might go out drinking and drink a little too much and vomit that night, you're not gonna get a metabolic alkalosis get food poisoning, though. And You vomited for three or four days straight, then maybe you develop metabolic alkalosis. So again, it's got to be a number of incidences that caused the alkalosis. Now, ha. So with all of this, it's going to cause a decrease in stomach acid. Well, how is that going to cause an increase in bicarbonate in your blood? Now, this is something that didn't go over in the digestive chapter, but it is actually in your notes. It's one of the things that we crossed off. So I'm just going to give you the cliff notes here really quick. So why would this lead to, and it will, an increase in blood, bicarbonate. This is the reason again, kind of the short version, here's the stomach. And I'll label it. And I'm going to write or I'm going to draw a parietal cell here not to scale. And we know that it is the parietal cells that are producing gastric acid and through the through the proton pumps are then taking that acid and pumping it into the lumen of the stomach. Now, these pyridyl cells have to make that H plus. And when they make that H plus the chemical reaction that takes place is that co2 plus water gives us h plus plus bicarb. That's what those

pyridyl cells are doing. They're doing that well as they make H plus they also make H HCL three, the reason that they're doing this is is that the acid needs to be replaced. The acid in the stomach needs to be replaced. And so what that means is is that the parietal cells are going to make a whole bunch of acid, which means you're going to create a whole bunch of bicarbonate, all the acid goes into the lumen of the stomach.

09:21
The bicarbonate goes into the blood so when you make gastric acid bicarb goes into the blood. And if you make a lot of gastric acid, you're gonna have a lot of bicarbonate in your blood creating a metabolic alkalosis. All right, this happens regardless, when you make gastric acid bicarb goes into the blood, but typically you don't have to make a ton because you didn't lose a lot. You make a little bit of gastric acid all the time. So the increase in bicarb in your blood under normal conditions doesn't change between 22 and 26. But if you vomited a whole bunch of stomach acids over days, then you have to make a ton of gastric acid, which means you're going to have much more bicarb in the blood. All right? Do you have to know this mechanism? You don't? What do you have to know, vomiting can cause a metabolic alkalosis. Taking too many Alkalyn drugs can cause it alkalosis. That's what you have to know, I just kind of threw that in there for you guys, so that you understand what's happening here. metabolic acidosis. Now, again, these are disturbances. Here we have a low pH, of course, we do, what's an acidosis. And the cause is going to be a bicarbonate level that's less than what's normal, and that would be 22. So it's gonna be some less than 22, causing pH to be too low. Now, I think I have four examples in your notes, I'll draw one of them out, and I'll talk about the others. One of the ones that I'm going to draw out here, the actually the only one I'm going to draw out here's kidney disease. So I will just put an asterisk here, and I'll put kidney disease here. Now, not all kidney disease, is this isn't gonna happen with all kidney diseases, just some of them. But it can happen with some of them. So we have a failing kidney here. Well, let's talk about what the kidneys do you guys already know. So we're gonna revisit reabsorption and secretion really quick. So there's our renal tubule. There's our peritubular capillary right there. And so we have the process of bicarbonate reabsorption, and we reabsorb a bunch of bicarbonate if you recall. So that's reabsorption, and then we also secrete a number of things, one of which is h plus. Well, if we have a failing kidney, that's not going to happen very well. That's not going to happen very well. And so what's going to happen as a consequence of that in the blood is that we are going to get a lower bicarb, that will cause an acidosis. And because we're not secreting as much H plus we'll get a higher H plus. And so there's your metabolic acidosis, because of kidney disease. The other examples have nothing to do with the kidneys. And by the way, neither does vomiting. And neither has taken too many alkaline drugs. So many of these, most of these metabolic disturbances that we discuss any of your patients are going to have the kidneys are not the problem. But so why do we say metabolic disturbances are kidney issues, it's simply because the kidneys can't keep the bicarb level normal, because they're just overwhelmed. The job of the kidneys is gonna take this bicarb right here and to lower it by doing what? Decreasing reabsorption. But not because there's the kidney issue that kidneys just can't keep up, that's all. But there is one incidence here, one example where there is a kidney problem, and that is the problem. But these next three, there's nothing wrong with the kidneys, they're just once again going to get overwhelmed, overwhelmed with what acid, they got overwhelmed with bicarb. And these two examples, in these next three examples are going to get overwhelmed with acid. And so what examples do we have? And so let's go to the notes. Okay, diarrhea. Now, again, chronic, not just one event, so to speak. So kidney disease, diarrhea would be

another one, I'm going to put chronic. So this is over time. Now, why would this occur? Well, when you have diarrhea, what's happening is is that you are excreting the contents of the digestive system or tract I should say too quickly. So it's leaving your body quicker than it's supposed to? Well, what that's going to lead to is a loss of digestive tract bicarb among a bunch of other things as well, but that's what we're focusing on. Is there a lot of bicarb in your digestive tract? Where's it coming from? It's in your saliva, right? It's in your bile, right? Your pancreas makes it as well. There is a whole mess of bicarb in your digestive tract. Well, where do you think the salivary glands, the liver, and the pancreas get the bicarb

