TRANSCRIPTION OF DR. EUGENE KERN'S LECTURE ON EMPTY NOSE SYNDROME

Youtube: https://www.youtube.com/watch?v=LUUbHSEsJjM

This is just to let you know that the problem is a real problem. This is mentioned in the International Rhinologic Society, the American Rhinologic Society actually and the Mayo Clinic. We'll be presenting presenting 'The Nose 2000 and Beyond' in Washington DC September 20th to 23rd.

The patients that I'm going to present, these patients are patients that I personally have seen. Every single one of the cases in the 220 cases some odd cases that I'm gonna review - I have seen them personally in the 30 years, or 29 and a half years that I've been on the staff of the Mayo Clinic. These patients represent a terrible problem. Now, as I look out into the audience I know a number of you who do rhinology - have you ever seen a patient who's had this kind of CT scan and has had symptoms? You have? What symptoms have they had? Stuffiness? The nose is wide open, but they have stuffiness. What else? Crusting, drainage, malodour, nasal obstruction. Any pain? We'll talk about this. Because this is not an innocuous thing. And I'm passionate about it because I see these patients, and I'm not making this up.

And when I first heard about it - oh, you can do a turbinectomy, there is no problem - I believed it, until I started seeing the patients. And as Jean Vining was talking about, she sees the patients that come in, and she allows 45 minutes to discuss this, and I do a little enactment and I say the patients look like this ... because they're carrying X-rays and operative reports, and they're depressed. And I can look down the hall I know which patients are mine that come in carrying all those X-rays and operative reports and this is what the X-rays look like. I didn't make this up, these are what these X-rays look like. And almost all of them have the same symptom complex. Difficulty breathing, crusting, bleeding, mucopurulent green-yellow discharge, pain, a change in their sense of well-being, and a good proportion of them are depressed because of their chronic illness. I never thought that they would have quote unquote an emotional component to this, but now I know differently. It's what we see. So I want to present two cases very quickly that I've seen personally, and I had to deal with this medically and emotionally.

54 years old white woman chief complaint stuffiness in the nose since her cosmetic rhinoplasty 4 years earlier. She did have a nasal injury in childhood. She also complained of a symptom of grogginess – and we talked about this at one of the tables over lunch yesterday - the inability to concentrate. Have you ever seen that in any of your patients? And that term in the medical literature is aprosexia nasalis - inability to concentrate because of a nasal disorder. I'd never heard about that until probably about 15 years ago, and then I read about it. It was first described in Europe probably in the late 18th century - correction - late 17th century or early 18th century by an otologist Gilles of Amsterdam (Gilles de Amsterdam). Six months ago she had a reconstructive procedure without any benefit, she had allergy treatment for two years without any improvement, and here you can see - you can't read this, I'm sorry - but this is a psych evaluation. I sent the patient to psychiatry, and this psychiatrist happens to have been an internist before he became a psychiatrist, and he described her difficulty breathing as dispnea and not anxiety-related - that's what he said. This patient committed suicide. And boy that got my attention. She committed suicide in 1988 and she warned me about it, and that's why I sent her to the psychiatrist. Well maybe that's just an odd thing that sometimes happens.

Here's the next patient that got my attention: 45 year old white male banker and farmer he was from lowa chief complaint sinus problems, had a nasal injury in childhood with difficulty breathing after the injury. He had septal surgery, polypectomy. Two years ago, he developed the inability to concentrate, the groggines, if you will, lethargy, irritability, difficulty in breathing, he was treated with antibiotics and he was seen by me for evaluation, I just put in a septal button 'cause he had a perforation. That was my operation in 1993, he had a septal perforation and I slipped in a septal button. And you see, it's deceased. He committed suicide too, in 1994.

In fact, I had to go to a deposition, this is an announcement for me to come to a deposition. Because then you have to be asked: "do you take the turbinates out?" And you're challenged. How are you gonna answer that question? Cause literature is replete with incidence of it's OK to do inferior turbinectomies. So I want to talk about the Empty Nose Syndrome very briefly - what I mean by that, what we mean by that, and whether or not any of you have seen this, and if you do, how do we approach the management practically. There is just I few ideas I want to generate here: one, is that the mucosa is the organ of the nose, it's the organ of the nose, that's where everything happens. The four main functions of the nose are: olfaction, defense, respiration and cosmesis. That's what I believe are the four main - four primary functions - of the nose. And whether it's charging the inspired air with moisture - that's part of the respiratory function. Secretary IGA, IGG, mucociliary transport, that's all part of the defensive function.

