Thank you very much for asking me to talk today, it’s a great honor, I’m Dr. Sally Carty, I’m the head of endocrine surgery at the University of Pittsburgh. Our division comprises 4 full time endocrine surgeons, we take care of thyroid, parathyroid, adrenal and some pancreatic islet tumors. I’m going to talk today about the modern strategy and techniques of minimally invasive parathyroid surgery which has changed a bit over recent years.

But first I must disclose a potential conflict that I’m also section editor for endocrine surgery for Up to Date, the online medical textbook that many are familiar with. Today’s learning objectives are summarized here, we’re going to allow participants to improve their diagnostic and management skills for primary hypoparathyroidism to closely follow the correct modern performance of initial parathyroid exploration for care. I’m not really going to talk today too much about reoperation and to learn about video assisted parathyroid exploration, cervical blood anesthesia and similar techniques for super minimally invasive exploration.

Primary hyperparathyroidism is common effecting about 1 in 500 women over the age of 40. The typical patient is a female over age 70 and there’s at least 100,000 new cases in the United States per year. The most important thing to grasp from this slide and throughout today’s talk is that the pathologic causes of primary hyperparathyroidism differ. 85 percent of patients have a single adenoma, take it out and cure the problem. One percent or less of patients have parathyroid carcinoma in that situation the alert surgeon has to recognize it and take out whatever it’s stuck to for example the ipsilateral thyroid lobe. And multiglandular disease affects about 14 percent of patients
in our area and I’ll show you some recent data confirming that from our group. Multiglandular disease can be simply double adenomata typically two superior adenomas. Or it can be all four glands enlarged and the job of the parathyroid surgeon is to discover it when its present and treat it because the intent of surgery is biochemical cure. I’ll say that several times.

Here are the clinical features of primary hyperparathyroidism grouped by color into severe, mild and slight. The classic bone, stones and abdominal ___ you see in yellow. And these are osteoporosis, nephrolithiasis, peptic ulcer disease, heartburn, pancreatitis. Bone joint and muscle pain is increasingly recognized and in my practice quite common, probably the most frequent presenting complaint. Fatigue, weakness and depression are also quite common and then we have the slight or mild or shading into asymptomatic features of memory loss, polyuria, the newly identified sleep disorder that can happen with primary hyperparathyroidism, hypertension, hyperlipidemia, cardiovascular disease.

Cardiovascular disease is well documented in the Swedish literature and not so well documented in U.S. literature. In Sweden there’s a chance of premature death with uncorrected primary hyperparathyroidism. The next slide shows which of these features get better with curative parathyroid surgery. And as you can see almost everything has been demonstrated to improve except for heartburn, pancreatitis, hypertension and hyperlipidemia. So those of you in the audience who are medical professionals, these are the papers that need to be written in the next three years, does pancreatitis get better after curative parathyroid surgery would be a great one. And also
hyperlipidemia. What we teach patients preoperatively is that hypertension is multifactorial. But it generally levels off in its severity after curative parathyroid surgery rather than continuing to worsen over time.

The features of primary hyperparathyroidism in children are subtle and trouble concentrating with a drop off in grades at school, is by far the thing that’s most often reported by baffled parents. Kids can also have depression, fatigue and weight gain. I have operated on children recently as young as eight years of age and they get better and feel better, it’s unusual for children to have hyperparathyroidism and it’s especially unusual for it to be a single adenoma. As you’ll see at the end of this talk when young people have hyperparathyroidism there’s a higher chance, higher chance of an inherited endocrine tumor syndrome.

Surgery or parathyroidectomy has been demonstrated to improve renal function, improve bone health, improve cardiovascular function, improve what ails you, neuropsychiatric health and has been demonstrated to be cost effective compared to surveillance as you’ll see in the next slide.

Only about 4 percent of patients are truly asymptomatic and there’s been hot debate in our country in the last ten or twelve years about the proper management of the truly asymptomatic patient. But overall many of the patients who choose parathyroid surgery, six months later, actually on objective survey form, validated survey forms, for example the PAS from Calgary by Dr. Piseka. Many people in that category actually feel better anyway and we’re aware that they didn’t feel poorly.
So most endocrine surgery experts nationwide do recommend that the patient, any patient with biochemical hyperparathyroidism seek consultation with a surgeon even if they’re asymptomatic and then hear about the pros and cons and potential benefits.