14:18

partly bind from the blood. So if you're losing all of this bicarb it needs to be replaced. Just like when you lose all this gastric acid it needs to be replaced. And so what's going to happen as a consequence of this, we get it from the blood, in part get it from the blood. What does that mean? Well, it means we're taking bicarb from the blood. So we decrease blood bicarb so that we can fill the digestive tract with the bicarb that was in the blood. So The diarrhea will cause the metabolic acidosis because it's going to lower bicarb because pancreas, liver, salivary glands need to make that bicarbonate. All right, next on the list. And again, that's not a kidney problem. Aspirin toxicity. Aspirin is acid. I mean, this is the new this is acetyl salicylic acid is aspirin. Now when I say toxicity what I mean by that you just took too much freakin acid? Well, you might think is that why the hell would somebody do that maybe they have arthritis, and they can't afford insurance. And so they can't get the good stuff. But they can go to Meijer and buy a whole bottle aspirin for $2. And they take that instead for the pain to be for the for the anti inflammatory effects. And so maybe they're taken 30 aspirin a day, every single day. And so you could develop once again, a metabolic acidosis because of the acid that's being introduced. And as you introduce that acid as the age plus goes up, the bicarb goes down, right? That goes back to this very simple chemical reaction, high acid bicarb goes down, plus the acid is causing an acidosis, as well, of course it is. And this is something that we talked about, again, in the last lecture, and it goes over here. Let me show you. And let me remind you, we already did it. Increase in acid, okay, causing a metal was causing an acidosis. Now, these are the acids that I introduced to you on that day. I didn't talk about aspirin, but certainly aspirin is an acid. What else ketoacidosis. So ketones here ketones are acids we know that talked about last semester, talked about it when we did insulin this semester, and diabetes, diabetic ketoacidosis. That would be a metabolic acidosis. Again, none wrong kidneys. But they get overwhelmed. So what do you have to know in the end, I need you to know what each of these disturbances is, what causes them, examples of why they would happen, that's what I need you to know. And I'm throwing a whole bunch of stuff at you kind of like I did with the digestive system. But then we put it all together in the end, remember that we're going to do it again. So we'll put everything together in the end. And we'll diagnose patients with disturbances and all that kind of stuff when we get to the end. So we had disturbances, what are we going to do about it? pH is out of this normal range. And that's not good, the body doesn't want that. We need a negative feedback mechanism to bring pH back to where it's supposed to be. So we're going to compensate. So I'm going to draw compensation here. So I'm going to draw a disturbance, both alkalosis and acidosis. Alright, so here's our normal range, some 45735. And so we're gonna be up here. So that's a disturbance. That's an alkalosis. Whether it is a respiratory alkalosis, or a metabolic alkalosis, it doesn't matter. We need to do this, we need to get back within our normal range. And so the body is going to attempt to do that. That's what compensation is. Compensation regulation, I could use those two terms interchangeably, I'll just use

the word compensation now, because that's typically the word that's used with acid base fits. This is also compensation. So here's our disturbance. The disturbance in this case is an acidosis. And we need to get our pH back where it belongs. And so the body will attempt if it can to do this. And once again, this is also compensation. Now, what compensates, let's remind ourselves go back to the last lecture. And I think it was on the second slide. No, it wasn't it was the third one, what keeps pH within the normal range? Well, buffers try. But obviously, the buffers had been overwhelmed. If we have a disturbance. That's something that we talked about. Barbers can't help us anymore. What can Well, we can regulate, we can compensate, because we have lungs. And because we have kidneys.

19:11
Lungs can cause disturbances. The lungs can fix disturbances. The kidneys can become overwhelmed during disturbances, but they can fix disturbances. And so that's what we're going to talk about. So let's talk about regulating pH back to where it's supposed to be after we've had a disturbance. There's two kinds of compensation. There's respiratory compensation, which means the lungs are going to fix it, or there's a metabolic compensation, which means the kidneys are going to try to fix it. And so let's talk about each of those. So first, we'll do respiratory compensation again, the lungs are doing this. So respiratory compensation this is about the lungs again respiratory lungs, lungs, compensate Eat in seconds, two minutes really fast, you can change the ventilation like this. And by the way, this is not a conscious thing that you do, it just happens. Because the the pH changes in the blood being detected by neurons in areas of the body whose job it is to detect those changes, and then you just change what's going on with the respiratory center and you change the way that you ventilate, you don't think about it, it's just a reflex that occurs. So lungs compensate in seconds to minutes, lungs, compensate, metabolic disorders, not disorders, disturbances. I'll write out the word metabolic disturbances. lungs, and I'm going to write this in big letters. And I'm going to actually underline it do not compensate. The respiratory disorders. Disturbances I'm sorry. Why? Well, because the lungs are the problem with respiratory disturbances. The lungs are the problem, respiratory acidosis. It's a lung problem, respiratory alkalosis. It's a lung problem. You can't ask the lungs to fix the problem when they are the problem. So they're only going to fix they're only going to compensate a metabolic disturbance. We're bicarb where the pH is off, not because we have a co2 problem, but because we have a bicarb pH plus problem. What else? Well, how? How are the lungs going to do this? Well, they're going to change ventilation. And so because we have metabolic disturbances that are going to be compensated by the lungs, let's put on metabolic disturbances here. So what's our what's the answer to that change, ventilation out, changing ventilation will cause disturbances. But changing ventilation causes disturbances because it changes P two and p co2 and then changes pH. Well, we can certainly change pH by changing ventilation that's been established. And if we need to change pH in such a way where we go from an alkalosis to a normal range, ventilation can do that for us. ventilation can do this for us as well. So let's talk about so let's talk about a metabolic alkalosis. We do that one first. So this is somebody who has been vomiting for a long time, and so they have a metabolic alkalosis. That means pH Si, right. So we have a high pH. Well, if we have a high pH, and actually you want let me do this over here, where can I put it, I'll put it right here after the court, our little equation, that's going to be one of your best friends on this exam that's coming up. So metabolic alkalosis, I'm just going to put here in parentheses, we have high bicarb. So the numerator is big, making pH big. What do we want to do to the denominator make it equally big. So if numerator is big, you make the dominator just as big pH will normalize. That means we need to raise co2. Well, what are we going to do the ventilation to raise co2