And so I just wanted to show you at least the thought that the mucosa is truly the organ of the nose. And think of it - I suggest, I offer it for you consideration — to please think of it as an organ system, just the way you think of the lung, or the liver as organ systems, or the kidneys as organ systems. And I'll show you why in a moment. How much of the organ is left here? There's not much functioning mucosa left. It should have cilia. What do you think is here? This is much cilia. I have a short video that we took in our laboratory, just to let you know what cilia look like if you haven't seen it. And simple things, like neosynephrine, can paralyze cilia. Jean Vining talked about the study that was reported last year, about hypertonic salt can paralyze the cilia.

I think we need to begin to think about the mucosa as the organ of the nose. Here we see normal ciliary beat activity... you can see the brush borders here beating. We now have methods to quantitate that, using photometric methods, photo-electronic methods we can quantitate ciliary beat frequency. We think this is a very important tool now because we can apply topical solutions, various pharmacologic agents and see what occurs to our normal ciliary beat frequency. And I want to show you some normal cells again beating and that would generate that kind of response. It is easy to see, and now we have methods that we can quantitate the beating of the cilia. So when you are removing all of this tissue, you're removing functioning ciliary mucosa. Here's 0.5% solution applied. There's no beat. You paralyze the cilia. Just neosynephrine, 0.5% neosynephrine can paralyze the cilia. Many things can paralyze the cilia, including hypertonic saline.

So I just suggest that we begin to think about, when we put various preparations in the nose, what are we doing to the cilia, what are we doing to the mucociliary transport, even though we may not objectively be able to reproduce that in our own offices, about a concept of what's occurring, and second of all, when we remove functioning tissue, what happens? This is in a rabbit model, this is the work of Judy Czaja and Tom McCaffrey in our lab, this is a rabbit preparation, we dropped some india ink, and we started ... studies yesterday, this is real time, and look how the india ink is moved by ciliary activity towards the natural ostium of the maxillary sinus in the rabbit preparation. We then infected the rabbit, and I think the rabbit was sick for about six weeks, six to eight weeks, we induced a chronic inflamatory condition, and now we repeated the india ink study, so a drop of india ink, and then you see what happens

in the infected situation. That's another reason why if we spray something in the nose when the patient is already infected, we can't expect that there's gonna be much mucociliary transport to get your medication to the site where you want to get it to.

That's why we believe that lavage, using both syringe and lavage in the cavity grossly is probably a better method to introduce medication into nasal cavities that are diseased.

Those are just some ideas, if you will, that I think we should have in our mind about this. And I mentioned the four important functions of the nose, at least I think they are important. Olfaction's first, but then for sure defense, respiration, and then cosmesis. When we destroy mucosa, injure mucosa, remove mucosa, these respiratory functions and defensive functions can be significantly compromised. And the respiratory functions... we know that wide open nose... we convert a nose to a mouth - we make it wide open, it's not aerodynamically efficient, and we know from all our patients' and from our own experience with upper respiratory infections that mouth breathing is not satisfying. So, is it more satisfying to breathe through your nose or your mouth? Well, it's more satisfying to breathe through a normal nose that appears to have the narrow contours for its respiratory function.

Wide open noses like this do not function for its respiratory function, not to mention the defensive function tissue being removed. Now this is what we see in the literature... just take your scissors and cut it out and throw it away. And there's other things in the literature, many, many papers, I couldn't put them all in, just talking about inferior turbinectomies, that it's OK to do inferior turbinate resections, total inferior turbinate resections, reduction of turbinates with lasers, inferior... at least partial... turbinectomies. So here's the second point. The first point is that the mucosa is the organ of the nose. Here's the second point: the functional residual capacity of the nose. The question is: how much tissue can you remove and still have normal function? We know you can remove probably 80 to 90 % of a liver and still have normal liver function. There's a wide margin of safety as it were. We know you can remove a kidney for sure and half of the second kidney and still have normal kidney function. How much nose can we remove?