This is ___ surgeon’s very good paper on cost efficacy for parathyroidectomy for asymptomatic primary hyperthyroid disease. So in the 4 percent who are truly asymptomatic is there a role for surgery and this paper from 2006 In Surgery shows that monitoring was the least effective option and that surgery was more cost effective than surveillance. Pharmacologic therapy is exceeding 211 dollars annually, were not cost effective. And this is a polite way of saying that Sensipar which is considerably more expensive is not likely to become first line therapy for primary hyperparathyroidism.

There’s been two national conferences to discuss management of asymptomatic primary hyperparathyroidism. In 2001 at NIH there was a formal consensus conference and then in 2008 ACE sponsored a meeting to update those recommendations. And the latest recommendation that came out in 2008, they were only slightly tweaked from the original ones in 2002. So that for a truly asymptomatic patient we go to surgery anyway, if there’s hypercalcemia that’s one milligram per deciliter or more above the upper limit of normal, if there’s a reduction in creatinine clearance by 30 percent, if there’s any hospitalization or life-threatening hypercalcemic episode, if they’re 50 or younger – so in any event less than or equal to, because during their lifetime, their expected decades
of survival, you want those patients to be able to live those decades in health rather than in disease.
If they have osteoporosis and if they have comorbidities that complicate nonoperative surveillance, both the 2002 and 2008 meetings also said that noncompliance to followup is an indication for surgery in a truly asymptomatic patient and so is patient request.

This is a paper from our group last year that looked at medication discontinuation after curative surgery for sporadic primary hyperparathyroidism and it’s another way of showing that there are benefits to parathyroid exploration that may not yet be fully appreciated. We compared the symptoms, comorbid conditions and pre and post op medications for 206 surgically, recent surgically cured patients with primary hyperparathyroidism to patients who had thyroid surgery, not for cancer but for other conditions during the same time period. And we looked at medication use before and after surgery in each group. We had two important kinds of findings, we found that preoperatively parathyroid patients took more medicine than did thyroid patients. And they also had more comorbidity, more of the comorbidity than in primary hyperparathyroidism. And they also had more of the symptoms.

Comparing the two groups postoperatively after parathyroid surgery even minimal symptoms improved. And medications were more often reduced or stopped. And the largest category of medications that were successfully reduced or stopped after parathyroid surgery were analgesics. My bones hurt, I’ve been taking Motrin twice a day everyday, now I don’t need to take it anymore. Or my bones hurt, I’ve been taking Ultram and now I’m downsized to Motrin.
So let’s move on to discuss the diagnosis of primary hyperparathyroidism, it is a biochemical diagnosis. It’s based on elevated fasting calcium and intact PTH levels. There’s something magical about fasting and in cases where the biochemical diagnosis is unclear, the lab should always be repeated after at least a 6 hour fast. Low or normal Vitamin D levels go along with the diagnosis. Normal or increased urine calcium excretion goes along with the diagnosis and so does an assessment of renal and vitamin D status. Imaging and its results have no role in the diagnosis. We obtained parathyroid imaging when we’ve made a decision to operate and not until then.

Errors in biochemical diagnosis of primary hyperparathyroidism are common. I saw four in clinic this week. Error in diagnosis is a common and preventable cause of failed parathyroid surgery. If you don’t have the condition we don’t to be doing unnecessary surgery for it. Biochemical diagnosis must be very clear before proceeding. Causes of diagnostic ambiguity include a prolonged tourniquet time, tourniquet is blown up, the arm gets acidotic, the calcium level changes and it looks like hypercalcemia when its not. That’s why all text books, endocrinology text books say repeated elevation in calcium level. The idea being that tourniquet time won’t be prolonged twice in a row. Dietary supplements in my experience can uncover mild hyperparathyroidism. The patients says I take 4 grams of calcium a day to treat my osteoporosis. But when the calcium stops - the calcium is stopped the serum calcium normalizes. Vitamine D deficiency is a common cause of biochemical ambiguity and it’s common in our dark city especially in winter.
Vitamin D deficiency of course is more common at northern latitudes. When there’s Vitamin D deficiency to oversimplify, the condition causes secondary or compensatory elevation in PTH and the patient can have a normal calcium and an elevated Vitamin D level. And then a not very common but very important cause of biochemical ambiguity is familial hypocalciuric hypercalcemia. This is an autosomal dominant disorder, the sole morbidity of which is unnecessary parathyroid surgery. In FHH the person’s always had an elevated calcium level. It’s been there all their life or since, since adulthood came on. It doesn’t change much. They can have family members with failed parathyroid surgery or who required parathyroid surgery and I’m not sure doctor what they found. The hallmark is a low urine calcium excretion generally under 100 milligrams. And that’s why we measure FHH — sorry urine calcium excretion in patients with mild biochemical hyperparathyroidism.