ventilation down ventilation up, ventilation down. So during a metabolic alkalosis, for the lungs to compensate the issue and get the pH back to where it's supposed to be you will decrease ventilation, and as a result, increase co2 Until pH is normal, this is going to be the goal of the lungs. And then the complete opposite is going to take place but they metabolic acidosis where we have low bicarb you're going to increase ventilation you want to breathe off more acid because pH is too low. So you're going to decrease the co2 until again, the goal would be to make pH normal. There you go. Now let's go talk about metabolic compensation. So now we had the kidneys fixing things or at least attempting to so metabolic compensation Now we talked about the kidneys. Good enough. Kidneys are not as fast as lungs, but they're more powerful than the lungs. Kidneys take days depending on how severe the disturbances but kidneys take. This is going to be an important point later. Well, it's an important point right now it's an important point from this point forward. Kidneys take days to compensate. It takes a while for the kidneys to transport enough bicarb and H plus to get the Ah to where it wants to be. Whereas ventilation again, you can change that like this. Like right now, if everybody held their breath for 20 seconds, you create a respiratory acidosis. That's how fast it can happen. When you change ventilation that way, when you hold your breath, are you breathing out any co2? No co2 is building and building and building and building up, that's a respiratory acidosis you're not going to die. Just start breathing again, it'll go away. Okay, so there's no pathological condition at that point. But you can create a respiratory alkalosis start hyperventilating right now, you'll create a high yield, you'll create a respiratory alkalosis. Anyway, kidneys take this, I'm just I'm saying that to tell you that you can change co2 really fast, you're not changing bicarbonates plus in the blood really, really fast you just not. So kidneys take days to compensate kidneys compensate respiratory disturbances should make sense.

26:05
And as

26:06
long as fix something that the lungs are the problem that was already established over here, lungs do not compensate respiratory services. But when it comes to these metabolic disturbances, like diarrhea ketoacidosis do you much ask for it? Is there anything wrong? The kidneys, no. vomiting, do any Alkalyn drugs anything wrong kidneys? Nope. The kidneys can compensate metabolic disturbances if they're not the problem. Now the kidneys aren't compensating this one. Because they are the problem this one being kidney disease. So, we have to write something else here. Kidneys

26:49
can compensate metabolic disturbances if they are not the problem, if they are not causing the disturbance, if they're being the kidneys,

27:10
not the problem, not the issue. It's going to be slow. But they can still do it. And so now let's proceed again. How, how are the kidneys going to do this? Well, they're going to change the reabsorption of bicarbonate secretion of H plus. That's the how. So what's the how change bicarb reabsorption and H plus secretion. That's how, and so about some examples, they do it. So we'll just go with respiratory first, respiratory acidosis. Where we have high co2. Let's put our little itty bitty equation up here. Just to remind ourselves, if co2 is high, what do we want to do to the neuron numerator, what do you want to

do the bicarb wanted to go up. Of course bicarb has a base too much acid. Let's get more base there. So how are we going to change the reabsorption in the secretion here we're going to reabsorb more by card and as you do that by card goes up. And also as an added benefit, you're going to secrete more H plus. All right,

28:35
get rid of the acid pH goes up. respiratory alkalosis complete opposite. Again, we're gonna put everything together. So low co2 causing the high pH. If the co2 is low, the denominator is small, make the numerator equally as small. So we want to get back by card down,

