A LANDMARK IDEA
AIMS TO CHANGE THE COURSE OF DIABETES CARE

ALSO IN THIS ISSUE:
• Can Aging Be Reversed?
• Fighting Every Pound: An Endocrinologist Gets Candid About Her Own Struggle With Weight Issues
• Updates on Osteoporosis Incidence, Recognition and Treatment
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22 Updates on Osteoporosis Incidence, Recognition and Treatment

Osteoporosis, a disease in which the density and quality of bone are reduced, continues to be a major medical issue, with annual treatment costs estimated at $16 billion in the U.S. alone. In this article, the authors provide expert insights on osteoporosis identification and treatment based on guidelines recently published by the American Association of Clinical Endocrinologists.

26 The Fascinating Discoveries Leading to Thyroid Treatment Over the Ages

Although thyroidology – the medical practice specializing in the thyroid gland and related areas – is relatively new, the discoveries that ultimately led to treatments being used today began a thousand years prior.
AACE adopted the universal endocrine logo design (left), which is intended to serve and be recognized by the scientific community and the public at large as an international symbol of recognition of all areas of the specialty of endocrinology (academic/research/clinical). In its simplest form, the logo represents a continuous loop that conveys the concept of endocrine science, education, communication, safety, and overall good endocrine health; lay focus groups identified “balance” and endocrinologists identified “feedback loop” — both are desired interpretative attributes.

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AACE is a professional medical organization with more than 7,500 members in the United States and more than 90 other countries. Founded in 1991, AACE is dedicated to the optimal care of patients with endocrine problems. AACE initiatives inform the public about endocrine disorders. AACE also conducts continuing education programs for clinical endocrinologists, physicians whose advanced, specialized training enables them to be experts in the care of endocrine diseases such as diabetes, thyroid disorders, growth hormone deficiency, osteoporosis, cholesterol disorders, hypertension and obesity.

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DR. FELICE CALDARELLA
Dr. Felice Caldarella graduated from SUNY Upstate Medical University, Syracuse, New York and completed his Internal Medicine residency program at Brown University in Providence, Rhode Island. He did his fellowship training in Endocrinology at the University of Medicine and Dentistry of New Jersey in Newark. He has been practicing endocrinology for the past 15 years at Hunterdon Medical Center in Flemington, New Jersey. He is the current Treasurer of the American Association of Clinical Endocrinology (AACE).

DR. MALINI GANESH
Dr. Malini Ganesh is Chief Fellow in the Division of Endocrinology and Metabolism at Rush University Medical Center and Cook County Health & Hospitals Systems, Chicago. She completed her Internal Medicine residency at Cook County Health & Hospitals Systems. Her current research interests are in secondary hypertension as well as in secondary hyperparathyroidism.

DR. ANISHA GUPTA
Dr. Anisha Gupta is originally from South Florida and attended the University of Miami for both her undergraduate and medical school training. She then completed a residency in Internal Medicine followed by a fellowship in Endocrine, Diabetes and Metabolism at Baylor College of Medicine in Houston, Texas. She is board-certified in Internal Medicine. She has joined the Texas Endocrinology Group in Houston, Texas where she sees general endocrine patients with a focus on obesity medicine.

DR. JAD G. SFEIR
Dr. Jad G. Sfeir is an Assistant Professor of Medicine at the Mayo Clinic College of Medicine and Science in Rochester, Minnesota. After completing his training in Endocrinology, he is currently training in Geriatric Medicine and studying the effects of aging on bone and mechanisms of targeting cell senescence to prevent age-related bone loss at the Robert and Arlene Kogod Center on Aging.

DR. DACE L. TRENCE
Dr. Dace L. Trence is Director of the Diabetes Care Center and Professor of Medicine at the University of Washington Medical Center in Seattle. She is also the University of Washington Endocrine Fellowship Program Director and Director of Endocrine Days, a medical education program for endocrinologists practicing in the Pacific Northwest. She has served as Editor-in-Chief of EmPower Magazine since 2011.

DR. ALAN WONG
Dr. Alan Wong is an endocrinologist practicing at The Everett Clinic/Davita near Seattle, Washington. He received his medical degree from Drexel University College of Medicine (then MCP-Hahnemann) in Philadelphia and completed an Internal Medicine residency at Cleveland Clinic and his Endocrinology fellowship at Oregon Health & Science University (OHSU).
If you have been diagnosed with diabetes, would you like to know your blood sugar reading without having to do a finger stick? Would you like to receive automatic alerts if your blood sugar is dropping too quickly? Would you like to be able to share your blood sugar readings with friends and family? Would you like to be able to lower your A1C – a measure of the previous three months’ average blood sugar – without experiencing more frequent low blood sugar readings?

All of this is possible today with a continuous glucose monitoring (CGM) system. And depending on your insurance plan, the cost of a CGM might actually be covered by your policy.

A CGM is a device that continually tracks blood glucose (blood sugar) levels in the interstitial tissue fluid, the fluid that surrounds the cells of your tissue below your skin. The CGM contains a small sensor wire that is inserted under your skin, usually on your belly, to take blood glucose readings on set intervals. The wire is attached to a pad on the skin surface that acts as a receiver of information. A chemical reaction occurs on the wire in response to the fluid, which the receiver then sends wirelessly to either a monitor, smartphone display or tablet, allowing you to track your blood sugar average over the previous minutes without performing a finger stick. Sensors are changed every several days (timing depends on the device brand), which you can conveniently do yourself at home.

There are currently three FDA-approved CGM systems available in the U.S. They are the Dexcom CGM, the FreeStyle Libre and Medtronic Connect.

The beauty of CGM technology is that it allows you to see your glucose level any time at a glance and how your glucose changes are trending over a few hours or days. Accessing your glucose levels in real time and how they are trending can help you make more informed decisions throughout the day about how to balance your food, physical activity and medicines.

Some people think only those on insulin pumps benefit from a CGM system. This is not true. CGM systems have been shown to also benefit patients using multiple injections of insulin per
day to manage their diabetes. So, if you require at least three injections a day—a combination of basal or long-acting insulin along with bolus or mealtime insulin—you could be a candidate for a CGM.