So let’s see some examples. Does this patient have primary hyperparathyroidism. The calcium is normal at 9.8, this is a patient from this week. The intact PTH is 79 pg/ml, mildly elevated. The Vitamin D level is low normal. Anybody know the answer? Okay. No. The might have primary hyperparathyroidism but they definitely have mild Vitamin D deficiency. And as I just explained previously their PTH elevation is likely to be secondary to Vitamin D deficiency, it’s not conclusive but in order to uncover and firmly diagnose the situation the standard treatment is Vitamine D burst therapy which is generally ministered by endocrinologists. I don’t, I’m not an endocrinologist, I’m an endocrine surgeon so I generally defer to my colleagues to supervise that therapy. Vitamin D is a fat soluble vitamin and you can overdo it if you take too much.
Vitamin D burst therapy will replete the low vitamin D level and when it comes back normal, if the PTH drops it wasn’t primary hyperparathyroidism and if the PTH stays the same or rises and the calcium rises then you’ve diagnosed primary hyperparathyroidism. Vitamin D burst therapy is not known to cause – in fact there’s evidence that shows it definitely doesn’t constitute a risk for severe hypercalcemia.

So here one more. Does this patient have primary hyperparathyroidism? The calcium is mildly elevated, the intact PTH is normal, and Vitamin D levels are normal. Anybody? Usually yes. Usually that intact PTH is 39, it’s inappropriately high for the elevated calcium level. But first we have to rule out FHH, familial hypocalciuric hypercalcemia by obtaining a 24 hour urine calcium and creatinine taking a personal and family history and if the patient has a history of malignancy getting a PTHRP level. PTH related peptide helps you diagnose the hypercalcemia of malignancy. Again, these labs should be drawn fasting, not the 24 hour urine but everything else.

So to summarize the indications for initial parathyroid exploration for primary hyperparathyroidism are anyone with a biochemical diagnosis plus symptoms or inclination for surgery. The decision to operate is based on lab test results and the specific intent is biochemical cure. While you’re achieving a biochemical cure at surgery you also want to address any concurrent thyroid disease, minimize the risks of dissection and minimize the fiscal and societal costs of having to have surgery in the first place.
A brief aside here about concurrent thyroid disease, it’s really common. It happens all week long and that’s one of the reasons that we routinely obtain a preoperative surgical – a preoperative cervical ultrasound.

This is a nice paper out of M.D. Anderson that shows that concurrent thyroid pathology occurs – is the rule, 84% of people. It frequently requires preoperative management for example FNA biopsy and it often requires concurrent thyroidectomy, 12 percent, that’s the rate in our process as well. Not infrequently those patients have thyroid cancer. Now, why might that be? Well in the olden days 80 years ago, cervical radiation was administered to large cohorts of infants who’s parents were told it’s to treat overactive sinus. Which as far as we can determine today was a way to try and reduce the incidence of crib death. It didn’t work in reducing the incidence of crib death, it did cause a higher rate of papillary thyroid cancer about 50, five decades after administration and ten years after that according to a nice paper from Cleveland Clinic about ten years ago, it also predisposed us to parathyroid adenoma formation. So in a very elderly patient the coincidence of thyroid cancer and parathyroid adenoma makes you presume or wonder whether they received XRT as a child.

Okay now we’re going to go on and talk about the complications of parathyroid surgery and they differ in some whether it’s an initial exploration or a re-op. A reoperation is done through stark gristle, it looks like gristle just at the end of the chicken bone in some patients. And trying to find a pea embedded in gristle is much harder than going in and dissecting through glistening virgin tissue ___ in the first place. The failure rate, failed parathyroid surgery, I have failed to correct the
biochemical condition is the most common operative complication in either operation. And initial failure rates among experts nationwide rates from 1 to 5 percent, reoperative failure rate are higher 5 to 10 percent. Our failure rate right now here is 1.8 percent, we’re very proud of that.