29:01
reabsorb less by card and secrete less H plus mores plus into blood lowers pH. And so we want to do because pH is too high to begin with respiratory alkalosis. Now we can have metabolic issues as well. So we can have a metabolic acidosis. Where we have, of course, low bicarb. If the kidneys aren't the problem, then well we can do what increase. Now let's use the same kind of wording reabsorbed more like art and secrete more base plus that were raised The pH. Again, if the kidneys aren't the problem, if this is a metabolic acidosis, because of kidney disease, that ain't happening. All right. And then last but not least, a metabolic alkalosis. Again, if the kidneys aren't the problem, that tells us that we have high bicarb. So we certainly want to reabsorb less bicarb, lower it and secrete less. Ah, plus, I don't know if you see the pattern here between bicarb reabsorption and H plus secretion is always in the same direction. All right, if everything is working well, and again, I'll put over here. Kidneys are not the issue if kidneys are not the problem. So I'm restating what I stated right here. Okay. So now what? Well, let's go through some examples. So what we just covered is that so that's regulation right there. This is all about the respiratory compensation, we just kind of did a little clip, not clip notes, but I just rewrote it in a different way. Here's our metabolic compensation, we just did it. Metabolic compensation if the kidneys are not the problem, again, only if there was no kidney injury or disease. And now we have some terms here. Now, I'm not going to tell you what these terms are, I'm not going to get into this, I'm going to tell you what they are, I'm not going to give you the details of the terms, let me just tell you that. But I will kind of give you just just really quickly what this all means no compensation means we haven't started to regulate yet. That's what it means. And so if we go back to our little trace here, what that means is that you have a disturbance. And we haven't, we haven't done this yet. So if it's an alkalosis of the disturbances and alkalosis, we haven't done this yet. We haven't started to compensate. No compensation, the disturbance, if it's an acidosis, we have not started to raise the PH yet. No compensation. Partial compensation means we've started to compensate. And we might be somewhere in here. If it's an alkalosis, we're still high on pH, if it's an acidosis, we're still a bit low on pH. But it has started. full compensation means we've gotten to about where we're supposed to be within our normal range. And then there's some other detail when it comes to you know, what you should be seeing. Once we get into the actual examples that we're going to be going through, it will make more sense, I promise you that it will. So what we're going to start to do is that but not yet. So we're actually going to go through actual values. On the exam, there's going to be at least a handful of questions where I give you the pH, I'll give you the co2, and I'll give you the bicarb. You tell me what the patient has? Well, that's the respiratory acidosis, partial metabolic compensation. And when you get good enough at it, it will literally take you three seconds if you're good at it. So it's easy money on the exam. Okay, and the reason that I have more than a handful of these is because well the students in

the past have asked me to put them on the exam. Because they're kind of fun to do. At least that's what I'm told, I think they're fun, too. And plus, again, it's not as hard as what you think, right now, it might feel, you know, a little daunting, given everything that I've thrown at you over the last, you know, last year and a half. But once we start to put these kinds of things together, all of this stuff is going to start to fall into place.

33:56
So now what? Let's take a break. When we come back, we're about 35 minutes, I usually stop at about 38 ish, but it's a good place to stop. We'll come back we'll finish this up. Okay, folks, we're all back. So what are we going to do now? We're going to do a couple of examples. And we're going to see terms like no compensation, partial compensation, full compensation, we're going to talk about what's compensating, we're going to do all kinds of stuff. So how are we going to approach this? Well, first, we're going to approach it in a qualitative way with arrows going up and down and stuff, then we're going to actually get into real numbers and we're going to do stuff like you see in this particular chart. So let's do this first. So a respiratory alkalosis by definition is low co2 causing pH to be hot, right. Because this person is doing what? hyperventilating? Correct, and so they have some condition causing them to hyperventilate. Late that we went over in the last lecture. And so we can actually even put it over here in parentheses, okay, due to an increase in ventilation, which lowers the co2 levels, you've read off too much co2, hyperthyroidism, you know, whatever. So this is how we're going to do this, we're going to put our equation here three different times with arrows. So respiratory alkalosis, by definition is a high co2, I'm sorry, a high pH. little squiggly. I see I made that too big, because we have to do it three times across the screen. So a high co2 I'm sorry, a high pH bicarb is going to be normal. Okay, and so we have low co2. And so by definition, that is a respiratory alkalosis. Now when it comes to a disturbance and how we are going to correct the disturbance, if it's a respiratory alkalosis, let's go back to respiratory alkalosis, which is right here. Okay, so we have our low co2 levels. What are we going to do to fix it? We're going to change bicarb, right? H plus. So let's go back to this. We have really low co2. What are we going to do to the bicarb? If co2 is low making PHSI? What do we need to do to buy car? Make it go down, right? If the denominator is too small, you need to make the numerator equally small. So that what happens on the other side of this particular equation goes back to normal, right? Well, that means bicarb is going to be going down. Do I have any arrows actually, I'm going to put a little n next to bicarb saying that it's normal. So clearly, bicarb has not changed yet. So what do we call this? We call this a respiratory alkalosis? With no compensation? How do we know we're not compensated? The other variable hasn't changed. But now some time passes by, you know, make that error, there's a teeny bit longer. We're going to draw the equation again. Now, tell me how we're going to compensate this what's going to happen to reabsorption of bicarbonate secretion of H plus, what do best what must we do? We must lower bicarb. So we're going to do what the reabsorption of bicarbonate go up or down. Down, Right, if co2 is low, bicarb is gotta go low. So we don't want to reabsorb as much into the blood, right? Right, reabsorb less bicarb secrete less H plus. So what I'm going to put here is reabsorb, I'm going to use arrows. We're going to decrease the reabsorption of bicarb. We're going to decrease the secretion of H plus. That's compensation. Its metabolic compensation because the kidneys are doing it. So there's our metabolic compensation. So now what should these values look like? Well, the pH is probably going to start to come down. So I'm not going to have as many arrows going up. Now it's only one arrow up.