I don't think we know that. First of all, how many functional tests do we have of nasal function that we use? Not many. So we don't know much about the nasal function, at least its respiratory function and its defensive function, so it's really a guess. Second of all, for the patients that I've seen, and I'll briefly show you the data, it took about six years for the nasal mucosa that was left behind to fail. It took about six years for it to fail. So, once the nose is challenged after these procedures, it takes time before the remaining tissue that's left behind, the functioning residual capacity of the nose, to collapse and fail. So that's the second idea. Think about what you leave behind. It's not what you take, it's what you leave behind that needs to function, it's respiratory function and it's defensive function for the future. Don't be a 'turbinator'! That's my message of the day, don't be a 'turbinator'. 'Cause how much kidney can you remove, how much liver can you remove, how much nose can you remove - that's the question we each ask ourselves. I've done it, I've taken pieces out, of the turbinates, and I've regretted it. So I'm just reporting to you what I've observed. And you have to base your own decisions on your own experience and integrate that with the experience of others. How does that saying go, "good judgement comes from experience, experience comes from bad judgement". So I've done lots of bad judgement.

Here's another wonderful case: there's your pre-op, post-op. I'm not making these up. Every one of these cases I've seen personally. There's another one. Nothing left. Look at this one. So how much of the nasal defense and respiratory function is this one turbinate gonna do? This is what the nose would look like. Actually it's the opposite side... it's been cut off. That doesn't look like healthy mucosa that's left. The other interesting thing is, I biopsied it. We biopsied what's left, what we see, this squamous metaplasia. That's not respiratory epithelium any more, it's skin! And so, you can't have mucociliary transport any

more, it's gone! Squamous metaplasia has occurred. And that helps me at least in thinking about about how we're gonna treat these patients. How are we gonna treat these poor patients who come in with crusting, bleeding, difficulty breathing, inability to concentrate, difficulty sleeping, depression, and some of them with aprosexia.

There's another one: pre-op, post-op. Same patient. Septum's straighter. There's another one. And this is - I wanna read this to you ... (...) Turbinates were resected. Excessive turbinate tissue ... removed... can result in the empty nose syndrome. And this patient had pain. And it's almost like a neurogenic pain. And I've spoken to the anaesthesiologists and the neurologists about the mechanisms of this amputation kind of pain. One pain that Dr. Vining was talking about this morning after Caldwell Luc at least in a few of the studies that I've read and some of the patients that I've treated over the years, if they've had a Caldwell Luc frequently you ask them where's the pain? Well I have pain in my face. If it's unilateral it makes it much easier to say I have pain in my... where? Point with one finger where it's ... it's right here! And then I usually block them, I take some xylocaine, I put some... in the buccal gingival fold - let it sit for about 5 minutes just the way dentists do, and I use a 30 gauge needle, and put about a cc, I don't necessarily go into the infraorbital ... but in that region. If the pain goes away, then I know it's probably neurogenic pain secondary to the Caldwell Luc. And I usually send them to a pain clinic and they treat them with nortriptyline or consider blocks.

I think that helps determine whether or not there's something going in the sinus especially if the CT is negative afterwards. It's a neurogenic kind of pain. These patients have a similar neurogenic amputation kind of pain. We call it the Empty Nose Syndrome of Stenquist, this is Dr Stenquist. These patient are nasal cripples. She was in our laboratory in 1994, and she noticed the CT scans and she said: "Kernie, those X-rays look like they're empty! These patients have empty heads!" And so, that was born the empty nose syndrome of Stenquist. There, that's the kind of picture we're talking about. These patients have crusting, bleeding, foul odour because of the bacterial overgrowth, and the odour as a result of bacterial metabolism, recurrent infections, pain and depression. Eric Moore, who is one of our residents, reviewed these quote unquote ...patients. He did it a few years ago, before he left his training. It's in for publication in the American Journal of Rhinology. These are all patients that I've seen, and 100 % had crusting, 50 % had some type of emotional depression, 33 % had epistaxis and anosmia.

These are the features of these nasal cripples, as we call them. And this was for non-malignant disease, this is not the result of lateral rhinotomy or medial maxillectomy for inverting papilloma. And these are the patients that I've seen, I've asked for the old xrays. Of the 222 we reviewed, that's what we see. Nasal obstruction, crusting, bleeding, recurring infections, pain, odour, dysosmia and depression. And no matter how we cut it and how we slice it, these are the symptoms that we see, and it took 6.1 years in Eric Moore's study for those symptoms to become manifest, for the functional residual tissue to fail. Be a conservative surgeon because the sequelae can be serious. There it is. It's what we're seeing. Remember, if we biopsy this, you see skin. There's a little bit of cilia here on this specimen, but it's mainly squamous metaplasia. It's skin in the nose now. No more respiratory epithelium. So what do we do? This man's had turbinectomy, what do we do? And that's one of the places I'll use the topical Wilson's solution, 80 mg of gentamicin in 1 liter of saline, at least to try to wash out the debris because there's no cilia in the skin obviously, so you have to mechanically remove it.