Hypoparathyroidism, in other words the surgeon accidentally took out all the parathyroid tissue, it supposedly happens in about 1 patient out of 50 at initial exploration. Our rates are a little lower, considerably lower. And it’s 5 times more likely when you’re operating through a scar. Recurrent laryngeal nodal paralysis can happen and is a feared complication of initial and reoperative exploration, again it’s five times more likely when you’re operating through a scar. Surgical bleeding, the patient has a hematoma requiring re-exploration, urgent reexploration. Happens to about one patient in 300, whether it’s a reoperation or not and there are a number of papers showing that initial curative surgery is a lot cheaper than having to go back in a proportion of patients on purpose. So this is why we try to do it right the first time with initial parathyroid exploration.

When a patient requires reoperation and this is all I’m going to say about reoperation today, we look for stricter or we look for a higher bar to send the patient to surgery. They have to have significant hypercalcemia or significant symptoms, nephrolithiasis, osteoporosis, significant hypercalcuria or reduction in renal function that’s already beginning to hurt their kidneys. And at most centers you can’t go back in if you don’t two concordant positive imaging studies with results that guide your exploration through scar.
Why is parathyroid surgery called exploration anyway? Time to consider that. It’s because you never know what anatomy you’re going to find. Most people have four parathyroid glands, two and two. The superior ones drop down the middle neck compartment, it’s the middle medial spine as they enlarge. And inferior glands are anterior – in the anterior compartment and tend to associate around the tip of the thyroid gland anteriorly but can also be in the thymus or undescended up at the carotid bowl. Superior glands can also I forgot to say hide in the tracheoesophageal groove and the retropharyngeal space.

So this bullet number two talks about and details all the weird locations that we routinely look for enlarged parathyroid glands at almost ever exploration. If you don’t find it right away you’ve got to look in all the weird places to find it. Parathyroid, enlarged parathyroid glands can also be intrathyroidal, encompassed by a thyroid lobe or nearly 90 percent embedded in thyroid tissue. And thinking about that and sometimes resecting thyroid tissue at difficult exploration is part of our armamentarium. Armamentarium. In addition, 9 percent of the population has more than four parathyroid glands. I have two patients who had seven and one who had nine. And it’s our job to find the enlarged parathyroid glands and treat them no matter how many are present.

Experience is needed not only to know where to look but also to compare an enlarged gland to a small one. Is this big and is this small or are they similar or is this multiglandular disease. A normal parathyroid gland is about 35 to 50 milligrams, it has a particular shape and color. And enlarged parathyroid gland can be much larger 14 grams or to be a microadenoma at 87 grams - last week. So
the alert, the expert surgeon is experienced at knowing all the places to look and at deciding during the surgery if this is big and this is small, if these are normal or abnormal.

This nice paper by Julianne Sosa who’s now at Yale examines the thresholds for surgery and surgical outcomes for patients with primary hyperthyroidism and in multivariate analysis she showed that not only do high volume surgeons have a lower threshold for operating, in other words our success rates are good, you look pretty good sir, let’s give it a try. But they also had demonstrably lower complications rates and this paper and others that have shown lower complication rates among experts are widely quoted in the last decade or so as society moved toward cohering parathyroid surgery at expert centers.

Strategies to prevent failed initial parathyroid exploration to review are clear biochemical diagnosis, defining success as curing the biochemical condition. And you’re not cured until your cured at 6 months - that kind of comes from old literature and is a proposition that somebody should write a paper on and test soon but it is the standard point of view and one that I adhere to. Operative failure is morbid and costly, adenomectomy is fun, it’s fun to go and find an easy one, take it out, go out and have coffee. But if it doesn’t cure the patient you have not done anyone a service and you never know when it’s going to be easy or hard. Imaging tests fail to identify multiglandular disease.
So now we’re going to talk about imaging tests. Imaging tests, their ability to predict multiglandular disease is 50 percent which is a coin toss, ____ has a 50 percent likelihood of showing multiglandular disease when its present and a 50 percent likelihood of missing it when its present.

Current techniques to exclude multiglandular disease are – there is an array of them but there are only two validated techniques. In other words, many papers and years of use have shown that two of these work and they are the gold standard four gland exploration, the surgeon goes in and finds all the parathyroid glands and looks at them and decides if there’s one big one and three little ones or two big ones or four big ones. Intraoperative PTH monitoring is a well validated technique that can, that has supplanted deliberate four gland exploration and I’m going to talk about it in a coming slide.

Techniques that are unproven and/or disproven include all imaging, imaging can not reliably distinguish between adenoma and hyperplasia and those slides are coming next. And there’s also been literature abroad looking at unilateral adenomectomy without PTH and having obligate failure rates. If you just go in and do one side you’re going to have to go back in at least 80% of patients.