38:48

And now I'm going to put an arrow down when it comes to by cart. The disturbance is still going to be present. So we have two arrows down when it comes to PCO two. So we've started to compensate, but we haven't fully compensated Now technically, you don't fully compensate, we're not going to get into that kind of detail. They probably get into it maybe in biochemistry courses, you certainly want to get into it into med school, like I taught acid base in med school, and we get into that kind of stuff. So again, this can get much more complicated, but it's still complicated enough for us. This is still a respiratory alkalosis. Are we compensating yes or no? Yes, clearly we are. But pH isn't back to normal is it? And so because of that, we call it a partial compensation. So let's go back to this this one right here. So we had an alkalosis. Initially, we didn't compensate which is why the pH was so high. Now over time, now we might be down to about here So the pH is starting to come down, but it's not down here yet. Which is why we call it a partial compensation. Now more time is going to go by and things will continue to compensate, the kidneys will continue to compensate, the kidneys will continue to reabsorb less bicarb. Lowering the bicarb, even more secretion of H plus will go down as well, that will help bring our pH down because it'll raise the H plus in the blood, this is still metabolic compensation. And so now what will we see? Well, now I'm going to put an N in front of Pah, I'm gonna say it's normal nap. And the bicarb is now two arrows down. And the co2 is still two arrows down. And so now what we have is a respiratory alkalosis. With full compensation. Why is it full? Because pH is normal? Well, if the pH is normal, how the hell do you know that there was an acid base disturbance because bicarb is screwed up. And so co2. If you just look at the pH if you have your patient, so your pH is normal go home? No, you want to do the blood gas, you looked at the bicarb. You look at the co2, you gotta figure out what the hell's going on. Now, given that, tell me what the body cares the most about pH, it's not even close. co2 is really low and the bike and in the body said, Oh, hell, we're lowering bicarb so that our pH is normal, why the hell would they do that? What's wrong keeping bicarb normal, that's that's homeostasis, keep them co2, no less homeostasis. But when you change pH, and it's outside of the range too much what starts to happen to protein, teenagers, and proteins or hormones, and enzymes, and a whole mess of other things in the body. And if that pH is really, really low or really, really high that patient risk death, and it doesn't have to change that much. This is a very narrow range 735745. That's not the point. That's a 10th of the pH. And I'm pretty sure I said it before you get down to a pH of seven 6.9. In a chemistry class, that's known as neutral, none, the body didn't pH seven in the body, that is some crazy acidosis right there. It's dangerous, especially if they have other variables that are contributing to whatever it is, their condition is they can die. So that's a respiratory alkalosis. And that's how we are compensating the kidneys are going to compensate. So now what we'll do is we'll do a metabolic disturbance. And we'll do it the same way. And then we'll get into some real numbers. Well, maybe not real numbers, I'm just going to kind of pull them out of my head. But there'll be it'll be numbers instead of arrows. So metabolic, what was that an alkalosis. Let's do a metabolic acidosis. And with a metabolic acidosis, as we know, our bicarb is low, leading to low pH acidosis. Here's our little equation here. I'll just put it over here in the corner. And a whole bunch of things can be going on. Right now. It could be kidney disease, it could be a ketoacidosis, this person might have taken too much aspirin, whatever. One of the reasons that we had stated early, but what we're going to assume is it's not kidney disease because we want to get the kidneys involved in his story. All right. So pH is going to be really low.

43:38

And it's really low, because we have really low bicarb. And so by definition that is a metabolic acidosis. At this particular point in time we have normal co2 levels. So the diagnosis of this patient would be a metabolic given the arrows a metabolic acidosis. With no compensation. We know we're not compensated because the co2 levels are still normal. Oops. Now what? Well, some time is going to pass and not a lot of time. What's going to compensate this by the way the lungs correct metabolic issues. I'll go back to it over here. Respiratory compensations lungs compensate metabolic disturbances. And so they're going to do that by changing ventilation. Right. And so and it's going to be fast, correct. minutes seconds. And so what's going to happen here is a respiratory compensation. And so you tell me, pH is low. What's gonna happen to ventilation? Bingo. pH is low. We want to breathe out for co2, right? We want to get the co2 levels down. And so Are we going to do this we are going to increase minute ventilation. And so what that's going to lead to is breathing off more co2. Another way to look at that is co2 is an acid. The pH is low don't want to get rid of acid. Yeah, looking at the equation helps as well. So now we're not as acidic. Why are we not as acidic? Well, it's not because we've changed bicarb, at least not yet. We've made co2 Go down. Because we breathe off more of it, we increased ventilation. And so our pH is yet to be normal. But certainly we're vet we're compensating because the other variable is low. In this particular case, low because the other variable was low. So this is a metabolic acidosis with partial compensation.