Research bears this out: In a recent trial, people with type 1 diabetes using injections of insulin wore a CGM system. Prior to wearing the CGM system, study participants were checking their blood sugar, on average, five times a day (many diabetes specialists often recommend four to six blood sugar checks a day). After 24 weeks, patients on the CGM system had better blood sugars on average and without increasing episodes of hypoglycemia (low blood sugar). This is an important finding given what we know about people with diabetes benefiting from lower, near non-diabetes-range blood sugars.

Plus, the A1c blood value (a measure of the previous three months’ average blood sugar) is a marker for the risk of developing diabetes complications such as eye, kidney and nerve damage. So, if you have type 1 diabetes, have been checking your blood sugar four to six times a day, and aren’t happy with your A1c value, perhaps a CGM system might be right for you.

Another reason to use a CGM system would be if you have hypoglycemia unawareness or severe hypoglycemia. Hypoglycemia unawareness is the loss of the ability to sense that blood sugar is dropping until the sugar is critically low. It typically occurs with people who have had diabetes for a long time and have frequent low blood sugars. This can be a very dangerous condition. Severe hypoglycemia is typically described as a person experiencing low blood sugar that requires assistance from another person to treat.

CGM benefits a user optimally when it is used continuously. Studies show that the improvement in blood sugar control wearing the CGM are lost when participants stopped wearing the CGM device. This is important to note if your insurance company decides to stop covering your CGM system because your diabetes went from uncontrolled to controlled on the CGM. In this circumstance, your doctor can advocate on your behalf with this information.

As with all technology, using a CGM takes some getting used to. One nice feature of CGM systems is that not only will they tell your current blood sugar reading, but also will let you know if your blood sugar is changing and in what direction. So, if you are going to eat and self-administer insulin but see that your blood sugar is 150 and dropping, you may give yourself less insulin. If instead your blood sugar is 150 and rising, you may decide to give yourself a little extra insulin with your meal. These adjustments can help fine-tune your diabetes management and might require a discussion or two with your diabetes clinical team as to what extra dosing (or less dosing) guidelines would be the best.

When you see your endocrinologist, be sure to bring your receiver to the office visit if you hadn’t already downloaded your device information to the Internet the night before the visit. The data helps your care team make the appropriate adjustments to your diabetes management program. Data can also be reviewed and analyzed between visits if you are having concerns with your diabetes control, but only when specific trends can be seen.

While CGM has proven its value to patients since its introduction in 2000, the device can be costly when not covered by insurance, especially for those who are older and on a limited income. Some good news on this front occurred in 2017, when the U.S. Centers for Medicare and Medicaid Services (CMS) issued regulations allowing for coverage for CGM systems as “Durable Medical Equipment” under Medicare Part B, just like glucose meters. Until then, people with diabetes on CGM systems who aged into Medicare faced a tough decision, because the cost of the CGM systems were not covered. Medicare’s change in coverage meant patients who had benefitted from CGM would no longer be faced with this difficult decision.

There are, however a few specifics that must be met in order for a system to be eligible for Medicare coverage. First, it needs to be “therapeutic.” This means the blood sugar readings from the CGM system are accurate enough that a corresponding finger stick is not needed for the patient to act on the reading. There also has to be a non-disposable component to the CGM system which would last for three years—the receiver, for example. Two FDA-approved CGM systems meet this criteria: FreeStyle Libre and Dexcom CGM.

Also, in order for a Medicare patient with diabetes (either type 1 or 2) to receive coverage for a CGM system, he/she must be injecting insulin at least three times daily or using an insulin pump and checking their blood sugars by finger sticks at least four times a day, supporting the understanding that frequent adjustments in insulin dosing are being done based on blood sugar readings. Patients must also see their doctors every six months at minimum.

It’s hard to believe we’ve gone from testing a person’s urine in the doctor’s office to using technology advanced enough to predict if a blood sugar is going up or down, all without a finger stick. And CGM systems continue to improve rapidly. Measurement accuracy has more than doubled since the technology’s debut, device size and costs have been reduced, and patient and caregiver satisfaction with CGM therapy has increased. So, as you consider your diabetes care options, make sure to speak with your endocrinologist about whether a CGM should be in your diabetes toolbox.

EMPOWERYOURHEALTH.ORG
IN THE 19 YEARS since the Food and Drug Administration first approved continuous glucose monitors (CGM) for use in the U.S., the still-evolving technology has become a potential game changer in diabetes care.

The small, wearable device consists of a sensor placed under the skin that relays information to an attached external transmitter that tracks blood glucose (BG) levels in the cellular fluid at regular intervals, 24 hours a day, without the need for a finger stick. The resulting contextual data, sent to a separate receiver, allows patients and – by extension – their caregivers and their diabetes healthcare team, to proactively manage glucose highs and lows while providing added, real-time insights about the impact that food intake, exercise, illness and even everyday stressors have on an individual’s BG levels, thereby reducing the guesswork that often goes into glycemic control decisions.

Despite CGM’s capacity to provide “actionable” BG information to guide daily diabetes management (and thereby decrease the potential for devastating disease complications), widespread acceptance of CGM has been slow-going, some would suggest. First, each CGM device manufacturer has its own proprietary software and, thus, produces a different report. The resulting lack of uniformity and standardization in CGM reporting has created significant challenges for healthcare professionals and their patients in how to best use CGM-generated information to improve everyday diabetes care.

“Additionally, physicians don’t fully understand CGM use or think it may require too much effort, plus many people living with diabetes and their caregivers are not aware of the CGM technology, how it works and the many benefits it may provide them,’ says Nancy J. D’Hondt, a clinical pharmacist and immediate past president of the American Association of Diabetes Educators (AADE).

That’s all about to change.

A collaborative of powerhouse organizations in the diabetes health care space is intent on taking CGM use to the next level, joining forces in an unprecedented effort to ensure all diabetes health care professionals and patients have the information and tools they need to make the most informed clinical decisions possible about CGM use.

Known simply as IDEA (Innovating Decisions and Empowering Action in Diabetes), the focus of the multi-year initiative is to create and deliver comprehensive, easy-to-understand educational/awareness materials and learning aids to stakeholders across the diabetes care spectrum, with materials designed for patients, diabetes specialists, primary care physicians, nurse practitioners and physician assistants, and educators such as registered nurses, pharmacists and certified diabetes educators. The materials will provide the uniformity necessary for CGM use to reach its full potential, notes D’Hondt, a member of the IDEA steering committee.