This is a SPECT CT scan, the patient is looking at you face on. I can’t go over and wiggle with the slides with my pointer so let me just tell you that the two biggest bright blobs at the top are carotid glands and the next two biggest bright blobs are salivary glands and the next on the left, the next two big bright areas are the thyroid glands. And the delayed images appear on the right and there’s
nothing, no real difference between the left and the right images except that the thyroid gland has washed out. This is negative imaging.

Here’s an example of a possible adenoma. Interestingly it’s seen on the early scans not the delayed which can happen sometimes. The arrow is pointing to an anatomic structure that’s taking up sestamibi dye below the thyroid gland. It could be a lymph node, could be extrathyroidal thyroid tissue. Most of the time that’s a parathyroid, an enlarged parathyroid gland.

And here’s what we call a light bulb a term that we coined to describe a clear cut, juicy, let’s go right in after it, looks like it’s only one. And the arrow on each side or on each image is pointing to an anatomic structure that takes up sestamibi dye and continues to have uptake on the delayed images. Now even though this is enticing preoperatively, is it reliable that a light bulb shows just one enlarged parathyroid gland.

We tested it and this is my partner Lynn Yip’s study from 2008 where she and Dr. Dan Prima from radiology, Dan looked blindly, was blinded to the results of 764 SPECT scans for patients who had been selected for surgical cure. In other words we know what their anatomy was, it’s not that we missed multiglandular disease. And the SPECT images, Dr. Prima’s categorized the SPECT images into five categories ranging from negative to light bulb, there was also category for multiglandular disease. The overall rate of multiglandular disease in the 764 patients was 14 percent just as I said at the beginning. And light bulb single focused scans had an 8 ½% rate of multiglandular disease, in
other words, it looked like one, the x-rays were enticing for this one, and the x-rays were wrong. How did we find those? We used intraoperative PTH monitoring. What is the old standby? Bilateral four gland exploration.

This follow-up study again by Dr. Yip in our group scored the SPECT results with and compared them side by side with the rate of multiglandular disease and she found that the more negative the scan the higher the rate of multiglandular disease and the next step was relation, a negative scan, this scan here had a 22% chance of multiglandular disease. And this has been also shown by other groups in the literature. Only a quarter of scans that were read as having multiglandular disease actually had it at exploration, thereby lateral uptake was from a lymph node or an extrathyroidal thyroid nodule or something else. Conclusion, expert parathyroid surgeons must use validated adjuncts to exclude multiglandular disease, you can’t rely on the x-ray findings.

So I’m going to pause and insert two of the pearls that we teach endocrine surgery fellows nationwide, this quote comes from Dr. John Dopman, a radiologist at NIH who did a lot of the very central radiology studies that I’ve – that form the basis for what I’ve just talked about. He was made an honorary member of our society after he stated the best localization technique is an experienced parathyroid surgeon. The second bullet I coined, and it’s pretty helpful, imaging tells you where to start looking, look there’s the first big one. And PTH monitoring tells you when you can stop looking.
So PTH monitoring is a technique developed by Dr. George Irvin in Miami, and further developed by his colleagues Denise Carneiro and Carmen Solozano, and it’s based on the short plasma half life of PTH, which is only 3 to 5 minutes. So you take out a hyperfunctioning parathyroid gland and traditionally 10 minutes or 15 minutes later depending on which protocol you are using you can measure accurately and rely on a nice drop in the plasma half life. QTH, QPTH protocols vary by center. Why is that? Some centers use different assays. Some centers have lab time and specimen transport to the lab to be assayed. Some centers draw the blood from different sites. There is no one perfect way to do it. The Miami criteria, and remember I disclosed at the beginning that I’m Section Editor for Up to Date, the Miami criteria are now listed in the Up to Date chapter devoted to intraoperative PTH monitoring along with a side by side comparison to the criteria that, that some other hospitals use.