45:55

And then as time goes by, and it doesn't have to be a lot, ventilation is going to continue to increase or it's just going to be higher, doesn't necessarily mean it's going to increase over time, as long as it's high enough to start changing co2 levels. Well, now I'm going to put an N in front of the pH because now it's normal, our bicarb levels are still low, because well, we still have a condition causing the BioCarbon, low bicarbonate levels would be low, and now our co2 levels are equally low. So this is what's called a full compensation. So a metabolic acidosis. With full compensation now, let's assume it's not kidney disease. Let's assume that this is a type one diabetic who doesn't have their insulin, and now they're in ketoacidosis. There's nothing wrong with the kidneys. So what I'll put over here is if the kidneys are not the issue, because kidneys can, as we already said, let's go back over here. Metabolic compensation kidneys can compensate metabolic disturbances if they're not the problem. And I'm going to assume, because I want it to be this way, that the kidneys are not the problem. If kidneys are not the issue. Well, then you tell me, what are the kidneys going to do? Increase the reabsorption of bicarb decrease the reabsorption of bicarb increase H plus secretion decrease H plus secretion you tell me? Do we need to raise bicarb? So what are we going to do to reabsorption of bicarb we're going to increase it, increase the reabsorption bicarb that will help raise the pH and at the same time, increase the secretion get rid of the acid, increased secretion. H plus. And there you go. So in this particular case, it looks as though the lungs and the kidneys are working together. However, how long is this gonna take? It's gonna take days. Now the kidneys may have to come to the rescue. And plus, optimally, we want the bicarb levels to be normal that way the co2 levels don't have to do to go out of whack. So ideally, solving this issue is getting the bicarb back to where it's supposed to be. I mean, ideally, it's getting rid of the problem that's causing the disturbance to begin with. Could go away by itself. Again, food poisoning might only last a few days. And this might not last too long. But it depends. I've given you this a few examples of what causes a metabolic acidosis. And all that kind of stuff. Did you know, you just don't know. All right. Now what? Now, let's do some numbers. Yes. If you were just given a blood gas and mph, could you tell if somebody was in respiratory or metabolic compensation? Just the blood gas? Yeah, like just the

numbers? If you're where were we going to like right here? Sure. Yes, you'd be able to tell, just literally like that. If you're here, the story is a little bit different. And I'll talk about that. So that's a good question. And it's a nice prelude to something that we're going to do when we actually get into the numbers themselves. Sometimes you can't tell you got to do more tests. And we'll actually talk about that. So what we're going to do here, and this is what you're gonna see on the exam, and I know you haven't looked at the practice exam, because I know you have anatomy on your brain and have probably for some time, but the practice exam is already there, and you're gonna see things like this. So on the practice exam on the real exam, which is by the way, when At what time 545 Okay, I mean, you can get here five if you want, but there probably going to be another class room with students and you're taking an exam, but we're in here, right if I 45 I'll send out the email to you guys. 60 questions, last two chapters. So this is what you're going to see on the exam, at least a handful of questions and maybe a couple of more that relate to something like this. I am going to give you a pH. I'm going to give you a PCO two. And I'm going to give you a bike card love. And you're going to diagnose a patient for me.

50:17
So I will pick one here. Some point, let me see what we've already done. So 7.11 PCO two, let us go with 38 bicarb, we're going to go 15. The first thing you do is you look at the pH, that's the first thing that we do.

50:51
And so pH high, well, you know what, let's do this over here. So ph 7.35 to 7.45. B co2, this is these are numbers that you have to know 35 to 45. By the way, do you notice that 35 and 45 for co2 is exactly the same as 7352745? When it comes to pH? Okay. By card normal 22 to 26. Okay, so given those pH high, low, normal, slow, co2, normal bicarb low. Tell me what the patient has. So let me ask you this, can normal co2 Cause you PAC change? No. Can low bicarbonate explain a low pH? By the way, I might pick numbers that don't work. Just so you know. Can a low bicarb? Cause a low pH? We can, okay, oh, let's put our little friend over here as well. Certainly, if the numerator gets smaller, well, then that pH value will get smaller. Right? So what's the diagnosis? Are we are we compensated? Tell me why the co2 is normal. Right? So this is a metabolic acidosis. Oops, with out. Come see how easy that is. All that stuff we just went through. Once you understand it, this stuff get like I said, once you get good at this, literally, you can look at three seconds, you're gonna know what it is. Alright, the way that we do it now is it can be more complicated than this. It is there's some other things that we just haven't talked about. But I promise you for this course. This is this is promise you is good enough. Let's do another one. We'll do a number of these. So pH to go 7.26. co2. We change that. Let's keep the bicarb the same. Let me change my PCO to let me go down to 31. So let's talk about it again. pH high low normal. It's low. It's not quite as low as 711. So you know what? I'm just gonna go two arrows down, or one arrow down PCO two high, low normal, slow, 35 is low and normal, but it's not that low. Is it? bicarb. Ready? did two arrows. What the hell, let's just do two arrows for this one as well. Tell me what this patient has. It's an acidosis. Right. So right off the bat, that's the case. Now, you know, we have two variables that are now no longer normal. So now I ask you, does a low co2 cause a low pH? If you make the denominator smaller, does this number get bigger or smaller? This gets bigger, right? So low co2 causes a high pH, right? This pH is low Correct? Is co2 the problem? Nope. Now bicarb, being low causes a low pH. We've established that and this first patient will be here, right? So what does this patient have? It's another metabolic acidosis. Now, why the hell would the co2 below because we're