“More widespread use and understanding of CGM can have benefits for all,” she says. “For physicians, it can help guide medication change therapies or insulin dosing and determine what’s working and, possibly, what’s not. For people living with diabetes, it will improve understanding of what and how food, activity, stress, medications and life in general affect blood glucose, mood, sleep and health. And family
members will be able to monitor glucose values remotely and ensure safety for their loved ones.”

At present, A1c – a measure of a person’s average blood glucose levels over a two-to-three-month period – is widely used and generally considered to be the gold standard in assessing blood sugar control to mitigate long-term risks of complications from high blood sugar levels. However, A1c can’t capture other critical data that matter to a patient on a daily basis, for example, low blood sugar (hypoglycemia) episodes that can be potentially fatal.

“This effort will shift the focus of diabetes management from A1c-centric to time-in-range-centric blood sugar goals and thereby improve overall outcomes and improve the lives of people living with diabetes,” says J. Hyun (C.J.) Chun, president of the American Society of Endocrine Physician Assistants (ASEPA), also a member of the IDEA steering committee. (Time in range is the percentage of time that a person with diabetes spends in the ideal blood glucose range.)

Although the IDEA program team began its activities in early 2018, the groundwork for the initiative was actually laid several years ago when the American Association of Clinical Endocrinologists (AACE) held a CGM Consensus Conference to examine the growing body of evidence supporting CGM benefits and develop strategies for overcoming barriers to CGM use and access.

“We asked ourselves, ‘How do we have a call to action to develop a unified approach to interpretation of CGM data that we could use to manage a patient’s diabetes, realizing, of course, that patients are being managed by a variety of healthcare professionals, and who is going to teach the patients and those providing the care how to capture the real-time data to enhance understanding and use of the technology?’” recalls endocrinologist Dr. George Grunberger, an AACE member and IDEA project steering committee chair.

“From there, the idea was to first get buy-in and agreement from each CGM device manufacturer to develop a unified report that could be used for educational purposes by everybody, from endocrinologists to primary care physicians to diabetes educators and so forth, so that the patient could be properly educated and caregivers could define what comes next in that patient’s treatment routine,” he adds.

Despite the highly proprietary nature of CGM manufacturers’ technology, all of the companies in the arena agreed to the proposition.

Another key development was the involvement of the International Diabetes Center, which owns the copyright to the Ambulatory Glucose Profile, the report forming the backbone for the IDEA unified CGM report.

The AACE team then identified key organizations that had a stake in the issue and solicited their participation in the program.

“Buy-in was quick,” Dr. Grunberger says. “It was impressive and exciting to see how many organizations enthusiastically joined almost immediately.”

Program participants include AACE, AADE, American College of Clinical Pharmacy (ACCP), American Diabetes Association (ADA), ASEPA, College Diabetes Network (CDN), Diabetes Patient Advocacy Coalition, Endocrine Society, JDRF, The Leona M. and Harry B. Helmsley Charitable Trust, International Diabetes Center (IDC), T1D Exchange and The diaTribe Foundation/Close Concerns.

“Right now there’s a lot of work to be done,” Dr. Grunberger notes. “We are finally organizing all these pieces and gathering content for the learning program launch, which is planned in late 2018.”

“The project is now taking shape and has the potential to change how doctors help their patients and how people with diabetes can better manage their blood glucose safely and feel better as a result…”

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To learn more about the benefits CGM has provided patients, read on for some first-hand CGM user stories.

(Continued on page 8)
THE CASUAL OBSERVER, Carol Logan might seem an unlikely fit for a person at risk for diabetes.

An accomplished linguistics teacher who has lived, worked and traveled across the globe, from France, Yugoslavia and Finland to England, Greece, Russia and Israel, Logan has led an active, productive life and is fit, trim and exceedingly health conscious.

So nobody was more surprised than Logan herself when she was diagnosed with type 2 diabetes in July 2001, at the age of 58. “I had none of the telltale signs of uncontrolled diabetes at the time of my diagnosis,” she notes. “I wasn’t thirsty, I wasn’t urinating excessively, I hadn’t lost weight, I wasn’t hungry. In fact, I found out about it from a random blood sugar test.”

Logan had been proactively having her blood tested every six months at her local pharmacy due to an extensive history of heart disease on her father’s side of the family. “They did a routine fasting blood sugar in addition to testing my cholesterol levels, which were excellent, as they usually are,” she says. “But they advised me at the time that my blood sugar was a little high.”

An A1c test done shortly thereafter – a measure of a person’s average blood glucose levels over a two-to-three-month period – was also slightly high, but still within a normal range, Logan says.

“My CGM Story:
CAROL LOGAN
Minneapolis, Minnesota

To my horror, I found that every time I tested myself after a meal, it was 170 or 180,” she recalls. For most people, a normal fasting (no food for 8 hours) blood sugar is between 70 and 130; a normal blood sugar level two hours after eating is less than 140.

“So, they put me on all different kinds of oral medications for type 2 diabetes to try to get my blood sugar back to normal, because they thought I had type 2 diabetes,” she says. “The medications never worked. I was constantly testing myself, and my results were always abnormal.”

In January of the following year, frustrated with the lack of results, Logan told her doctor she was willing to consider insulin therapy. Ultimately, Logan was correctly diagnosed with type 1 diabetes through a C-peptide test that indicated her body was producing no insulin. “And that’s when I started taking shots,” she says.

“In my case, I knew all about diabetes therapy because we had had a baby with the disease who was on insulin shots, so I wasn’t afraid of them,” Logan says. (Her son David now in his 40s, was diagnosed with type 1 diabetes at the age of 20 months, although nobody on either her side or her husband’s side of the family had ever been diagnosed with the disease.)

She continued conventional insulin therapy and tested her blood sugar frequently to stay on track in the ensuing years. “I’ve always been an advocate of tight control to avoid the horrible side effects of type 2 diabetes and know exactly what all of my blood sugars are at any moment,” she advises. (Continued on page 13)
When children with type 1 diabetes experience the everyday fun and freedom of camp with others just like them, something incredible happens. Diabetes isn’t the focus of their day. Lilly Diabetes believes that every child should have the opportunity to go to camp, and that’s why we’ve provided insulin and a variety of carefully designed resources to diabetes summer camps for more than 10 years. We help camps care for your child’s unique, personal needs so your child can focus on what’s most important — having a summer to remember.