Here we adhered to the 50% drop from the baseline level and we also required a drop into the normal range. The punch line is that when the PTH level stays elevated the surgeon keeps exploring, and that’s why we counsel patients that your surgery might take 45 minutes or 3 hours depending on the anatomy that we uncover. PTH monitoring is a handy tool as well if you are having a difficult or unsuccessful exploration, you can get bilateral JPTHs, it helps you discover if there are three abnormal glands on one side, and it helps you discover if there is – by comparing the PTHs if there is no difference, it helps you deduce that it might actually be mediastinal adenoma, it’s rare but it happens.
Now I’m going to go over the principles of minimally invasive parathyroid exploration. Minimally invasive is a nebulous term that means I kept the dissection as focused as I could and I made as short an incision as I could. And there are many different ways to crack that knot. The punch line is that small incisions, most incisions are smaller nowadays anyway. So if you read a paper that says our new technique is wonderful because we’re comparing a 2 cm incision to a 12 cm incision nobody uses a 12 cm incision anymore, they haven’t done that for 10 years. So you can’t setup a straw horse to prove the validity of a new technique.

With that preface let me go back again over this list. Minimally invasive parathyroid exploration uses a small incision, Jean-Francois Henri in Marseilles, France uses insufflation and punctate holes laterally, a beautifully elegant technique that’s a little bit hard for some of the rest of us to absorb and perform quite as well. The video-assisted technique that I’m going to talk about in more detail comes from Paulo Micheli in Pisa, Italy. All – okay, techniques that minimize intervention include ones that shorten the OR time, Labutesman at Yale is the force behind routine cervical block anesthesia during parathyroid exploration, focused dissection guided by imaging can include use of a probe to rapidly discover exactly where an enlarged parathyroid gland is, make a cut right over it, again a small incision, that must be combined with PTH monitoring to ensure that there is no multiglandular disease. When you are devising a minimally invasive parathyroid operation or fine-tuning one you want to make sure that your risks are the same, your success is the same and your costs are the same, all the same or better.
Current parathyroid – this slide is out of order – current parathyroid localization tests are listed here and we obtain them only with a decision to operate, and they are SPECT Sestamibi or SPECT CT which combines die uptake in 3-dimensions with CT imaging and then fuses the images together so that it comes back to you with a nice arrow and a color change and says look, it’s right there; that, we use that. Ultrasound is operator dependent, so it’s more accurate at high volume centers. If it’s not accurate at your center then it’s not worth the healthcare dollar to use it, it is very useful in discovering as we said at the beginning concurrent thyroid disease. Probe radio guidance is used at some centers. If you plan to use four gland exploration, localization is not recommended. This is a new recommendation, again comes out of the Up to Date chapter on parathyroid exploration.

So this is our operating team and on the left you can see Miss Sarah, our scrub nurse, and to her – standing at her side is Dr. Yip, whose imaging papers I talked about earlier, then the fellow is giving her a very weird look, okay, and that’s me standing there holding the camera and behind Lynn’s head is a screen that shows the parathyroid adenoma that we are removing with video-assisted parathyroid exploration a 2 cm incision. Our experience with minimally invasive parathyroid exploration has undergone evolution. I came in 1991, I’ve been here 20 years, and since they we have 2 decades of a 98% cure rate with less than 1% morbidity, knock on wood, we are very proud of that.

We have now as of last week 3½ thousand parathyroid operations in our clinical database and we follow those patients so that we can determine our cure rates and counsel the next set of patients. We traditionally use an incision about 5 or 6 cm, in the last decade we use incisions that are by data 2 to
5 cm in length with short OR times, early discharge and prompt recovery. And one of the ways we do that is by looking at – is by utilizing Dr. Micheli’s technique for video-assisted parathyroidectomy. And this is based on a small central incision, tiny little instruments that are very narrow, and adenomectomy, take out the first big one that is seen on imaging and then measure the PTH level and if there’s a nice drop you are done, and if it doesn’t drop you have to keep looking. It’s direct, there is no gas insufflation so it’s quicker, and it has great intraoperative flexibility, it’s the work of a moment to open to the regular minimally invasive incision from the 2 cm ¾ of a inch video-assisted incision. If there is bleeding or thyroid cancer or multiglandular disease it’s the work of a moment to open and get the exposure that you need.

It’s contraindicated with non-concordant imaging results. In other words the ultrasound shows one thing, and the sestamibi shows another, perhaps on different sides of the neck, that’s not a good patient to choose for the video-assisted technique because you are going to need to look around. It’s contraindicated in reoperation through scar, it’s contraindicated if the patient has – is obese or has a large thyroid gland, two tissue effects that make it hard to work. And remember you are working with sticks looking at a TV screen, so you need room in the front of the neck. And it’s contraindicated with no multiglandular disease because in that situation you are going to need to see all the parathyroid glands anyway.