compensating right? The co2 is going down to compensate the metabolic acidosis. We are getting rid of co2. The bicarb in the numerator is low, we need to make the denominator equally as low. So how is this patient doing that by the way, what they do to ventilation they increase it or decrease it. They increase ventilation to get rid of the co2 correct We lower co2 By doing what the ventilation we already know, by increasing ventilation. So this patient at this particular point in time is hyperventilating. Now, it doesn't mean that you could visibly see them hyperventilate, and they might not even feel that they're hyperventilating, but the ventilation has definitely gone up. pH is not normal, though. Right? And they're compensating right. So what kind of compensation is this? It's partial compensation. Let's do another one. 7.37 p co2 24. Aki bicarbonate the same? Et 15 ph ILO normal. It's normal now. co2. pretty stinking low. Now, it's couple arrows low bicarb is still a couple of levels low.

55:58

Now, you have a normal pH. Obviously, there's a disturbance here because the co2 levels are off and the bicarbonate levels are off. This isn't this is by far not normal. So how are you going to know what this patient has now? This is the same patient just over time, that's all I kept the bicarb the same. All right. And so all this patient did was lower thereby or lower the co2 Lower the co2 until the pH was normal. So these values that you see appear on the screen, if you got the luxury of seeing this patient through this entire thing, and drew blood glass, but blood gases over time, you would know it's the same patient and you would know that this is a metabolic acidosis. But now, what kind of compensation? It's full, because pH is normal. But what if you saw the patient at this particular point in time and your first blood glass blood gas looked like that? Is there another possibility? So in this particular case, bicarbonate went down first, and co2 went down to compensate it? Well, what a co2 was low to begin with, and bicarbonate went down to compensate it? What would the diagnosis be? So we would have a co2 problem that started it. So that's a respiratory problem, right? acidosis or alkalosis. Low co2 causes a wide and pH to go up. So it'd be a respiratory alkalosis. With a full compensation. So in this particular case, co2 went down first, this person was hyperventilating for whatever reason. And then the kidneys did what to the reabsorption of bicarb, decreased it to lower bicarb. So those are your two possibilities with that kind of bloodwork when it comes to an arterial blood gas. Now on the exam, if I do give you value similar to that there will only be one of these two as answer choices. I won't put both of them there. Because I'm not going to do to do that on the practice exam. I don't do it on the real exam. That's not a good way to write questions, by the way. All right, the two of the above three of the above all that's that's a shitty way to write questions. I've taken courses to where I've been taught how to write questions, and that's a terrible way to do it. Right, which is why I don't do it on the real exam. I do it on the practice exam, because it makes you go into the notes and look at more stuff. Alright, but a lot of the quips around the practice exams are kind of sucky questions, but it's, it's a learning experience. Okay. All right. So what else? Let's do another one. All right, let's just kind of let you know others kind of start doing some random things. Oh, by the way, this that I have up on the screen, I'm going to show you where it isn't a table. It's not the same numbers. I don't know what the hell I mean, if they're the same numbers, and I'm freaking clear when and when I'm not so so what do we have in this particular table? We have three columns. And, or I'm sorry, yeah, three columns and four rows, respiratory acidosis, no compensation, respiratory acidosis, partial compensation, respiratory acidosis full compensation. So I had each of the disturbances and from left to right, no compensation, partial compensation, full compensation. Well, if we look at this one, it was a what? metabolic acidosis. So that is right here. metabolic acidosis, no compensation, low pH, normal co2, low bicarb. low pH

normal co2, low bicarb. And then as we go do partial compensation while then the numbers start to change the exact same way that I haven't changed here. Although these numbers are not random. I actually did the calculation with the headers and hustle box equation. These are all like real numbers. I don't know if these are even close. When it comes to headers and also block I'm not a freakin math savant. So I have no idea but it's probably at least close. If you look at these values, normal pH, low co2, low bicarb, metabolic acidosis, full compensation, I'll show you. Where am I over here? Okay, normal pH, low co2, low bicarb. But look, where am I from here? normal pH, low co2, low bicarb. respiratory alkalosis, full compensation, metabolic acidosis, full compensation, the values look pretty stinking the same, or similar to each other. Just as we did it here. All right.