LillyDiabetes.com
To register for a camp near you, visit www.diabetescamps.org.
DAY-TO-DAY LIFE CHANGES instantly for people when diagnosed with a chronic medical condition like diabetes. It often means big changes for their loved ones as well.

And so it was with Stacey Simms and her family – daughter Lea and husband Slade – when her youngest child, son Benny, was diagnosed with type 1 diabetes in December 2006, just before he turned two.

“I was slightly familiar with diabetes, and Benny had all the classic symptoms,” says the award-winning broadcast journalist. “Through the radio station I worked for, I had been involved in several local JDRF (formerly the Juvenile Diabetes Research Foundation) events, so I was familiar enough with the signs that I called my pediatrician and suggested our son needed to be tested.”

At the time of his diagnosis, the toddler’s A1c (a measure of average blood sugar levels over a two-to-three-month period) was 11.5. A level below 5.7 percent is considered normal. His blood sugar was over 800; less than 140 is considered normal.

“It was straight to the pediatric hospital, but he responded to treatment really well, so they only kept us for three days,” Stacey recalls. “He felt pretty good and was running up and down the hallways. The nurses were so happy, because a lot of kids with the same diagnosis don’t feel well enough to do that.”

Once the family brought Benny home, it was another matter entirely.

“Type 1 diabetes in a toddler is a very, very different thing,” Stacey notes. “The first two weeks were horrendous. He would run away from us, he would cry, he didn’t want the shots, he didn’t want the finger sticks, but after two weeks he began to adjust beautifully. Kids that age get used to things very quickly if you just get on with it.

“Our care at the time was to give fast-acting insulin when he ate, but our endocrinologist didn’t supply us with a strict eating plan, so whatever he ate, we gave him a shot to cover for that,” she continues. “He was up to six or seven shots a day, but he didn’t care how many shots a day he got, as long as he didn’t have to stop playing or interrupt his ‘fun’ time.”

Still, challenges remained.

The family had a big scare only a month after Benny’s diagnosis during an outing to visit relatives. “We were all at an indoor play area and I thought he was taking a nap, but I decided to check his blood sugar just in case,” Stacey says. “I don’t recall the exact number, but it was definitely under 50.

“We were terrified because it was so soon after we found out he had diabetes, and we were running to grab juice and gummies, but he was too tired to eat the gummies, so we went back to the juice to increase his blood sugar,” she continues. “After the episode was over, I remember thinking at the time how amazing it was that he bounced back from the low sugar so quickly.”

“Still, the most difficult thing was that we never knew if he was falling asleep or if he was experiencing a low, because most children that age can’t articulate how they’re feeling. No two-year-old says, ‘I have an ear infection,’” Stacey recalls.

“You’re checking blood sugars through your child’s crib’s slats at night, and finger-sticking before, during and after any ac-
tivity, even if it was only half an hour, because his blood sugar levels could go down very quickly from 120 to 40," she adds. "After all, he only weighed 35 pounds! It's a combination of heartbreakingly funny in an odd way."

Over time, Benny's care became easier and the family settled into a routine, even though there were still ups and downs. And Benny eventually transitioned from shots to an insulin pump.

Meanwhile, Stacey had become a passionate advocate for her son and others with diabetes (a so-called “D-mom”), joining the local JDRF board in Charlotte six months after Benny's diagnosis and blogging as an outlet for the emotions and challenges the family was experiencing. "It lifted the burden just to share it, to realize that we were not alone," she says. That yearning for a sense of community ultimately led Stacey to launch her own podcast, Diabetes Connections (https://www.diabetes-connections.com/). The popular weekly talk show includes a mix of interviews with prominent diabetes advocates, authors and speakers, researchers, and people with diabetes, their friends and family members.

Stacey's involvement with the diabetes community, as well as Benny's participation in camps for children with diabetes, initially made the family aware of CGM technology, “We saw people using the gear, but Benny didn’t want something else on his body, and we didn’t want to push it,” Stacey notes. “And then he came home from camp in 2013 after seeing another child using CGM, and said, ‘I want to try this.’”

Her son began using CGM Christmas day 2013, “and it was just INCREDIBLE,” Stacey says. “We all could see his blood sugar number on the receiver and that was such an eye-opener. It was so interesting to see and absorb, I remember we just stopped in our tracks and stared at it for the longest time.

“It took a while for us to learn how to use it, because when you finger stick, even six to eight times a day, or 10 times a day, as we often did with him, you’re not getting the full picture,” she adds, “but you’re also not overreacting. It took me a little while to learn how to not overreact because with CGM you get so much information that you didn’t have before, it’s an art form to find balance.”

With the advent of CGM remote monitoring, in which CGM data from a receiver can be stored on a web-based server instead of specific devices, allowing multiple caregivers to remotely view a person’s glucose data in real time, there were more lessons to be learned...and gains to be had.

Although acknowledging her instincts after Benny’s diabetes diagnosis were to “put my child in bubble wrap and never leave his side,” Stacey and Slade had agreed that teaching their children independence and confidence were priorities. “So, our remote monitoring of Benny was, and continues to be, a whole other bag of beans. We've got his blood sugar numbers on our phones, we know our child can handle it, but at the same time he's showing as having a high and you want to tell him, ‘knock that down.’ However, you can’t text your child 400 times a day. It’s taken some really honest conversations to work out a system that works for everyone in our family.”

The good news? Benny is now an active, strapping 13-year-old living the life of a typical teenager, thanks, in part, to CGM technology.

“CGM has taught us so much about how insulin works for Benny, when to dose and how certain foods affect him,” Stacey says. “My husband is one-half Italian and owned an Italian restaurant for many years, so food really plays a big part in our family’s life. If we're going to eat a high-carb or high-fat meal, CGM helps tell us how his body reacts so we can time his insulin a lot better, and he doesn’t have to give up his favorite foods. It’s also helping us ride the swings of puberty.

“CGM has become integral, almost essential, to our care.”
Mike Ratrie's pathway to continuous glucose monitoring (CGM) use was a long time coming.

Forty years, to be exact.

Diagnosed with type 1 diabetes (T1D) in 1973 at the age of 19, the active, outdoor enthusiast was being cared for by an internist who prescribed intermediate-acting NPH insulin once a day and was enduring “dozens of daily finger sticks” to assess his blood sugar levels. “Keep in mind, this was back in the day of peeing on test tape or measuring urine sugar level in test tubes,” Ratrie says, “long before there were meters for measuring blood sugar.”