This is a nice little paper that our fellow Adrienne Melck made last year and it’s just coming out in American Surgeon and it shows our implementation of the video-assisted technique. We started in
2004, my first patient a plastic surgeon’s wife was very happy and since then have become increasingly comfortable with offering and using the video-assisted technique. It’s now routine, I use different equipment and a different strategy but it’s par for the course. We compared 125 video-assisted patients to 95 regular minimally invasive patients. In each group they only had one apparent focus on imaging, they had single focus concordant imaging.

We found that the mean OR times, their requirement for pain medications and the complication rates did not differ. We found that the rate of conversion to a regular 4 to 5 cm incision was 14% which is at least twice that in Pisa, their published rate of conversion is 6%. Maybe I have a lower threshold, maybe patients are heavier here, it’s hard to say. But we also found that video-assisted patients were less likely to need to an overnight stay, and we are not sure why. Remember that, that video-assisted patients have a short – what is the real difference in their operation, they have a shorter incision. Maybe they perceive that they are going to get well quicker, we are not really sure why they were much more ready to leave the hospital with alacrity, but they were.

Here is the learning curve, my learning curve as a surgeon during video-assisted parathyroid exploration implementation. It starts in 2004 and goes up to 2009, and this is operative time on the Y axis. And you can see I got better at it over time. Every time there is a peak that would be somebody who we opened, multiglandular disease for example. About half of the patients who were converted to a regular incision had multiglandular disease as the reason.
Now a couple of slides about cervical block technique, this was developed and as I said is championed by Dr. Rob Udelsman who is Chairman of Surgery at Yale and past President of our association. And Dr. Yip went up to Yale to learn it, and it’s a terrific technique. It requires a patient who wants to be awake during the operation, because as you’ll see on the next slide they can’t have any claustrophobia or discomfort with the draping close to their face. But it’s also a useful technique in patients who have severe heart disease and you are trying to minimize the risks of anesthetic, the backup plan is to convert to a general anesthetic if there is bleeding or multiglandular disease or the patient can’t tolerate the cervical block technique. But we haven’t had to use that very often.

So the surgeon, me, Dr. Yip, in prepped and draped sterile skin performs the cervical block as demonstrated and here is a picture from the patient’s left of what it’s like to be that patient. Oxygen has to be suctioned away from the field because oxygen is flammable, and so the drapes have to be adhered to the skin very firmly so that there is no mixing of the air under the drape or the air in the operative field. But it’s a wonderful technique that’s used readily here, it requires experienced anesthesiology in case the breathing tube needs to go in during the surgery. We’ve only had to do that as you see her twice in about 35 patients who have been – had parathyroid exploration with cervical block since we implemented it two years ago.

Let’s go back to that. So this is Dr. Udelsman’s graph showing reasons for opening to a regular incision and, and with general endotracheal intubation during cervical block, and in other words the first one concomitant thyroid pathologic features is a big goiter. Multiglandular disease, I just can’t
see what I need to see, the patient couldn’t tolerate it. Intraoperative diagnosis of parathyroid carcinoma, definitely could happen, toxic reaction to Lidocaine, it’s unlikely but you have to be careful that you are not injecting intravenously.

Our routine postop care after parathyroid surgery is very streamlined, and a paper will be coming out hopefully next year that shows that the postop care of all the program directors for endocrine surgery fellowships in our country use very streamlined postoperative care that’s mostly based on patient education. We have two different handouts, wound care and how to manage your calcium supplementation. We routinely use calcium supplementation for 6 months. In the short run it helps prevent paresthesias, numbness or tingling in the lips or fingertips, numbness or tingling in the lips or fingertips after parathyroid surgery is generally a good sign that your surgery worked, it’s only rarely associated with permanent low calcium levels. But you have to counsel the patient about when to call based on mild versus severe symptoms.

In the long run postoperative calcium supplementation in several papers including our own helps you assess operative care at 6 months by removing the possibility of secondary elevation in PTH from vitamin D deficiency. I routinely use Marcaine local anesthetic, it’s pretty long acting, it’s preemptive and by using – with the use of Marcaine local anesthetic most patients require Tylenol or nothing for pain after the surgery. Once they have been counseled that their sore throat is generally related to intubation they get right over that, happily.
We have our patients shower and behave normally postop, we don’t want any DVTs, we don’t want any dehydration. They return to work at 1 to 14 days depending on their work ethic. And we check their labs at 10 days postop and again thoroughly assess them for biochemical cure at 6 months. If they are not cured we follow them, we want to know about operative failures. Our current failure rate as I said is 1.8% and that’s how we are able to educate patients about it by following for the group.