1:00:46

This is how you should study for these kinds of things. In my opinion, you randomly pick numbers right out of your head, or you call a friend, Hey, friend, give me a value between seven and eight. What? Like, shut up and give me a value between seven and eight? Okay, seven point are you doing give me a value? Give me pH value? Anybody? 7.24. All right, somebody give me a co2 value. Anything between 20 and 6020? Let's go 20. Okay, how about a bicarb? Anything between 10 and 44? All right. I said 10 and 40. That's not your strong suit. Want to try again? We can go for if you want. Alright. 544 is fine. Okay, let's diagnose the patient. Tell me acidosis alkalosis. Normal acidosis or pH is little on the low side co2 High, Low normal. It's low bicarb. High, Low normal. Christ almighty. We're really freaking low for this patient. All right. So what's our diagnosis? Dead? Yeah, no. Of course. 74 is actually okay. But that is a stinker. That's a really low bicarb. So tell me what this patient does a low co2 cause a low pH? No. So it's not a respiratory problem, right. Does a low bicarb? Cause a low pH? Yes. So this is a what? Metabolic? What? acidosis? Are we compensating? Yeah, right, yeah, co2 would go down to compensate. Are we fully compensated or not? Partial compensation. Okay. So how about 7.73? P co2? I don't think we've got high yet. Let's go 61 bicarb. Let's go to T, pH islandwide. Normal, high co2 by low normal, high bicarb. High Low, normal, low. Okay, so we have three variables that are wrong, right. And so far, when we've seen that we've had partial compensation, three variables that are off we've had partial compensation. So clearly, this is partial compensation. But let's figure out what chi pH being hired as a high co2 Explain a high pH. So if co2 goes up, what happens? The pH goes down. So can you explain that IPH? Nope. So this so this is a metabolic problem, right? That's a low bicarb explain a high pH. Low bicarb. pH should go down, high co2 pH should go down. I pick those on purpose. That's not possible. But this is that's good. If you realize it's not possible. That's a good thing. So when you call your mom or your brother or your friend and you ask for these random numbers, and they give you numbers that don't work, it's good that you know they don't work. So when you pick the random numbers, they might not work. If you don't realize they don't work. That's bad. If you realize that they don't work. That's good. Let's do another school Cisco 7.1. Let's go 61 again Ah, hello, normal, low co2, either normal bicarb High, Low normal. All right. Let's diagnose our patient as a high co2 explained a low pH. Yes. Right. So respiratory acidosis. Does a low bicarbonate explain a low pH? Or are we compensated? So in order to compensate, let's do our little equation here. So if our co2 levels are high, making that small, well, we have to do the numerator. So if these are big bicarb has to be big. Is that what's happening? But does a blow bicarb explain a low co2? Or I'm sorry, low pH?

1:05:40

Do we have two problems? We have what's called a mixed disturbance. A mix disturbance is when you have more than one disturbance. You can have two, you can have three this you can have somebody if somebody has food poisoning. They can have diarrhea and vomit. Can they not? This diarrhea causes metabolic acidosis? Yes, this vomiting column because of metabolic alkalosis. Yes, and what if that person had COPD, then I have a respiratory acidosis on top of it. So you're gonna have all kinds of crazy things. I'm not going to get all kinds of crazy on the exam like that. But this could very well be a person with what? COPD and kidney disease, right? Kidney Disease, low bicarb, COPD, high co2 Because your ventilation is low. So this is a respiratory acidosis. And it's a metabolic acidosis. At the same time, this person again, COPD, can you think you're gonna encounter people with COPD and kidney disease in a clinical setting? Of course you are. Most times when people have things going wrong with them, they have multiple things going wrong with them, because they're just didn't take care of themselves. That's not always the issue. But a lot of times it is smokers who don't take care of themselves. Are you kidding me? Of course, that's probably going to be the case many, many times. And so what we call this is a mixed disturbance. And again, it can get all kinds of crazy now on the exam itself. And I think I give a lot of the examples in the notes. I won't get all nutty with that. But again, you can have you know, these kinds of things and more, here's one with three.

1:07:39
But we're not going to get all crazy like that on the exam. Will there be examples of mixed disturbances on the exam?

1:07:44
Of course there's going to be, but we'll just call it a mix disturbance. All right. So you tell me, when do you know the one enough? Or you just want to go and relax before your exam? Where do you want more? Which one do you want to do? Just what? A respiratory one. Okay, you know what if we're gonna do a respiratory one, give me the values. So you know what, this is what we're going to do? Give me a respiratory alkalosis with partial compensation. Give me values for that. So I want you to give me a respiratory alkalosis. With partial compensation, what would those values look like? So it's an alkalosis. So the PHS to be high, give

1:08:37
me an up 7795 co2, partial compensation. Now, respiratory alkalosis. What's our problem? It's a lung problem, right? It's a co2 problem, right? So high pH is caused by what level of co2? A low level give me number.

1:08:59
15 is fine. What's been happening with this particular patient and an increase in ventilation or decrease? Ventilation is high or low. And so what do we need to do to buy a car to partially count? Well, we need to we want to compensate it fully. But it's it's a partial compensation. So by car hire low it'll be low. Give me number

1:09:26
15. Again, that's fine with me. There you go. Oh, and by the way, how did the kidneys do that?

1:09:33
What they do to reabsorption? Did they increase reabsorb to bicarb or decrease reabsorption of bicarb? They decrease the reabsorption of bicarbonate. Of course, that'll lower bicarb that's an excellent way to do things. Give yourself the diagnosis and come up with the numbers. Okay. What else? That's it five minutes early. Not bad for 32 weeks of course and I got done five minutes early How about that okay folks Good luck I'll see you guys Tuesday evening