Remarkably, Ratrie’s diabetes regimen remained consistent for more than two decades. Over time, however, his insulin dosage had increased from 35 to 40 to 55 units per day, “and I still wasn’t feeling that great,” he recalls.

Following his internist’s retirement, and after hearing from a male acquaintance about his state-of-the-art T1D care, “that got the wheels turning,” Ratrie says. “I did some research and discovered endocrinologists who specialize in treating patients with diabetes. I didn’t even know such an animal existed.”

Shortly thereafter, he made his first visit to a diabetes specialist. “She opened up a whole new world for me. She was truly, in diabetes care terms, really a world-class endocrinologist,” he says.

“She started me out doing finger sticks,” Ratrie recalls. “I came back after a month, and she took a look at my blood glucose numbers, which were mostly up in the 180 to 250 range, and she told me I really needed to be down below 120. And I was like, ‘Oh, no.’ At 120, I’m eating everything in sight because that’s too low a blood sugar for me,” he continues. “So she suggested we try to get the blood glucose down gradually.”

Ratlie’s endocrinologist also educated him about the complications that happen internally to a person with diabetes when their blood sugar isn’t under control. “Those were some really eye-opening times for me,” he says. “She wasn’t dogmatic, she talked about goals we could achieve together, as partners in my care.”

Despite their productive partnership, Ratrie metaphorically “moved on from her” for five years while he lived on a sailboat, traveling from Maine to Chesapeake Bay to the Bahamas and down to the tiny southern Caribbean island of Aruba. “But she was my endocrinologist from afar, as I was coming back to the U.S. once a year to see her and get prescription renewals, test strips and so forth,” he says. “I actually had an area on the boat that was dedicated to these huge containers where I had my test strips to check my blood sugar levels.”

After his return to the U.S. in 2011, he noticed people were starting to talk about CGM. “I was doing a lot of reading on the technology and was involved in a lot of online forums, but I was resistant to it initially because I was reviewing literature that talked about how you calibrated CGM with a blood stick device, which had about 20 percent plus or minus accuracy, and the logic wasn’t really striking me as being very compelling,” he says.

“I finally got to the point where I wanted to get more data, because even doing finger sticks 12 or 15 times a day, which is what I was doing, you’re only getting a snapshot,” he adds.
“Imagine trying to tell a story with 15 pictures, or with a movie that is a series of hundreds of pictures. That’s the difference.”

Ratrie began using a continuous glucose monitor in 2015... and has never looked back.

“I don’t recall if I was necessarily taking advantage of all of the information I was collecting, but I really picked up on the fact that this technology was going to help me immensely, because you can tell what’s going on with the blood glucose trending, which allows me to take corrective steps sooner,” he notes.

“What was eye-opening was seeing things I had been missing,” he continues. “For example, I would do a finger stick and have a picture of a post-meal number of 180, and then I’d see on the CGM that that’s not my peak. My blood sugar was still going up. And it really helped me understand I needed to be a little more aggressive if I hadn’t counted my carbs correctly, or if there was something going on with my metabolism that would tell me that I was going to be out of range, either high or low. The CGM helps me take advantage of the information and take steps sooner before I get in trouble from extreme blood sugar highs or lows. That’s really key.

“I’ve realized how much better my level of care is with CGM than it is with finger sticks,” Ratrie adds. “I can’t tell you before I started using a CGM what percentage of time I was spending over 180, but just as an example, in the past six months I’m spending maybe 3 or 4 percent of the time above 180.”

Ratrie’s CGM use was crucial to his most recent adventure, a 2016 cross-country bicycle trip with his wife, Lucia. He says the four-month, 5,885-mile journey in which they rode their ICE (Inspired Cycle Engineering) recumbent tricycles from Seattle, Washington through 15 states and back to their Florida home would not have been possible without his CGM.

“My CGM Story: CAROL LOGAN  (Continued from page 8)

It was through an Adults with Type 1 support group that she first learned about continuous glucose monitoring (CGM). “Word-of-mouth from the many people with diabetes I knew who had a CGM and who raved about how fantastic it was and how much it helped their control was what piqued my interest,” she says.

The technology had particular appeal due to a potentially dangerous condition Logan suffers from called hypoglycemia (low blood sugar) unawareness, a complication of diabetes in which the patient is unaware of – or doesn’t experience – the hallmark symptoms that accompany a deep drop in blood glucose levels. Hypoglycemia unawareness can lead to seizure, loss of consciousness, even brain damage. It also makes intensified blood glucose control more difficult and puts the patient at risk for severe hypoglycemia-related complications.

When she mentioned conversations about CGM with her fellow support group members to her endocrinologist, Logan’s caregiver agreed that it could be a valuable tool in her diabetes regimen. “Her concern was that with my having this unawareness problem, I could become unconscious while driving, for example,” Logan says. “But with the CGM, it starts beeping to warn you that your blood sugar is getting low.”

She began using the technology in 2008.

“The hardest thing about having diabetes is not taking your shots or testing your blood, it’s that you can never forget about it,” Logan concludes. “But still, it’s so much better now than when my son was diagnosed, when there was only urine testing available. I mean, it’s like a miracle when you compare the two time periods in history.

“To be honest, I can’t even remember what it was like in those days before I started using the CGM,” Logan says. “Because I’ve had diabetes for so long, it’s comforting to be able to look at the CGM at any time, day or night, and see what my blood sugar is approximately. And then if I have any doubts, I can always check my blood sugar with a finger prick. I still test myself about 6 to 8 times a day, and I don’t mind doing it.

“I’ve come to depend on it so much, if I ever happen to forget it and go somewhere without having it with me, it’s a horrible feeling,” she continues. “I feel like I’m naked. And I’m sure everyone who has a CGM feels that way.”
Can Aging Be Reversed?

By Jad G. Sfeir, MD, FACP

MEMORY DECLINE, arthritis, heart disease, hearing loss, diabetes, frailty, osteoporosis, decreased kidney function, cancer...as we grow older, it seems our bodies accumulate more and more ailments and diseases.

Aging is a process that seems to be universal, as all species – and all organisms within a species – experience it. It is also progressive and intrinsic, meaning aging is naturally built into our bodies, and we have no control regarding when and how it starts or how quickly it progresses.