Now these are some – the hereditary forms of hyperparathyroidism and we are going to cover this briefly and then take the quiz. So MEN1 is by far the most common in our region. I don’t know if there is a founder effect or what, but I see 45 or 60 MEN1 patients in chronic surveyance each year. MEN1 there is a slide coming up on it so I won’t say it here. MEN2a is common in the St. Louis area, it’s pretty uncommon here, consists of medullary thyroid cancer, hyperparathyroidism that’s not – that’s technically multiglandular but there’s great heterogeneity in gland size, some – many patients with MEN2a have one big one and several normal looking ones, parathyroid glands, and pheochromocytoma.

Jaw Tumor Syndrome can occur in patients who have had a bony tumor, there’s a nice gene test for it now. And it’s particularly something that you think about if the patient has parathyroid cancer. And then there can be Benign Isolated Familial Hyperparathyroidism, it’s just hyperparathyroidism running in the family without any associated endocrinopathies. Confounding this diagnosis is FHH, which is also autosomal dominant, we discussed it earlier.
MEN1 is autosomal dominant, highly penetrant, there’s great phenotypic heterogeneity, it’s just not true as older books say that if you don’t have hyperparathyroidism by age 50 you don’t have MEN1, I have three patients like that. Didn’t have hyperparathyroidism by age 50, 60 or 70 and have gene positive MEN1.

The three Ps of MEN1 parathyroid hyperplasia, multiglandular disease, pituitary adenoma and pancreatic islet cell tumor, which can be insulinoma, nonfunctional, gastrinoma, glucagonoma and so forth are the three Ps are the hallmark but nowadays we know that there’s actually five features of MEN1, carcinoid tumor and Benign Adrenal Adenoma are the fourth and fifth. And there’s even now multiple dermal collagenoma. One dermatologist suggested that if you have six axillary skin tags you have MEN1 pathognomonically, I’ve yet to see that in print, although I think it’s coming.

MEN1 is defined in three ways, by the concurrence of two clinical features, by – that’s supposed to be one feature plus a first degree relative, or by a positive menin gene test. And the treatment of hyperparathyroidism in MEN1 is now coming back, the pendulum is starting to swing away from total parathyroidectomy and more back towards subtotal parathyroidectomy. Total parathyroidectomy is traditionally done in MEN1 with nondominant form autotransplantation. We did a paper 2 years ago and so have others showing that there can be a lot of technical difficulties with that, and when hyperparathyroidism recurs you are not sure if it’s in the arm or the neck from a supernumerary gland, so I generally do a subtotal after a thorough discussion with the patient.
This is nice paper to close with, it’s incredibly helpful in your clinic. We are taught as medical students that a preoperative query about a family history of MEN1 is widely advised but rarely positive. These are all my patients that Dr. Yip and I studied when she was the fellow in her endocrine surgery. Around 2000 I instituted a use of a – routine use of a simple 6 question panel which we call 6Q for this in the next slide and I used that to further screen for inherited MEN1 hyperparathyroidism in 939 patients in this study who presented for parathyroid surgery. Their PCP, their endocrinologist or both decided that they had sporadic disease and sent them to me for routine initial parathyroid surgery. And 5% of them had MEN1, when I used the 6Q panel. This paper also found that MEN1 patients were more often male and more often young, typically under 30, and it also produced the useful tidbit that when you find multiglandular disease at surgery it’s actually 1 in 4 patients do have MEN1. We often wonder should we send such a patient for testing?

Now I’m not going to tell you what the 6 questions are quite yet, I’m just going to go on and on about them first by saying that compared to family history the 6Q panel was more likely to identify MEN1 with a lovely P value and that in multi-varied analysis each of the 6 questions, the more questions you had a positive answer to the higher the likelihood of MEN1, it was a step-wise association. Okay?

Now we present these data this way because if I – when I tell you the 6 questions you are going to say oh, well that’s nothing big. But here are all the P values first, okay, it’s really helpful. Okay, so here are the 6 questions. Sir, do you have any family history of brain tumors, pancreatic tumors,
neck surgery, high calcium levels, kidney stones or ulcers? If you need to say it again you can just say it again. Mam, do you have any blood relatives with brain tumors, pancreatic tumors, neck surgery, high calcium levels, kidney stones or ulcers? It’s that simple.

Okay that concludes my presentation, thank you for listening, good luck on the quiz.