Second of all, systemic antibiotics I don't believe work as well, because you have to see, envision how it's gonna get through the stroma, through the basal membrane, through the skin and get out in high enough concentrations to affect the bacteria that's growing in the sinus cavities. So remember, the skin is there, and so topical lavage is I think the corner stone of treatment along with support, and sometimes we use some rose geranium, which is a preparation that has a pleasant odour, cause frequently these

patient complain of, family members, a lot of people complain of the odour, a the odour is really the saprophytes in the nose, if you will, that are the result of the bacterial metabolism giving off its gases, so you want to reduce the bacteria. So we use Wilson's solution, 80 mg in a liter of saline, 80 mg of gentamicin, topical irrigations to remove the bacteria. Sometimes when they say they can't breathe, we put cotton in their nose, right into their nasal valve angle, and sometimes they'll grab their chest and I'll say, take a deep breath - "yeah, I can breathe now". And I've just seen it many times, and we're just trying to replace the resistors. I have used some endonasal microplasty procedures, other attempts that use hydroxylapatite, one of the colleagues in Europe is using hydroxylapatite, and I think in about a dozen or two dozen cases. Mucous membrane transplants don't work, at least not in my hands, and I've not used tissue expanders, to try to produce a temporary resistor.

The control of pain is difficult. We use 4% xylocaine in the sphenopalatine ganglion to see if the pain goes away. I like to see the patients at the time of the pain, and I'll spray their nose and put some 4% xylocaine on a cotton carrier into the sphenopalatine ganglion and they say yeah, yeah the pain is better, the pain's gone away. Then I use phenol, and I learned this from Vernon-Grey about 25 years ago. I use 88% phenol, you dry it out, after you well anaesthetize the nose, and we've reported this in the literature in, I think, 12 cases, 12 patients, and about 80% of them have control for at least 6 to 12 months. You're trying to break the reflex arc. If you can't block the nose with 4% xylocaine, then we use nortriptyline, and I have the pain clinic physicians managed that systemic use of nortriptyline. And so we're still in the process of analyzing that data.

Conclusion: be a conservative surgeon because once you remove that tissue, it doesn't regrow. I think I'll conclude my talk at this moment.

Any questions? Yes sir? Thank you. I think first of all, the question is with the new work being done looking at ciliary activity, what do I think about that essentially, evaluation of the various preparations. I think it's a long time in coming, and I think we need to base our decisions regarding what topical solutions and the concentrations and the preservatives that we're going to use based on objective evaluation of ciliary beat frequency. I mean it just makes sense. I think that sometimes you're forced to make a decision and the way I think at least the way about it, the topical sprays aren't really going to be effective, the cilia isn't there, number one. Number two - if there's skin, that obviously the cilia isn't there because there's skin replacing the lining, so therefore I did washing. If you use a waterpik, or a bulb syringe it doesn't matter to me, but removing the debris I think is the tremendous advantage for the patients, the patients seem to benefit from that. But I think the future of course is - all the things that we put in the nose we need to know what it does to the basic physiology.

Yes sir. The question is: what do I think produces more damage - inferior turbinate or middle turbinate. From the patients that I've seen this is purely anecdotal. I think the inferior turbinate – first of all, it's more tissue. So if you think again - it's an organ system, you're removing a bigger piece of the organ. And the interesting thing is, if you look later, you'll see the middle turbinate is trying to compensate. They're trying to hypertrophy, to fill the air space, to become, if you will, more physiologic. So I think a large section of the inferior turbinate removed is probably more deleterious. I think, and I didn't show this data, but we have now I think about 12 cases, somewhere in that magnitude, not very many, patients who did not have frontal disease. They had their middle turbinates taken out, and then subsequently they developed frontal disease. Has anyone seen that? And what was your reason as to why that occurred? Synechia is one cause, and the other cause that we speculate on is that the drying effect of the inspired air producing disturbance in ciliary function. Dryness is the death to cilia. And then once the bacteria is there, if there's no mucociliary transport to sweep it away, it certainly can obstruct that region and produce the frontal sinusitis.

Yes. I don't know for sure. I just haven't studied that. But as a guess, I think probably one to two hours. Does anyone have any information on that? The question is, if you apply topical decongestant or anaesthetic, say 0.5% xylocaine, and induce paresis of the cilia, how long does it take for normal ciliary beat to recover? It depends upon the agent. OK. It's an area of interest, and I should try to learn more about it.