In fact, cells in the body start aging as soon as we are born. And by our late 20s, many organs in the body start deteriorating and lose their full functioning potential. Plus, the older we get, the more we become frail, meaning that our ability to recover from injuries or stressful health-related events is diminished.

But is aging irreversible? The short answer, surprisingly, is: not necessarily!

We know that some species such as jellyfish can reverse the aging process and go through periods of growth and degrowth. Unlike the jellyfish, our bodies do not have the ability to reverse or stop this process. The aging process in humans leads to changes in the metabolism of our organs that accumulate over time and cause a number of chronic diseases such as those mentioned above. However, recent advances in medicine are leading to insights about how aging develops and, even more important, how it can be delayed or slowed down.

So how can you give your internal organs a “facelift”?

Rather than aiming to live longer, perhaps the goal should be to live healthier. It is an important distinction that researchers in the field make between “lifespan,” which is how many years we live, and “healthspan,” meaning the length of time we live in good health. Unlike Meryl Streep and Goldie Hawn’s characters tried to do in the film “Death Becomes Her,” we aim to “add life to years rather than years to life,” as renowned aging researcher Dr. James Kirkland said.

Many factors contribute to how long and how healthy we live. Our genetic makeup is probably the most important of these factors. Jeannette Calment, from Arles in southern France, has the longest confirmed lifespan of a human, having survived all members of her long-living family. She died in 1997 at the age of 122 years and 164 days. She must have good genes, one would say! She probably did. In fact, hundreds of genes have been discovered that can impact longevity. But there are additional factors such as our diet and the environment in which we live that also impacts longevity.

Take, for example, the small Italian town of Acciaroli in the Mediterranean basin: It has a population of 2,000 people of whom about 300 are 100 years of age or older and are free from dementia or heart disease. Scientists proposed many theories...
to explain this phenomenon, including the seaside weather in that town and the (now-famous) Mediterranean diet. Or maybe the Mediterranean Sea is feeding into a fountain of youth in Acciaroli!

**How can we get to the fountain of youth?**

Until now, chronic diseases associated with aging have been treated individually, using a one-disease, one-drug-class approach. For example, bisphosphonates (such as Fosamax, Actonel, Boniva) are used to treat osteoporosis, antihyperglycemic agents (such as metformin, glimepiride, sitagliptin and empagliflozin) are used for diabetes, and so forth. This leads to an increasingly large number of drugs taken simultaneously by one person who requires treatment of multiple conditions (called polypharmacy), with an increased chance for drug interactions and potential drug side effects.

But what if we find a common pathway to all these aging-associated chronic conditions and diseases that can be targeted with a single drug? Multiple research studies around the world have identified the fundamental process of aging to be behind a number of changes in our bodies, such as inflammation, poor metabolism, and damage to the cells and DNA that eventually lead to development of many age-related diseases. By targeting this common process, we can theoretically slow or prevent many of these diseases with one single drug.

This common pathway is what is known as “cell senescence.” It means that, as we grow older, the cells in our body accumulate damage over time and retire from their normal function; they don’t die, but rather become “senescent cells,” just like a zombie, some would say. They accumulate in many organs and tissues in the body and now have a new role: releasing a number of chemicals, known as SASP (Senescence Associated Secretory Phenotype), that can cause a number of metabolic changes, such as resistance to insulin that eventually leads to diabetes and bone loss that eventually leads to osteoporosis.

It’s important to realize that these senescent cells are not all bad. They are important in healing wounds. Also, if the cells that accumulate damage over time don’t retire, they may turn into cancer! This means that the best way to improve the healthspan is to let them retire and then either kill them or block the release of the SASP chemicals. That way you prevent the damaged cells from turning into cancer, but at the same time prevent them from causing chronic diseases.

Over a dozen senolytic drugs (from the words “senescence” and “lytic,” which means destroying) have been discovered and, when tested in mice, have proven to be effective. When old mice received these drugs, their number of senescent cells went down and the amount of SASP they release decreased. After treatment with these drugs, their bone strength improved, their cardiovascular function and insulin sensitivity – how well the body responds to the effects of insulin – also improved and they had fewer symptoms of frailty.

As the drugs that clear the senescent cells, senolytic drugs work using a “hit-and-run” approach: They are given once to kill the senescent cells, and then the tissue is allowed to rejuvenate over several weeks before another dose of the drug is given. Senolytic drugs do not affect normal healthy cells.

These drugs have the potential of changing medicine as we know it today. However, it will be some time before they make it to the market. What works in mice does not always work in humans, plus there might be side effects in humans that have not been seen in mice. So, although experiments in humans are already underway, these drugs are not ready for prime time yet.

Another area where senolytics can be useful is preventing side effects related to radiation therapy and chemotherapy – drugs used to treat cancer and HIV. While radiation kills cancer cells, it also causes damage to normal, healthy cells and increases the number of senescent cells in tissues. Using senolytic drugs can prevent this. For instance, in mice, if you treat one leg with enough radiation, after three months, the mouse has trouble walking. If you give a single dose of senolytic drugs, they are able to walk quite well.

**At what cost?**

Senolytics have to go through the clinical trials process. The first step in human experiments is to test the safety of these drugs. Researchers will then have to identify the best dose and frequency that will give the anti-aging benefit while minimizing the number of side effects such as poor wound healing. Next, the drugs will go through the U.S. Food and Drug Administration (FDA) approval process. Once approved, they will be available for widespread use.

Some scientists expect that these drugs will cost as much as some chemotherapy agents, which can be costly. On the other hand, they will theoretically decrease the number of hospitalizations. The overall savings in healthcare will possibly offset the cost of these drugs by a long shot.

There’s no doubt exciting developments in this area of science are underway, so be on the alert to new information and developments as researchers explore how to live well in our “senior days.”
By Dace L. Trence, MD, FACE

EMPHASIS ON LOOKING YOUTHFUL and feeling energetic surrounds us all. Whether in magazine ads, on TV, or the Internet, photos of people who appear to look decades younger than they reportedly are and articles about vigorous 90- and even 100-year-olds with energy that seems boundless – who cannot help thinking, “I want some of what they have!”

We’ve all heard about hormones that claim to offer the benefits of a proverbial fountain of youth. But what is behind these claims? What support or hard science is there behind those ads for DHEA, growth hormone, estrogen and testosterone? Here we examine the facts.

DHEA

DHEA, or dehydroepiandrosterone, is a hormone that is normally produced by your adrenal glands. Blood levels of DHEA naturally decrease after about age 25 to 30, and by age 70, DHEA levels typically have fallen by about 80 percent. People with certain major chronic diseases tend to have more rapid declines in DHEA, so it’s tempting to wonder if replacing DHEA to match that of younger age levels could be beneficial. To that end, DHEA supplements are sold as over-the-counter, non-prescription medication in the U.S. – which many people think are safe to use. However, in other countries such as Canada, DHEA is only available by prescription.

DHEA is typically advertised as an aid for people who want to “reverse” aging, boost their immunity, improve their brain function and mental sharpness, and improve muscle strength. But studies do not support DHEA actually providing any benefit for these functions. DHEA has been studied as a treatment for uses ranging from preventing the development of cardiovascular disease to delaying or treating the symptoms of menopause. Even prevention of Alzheimer’s disease has been proposed as a DHEA benefit, although clinical results are murky at best.

The appeal seems to be that DHEA, at times called a “parent” or “master” hormone, is converted into other hormones, specifically testosterone and estrogen. Many of its so-called benefits (and possible risks) are attributed to its potential conversion to these hormones.

However, DHEA does not have the same effect in everyone. The chemistry is complex, and the results can vary consider-
ably and can be unpredictable. One of the better studies on DHEA took place approximately a decade ago. In this study, elderly men and women were given daily DHEA – 75 milligrams for men and 50 milligrams for women – to achieve blood levels present in young people of the same gender. Measurements were taken over the course of two years for muscle strength, exercise tolerance, quality of life, and measures of insulin sensitivity. The expansive study did not support claims that taking DHEA showed benefits.

It was noted, however, that the elderly women in the DHEA group had a small but significant increase in bone density at the wrist, while men in the DHEA group had an increase in bone density at the hip. Study investigators felt that the very limited bone effect did not support the value of DHEA for either preventing or treating osteoporosis in elderly men or women, but they added that there did not appear to be any side effects with the medication.

One form of DHEA supplement known as 7-Keto has been advertised as an aid for reducing body fat and increasing metabolism. The idea is that leaner body tissue and higher metabolism will burn calories more efficiently, making it easier to not only lose weight, but to keep it off as well. Unfortunately, most studies have shown little effect from DHEA in losing weight or increasing metabolism.

Plus, there are the potential side effects to be considered:

- Oily skin and acne, as well as skin thickening
- Hair loss
- Upset stomach
- High blood pressure
- Changes in menstrual cycle
- Facial hair in women
- Deepening of the voice in women
- Fatigue
- Rapid or irregular heart beat
- Insomnia
- Unfavorable changes in cholesterol levels

**What about testosterone?**

Many drug commercials promise instant relief from a decreased sex drive, loss of energy and moodiness for men who have low testosterone levels. It appears that message has hit home as use of testosterone replacement therapy has skyrocketed during the past 10 to 15 years. As men age, it is natural for the amount of testosterone in their bodies to gradually decrease. This decline begins after the age of 30 and continues throughout life. According to the American Urological Association, as many as four in 10 men older than 45 have lower-than-normal levels of testosterone. This decline has been associated with symptoms such as loss of energy, reduced sex drive, erectile dysfunction, loss of muscle mass, loss of strength, loss of bone mineral density, increase in body fat, decreased sense of well-being, and several other undesirable effects that can dramatically decrease quality of life.

But, the results of replacing testosterone in those with low levels have been mixed. For example, the TEAAM study, a well-done investigation with rigorous qualifications, researched whether testosterone administration for three years would improve mind sharpness and other physical factors in men 60 years and older who had baseline low or low-to-normal serum testosterone concentrations.

The study findings included no significant change in how open major blood vessels were. Additionally, overall sexual function and quality of life were no different between the testosterone group versus the group taking a placebo (a substance that has no therapeutic effect, used as a control in testing drugs). But men in the testosterone group had side effects, including increased hemoglobin in the blood and increased concentrations of prostate-specific antigen (PSA), which can be an early indication of prostate cancer.

In a subset of the participants, tests that evaluated mind sharpness and thinking ability showed no significant difference between the group taking testosterone and the group taking a placebo. These negative findings are the best evidence so far regarding the lack of effect of testosterone treatment on cognition in fairly healthy older men with low testosterone. One could argue that perhaps the results could be different in less healthy men, men with chronic illness, or perhaps even an older-aged group, but those studies remain to be done.

Possible side effects of testosterone therapy in men include:

- Increased acne
- Fluid retention
- Increased urination
- Breast enlargement
- Decreased testicular size
- Decreased sperm count
- Increased aggressive behaviors

*(Continued on page 18)*
Anti-Aging Hormone Supplements: Hope or Hype? (Continued from page 17)

What about estrogen?

Estrogen is another hormone that normally decreases with time in women and is known to be associated with bone density loss, skin changes, hair thinning and (perhaps) changes in mind sharpness. At menopause, hot flashes, sleep disturbances and vaginal dryness can prompt many women to consider whether hormone replacement therapy could be helpful for them.

By age 50, most women will have half the amount of estrogen they started with, and levels continue to fall after menopause. Why some women have severe low-estrogen symptoms and others don’t is unclear. In the 1990s, two of the largest studies of hormone replacement therapy were initiated. One was a randomized trial in the U.S. [Women’s Health Initiative (WHI)], and one was an observational questionnaire study conducted in the U.K. [the Million Women Study (MWS)]. The published results of these studies during 2002 and 2003 raised concerns regarding the safety of hormone replacement therapy (HRT). These concerns emphasized two main issues:

1. That the extended use of HRT may increase the risk of breast cancer, and;
2. The use of HRT may increase (and not decrease, as was commonly thought at the time) the risk of heart disease.

Subsequent analyses, particularly of the U.S. study results, have suggested that the results show a more complex picture than initially realized. The apparent increased risk for breast cancer was only found in those who had taken HRT before entering the study. In addition, the age of the participants made a huge impact in the results of hormone replacement. Re-analyses of different age groups showed no increase in heart disease in women starting hormonal replacement within 10 years of menopause.

Subsequently, a large controlled trial from Denmark in 2012 demonstrated that healthy women taking combined hormonal therapy for 10 years immediately after menopause had a reduced risk of heart disease and of dying from heart disease.

Studies regarding estrogen’s possible effects on mental sharpness have been contradictory, but generally do not suggest a benefit.

The effects of estrogen use on skin – increased skin thickness and improved skin moisture – have had the most positive results. In menopause, as estradiol levels decline, skin thickness decreases by over 1 percent each year for the first five years, while collagen – a protein in the body that helps give structure to our skin – decreases yearly by 2 percent. Wrinkles are caused by a reduction in elasticity secondary to loss of connective tissue, which decreases 1.5 percent each year. And the face is preferentially involved, as estrogen receptors are higher in the face than in the breast or thigh, for example.

Many wonder if skin changes are reversible with estrogen use. In one study, an estrogen cream applied to the face for 24 months produced significant increases in skin thickness and decreases in wrinkles, while a different study in which subjects took 0.01 percent estradiol (manufactured estrogen) versus 0.3 percent estriol for six months produced no changes in systemic hormone status, but both dosages increased skin elasticity, skin moisture and firmness, and reduced wrinkles.

Still, you may not need estrogen to enjoy this type of benefit. In study participants given either 0.01 percent estradiol, or 15 percent glycolic acid alone or in combination with estradiol, epidermal (skin) thickness was increased 23 percent by estradiol alone, 27 percent by glycolic acid alone, and 38 percent by both in combination. Glycolic acid, a weak acid with small molecules that penetrate the skin easily, dissolves oil gland secretions and other substances that hold superficial skin cells together, which encourages new cell development. Other agents with a better safety profile continue to be researched.

What truly keeps us youthful?

There are countless books, blogs and articles that offer advice on how to stay forever young. Some of the common themes include keeping physically active, eating more plant-based and low-fat foods, decreasing stress levels, and staying engaged socially.

Researchers at University College London asked approximately 6,500 men and women who were age 52 and older the question, “How old do you feel you are?” Of the group (whose collective age averaged 66 years), about 70 percent felt three or more years younger than their actual age. Twenty-five percent felt close to their actual age, and 5 percent felt more than one year older than their actual age. The really interesting association came eight years later, when the same researchers looked at who was still living.

The results showed:

- 75 percent of those who felt older than their age were still alive
- 82 percent of those who felt their actual age were still living, and
- 86 percent of those who felt younger than their age were alive

Suffice it to say, a positive attitude may not be the key to eternal youth, but it clearly can make an impact on how we age.
Point, Click, Learn.

Free educational resources featuring expert content curated by our own member endocrinologists to further patient understanding of endocrine-related health issues.
IN TODAY’S WORLD, I find that everyone has weight on their mind, whether thinner folks who can’t seem to put on pounds or those of us who can’t shed a single digit on that scale, no matter how much we try.

As someone who has struggled with weight all of my adulthood, I used to fall into that second category. I’d think, how do people do it? They keep a fantastic physique and eat it all, while I watched what I ate and still put on 30 pounds before even realizing it!

Despite going through medical school and intensive training to learn what affects metabolism, it has taken me until now to realize one important fact: weight loss is not a formula, it is not an algorithm. It takes an individualized approach to achieve success.

My story might be a very different one than yours...or maybe not.

I am an endocrinologist in Houston, Texas, but it has taken me five years of training and then some to learn a few important rules about weight loss. When it comes to losing weight, the key is to find something that works for YOU and to stick to it. Studies have shown that dieting is successful, but those studies don’t show which diet to follow for the “best” results.

I found that starting with simple calorie counting was a big eye-opener. I mentioned that I considered myself a healthy eater, yet I did not realize what I was putting into my body! Simply putting it down on paper (or, in my case, the free My Fitness Pal app) was the best thing I could have done to start identifying my “problem foods.” Chai lattes are not worth the hundreds of calories when a tea bag with honey does the job. I didn’t need mayonnaise on my sandwich, but instead maybe a handful of my favorite chips were worth the tradeoff.

Making these small changes, I started to see a difference. And really, that was what it took. I think years of running into the weight-loss wall of no pounds lost was taking its toll on me. I was fit, I exercised at least three times per week, was active at work and was eating healthy. Yet, I had seen no results. Now this seemingly little change of calorie awareness was making such a big difference. It was incredible! I realize now that it was...
simply putting control into my hands and not into the hands of my yoga teacher or my barista. I was controlling my food, I was feeling confident that I was making a positive change, and I felt like I could do more than ever before.

So, it was then that I finally tried one of the fad diets, Whole 30, just to see what it could do for me. And, not to my surprise, it was the hardest thing I have done for myself. Ever. But again, because of this newfound feeling of self-control, I stuck to it more than 90 percent of the days in the month. I also did not monitor my weight (the diet does not allow it), and at the end of 30 grueling days, I had lost 15 pounds! It was fantastic, people were noticing at work and asking ME what my story was. I was shocked!

I tried sharing my secret, but soon realized that people did not want to count calories or restrict five major food groups from their diet. It was just too hard. So, they juiced, or they fasted intermittently, or they cut carbs completely (ketogenic diet). But as I came across more and more success stories, I realized that they, too, were losing weight, toning their body, or maybe just feeling better in their own skin. And for me, that is what is important. I would love to lose another 10 pounds, but in the past 24 months, the scale has not moved. Still, I feel more fabulous now than before.

The other biggest learning point for me was that weight maintenance is much harder than losing the weight itself. Yes, I can juice or fast and lose 10 pounds quickly. But that is all water weight. Although that will make me look great in a dress two sizes smaller, I risk fainting and it just won’t last. The problem with fad diets is not that they don’t work. They do. It’s that they are near impossible to stick to. People love to chew their food and they love to taste it. Even if you are not a foodie as I am, it’s just a basic need. So eventually a fad diet will always fail. It doesn’t mean that you can’t pick up and lose the weight again after a short break, but wouldn’t it be better to take an approach to food that can be sustainable?

The first basic principle in losing weight is less calories in than out. Now, this is easier said than done by diet alone. But diet is where you should start. Making those changes in your meal choices means first recognizing from where you’re starting. There are many apps that can be helpful – My Fitness Pal, Loose it, CalorieKing. Check them out.

I mentioned that I like tea in the morning. I now choose a bag of breakfast tea with honey (22 calories) rather than my previous choice of a store-bought Chai latte (190 calories). For yogurt, avoid the “Fruit on the Bottom” variety and opt for the original flavor, but add your own honey or brown sugar with fresh fruit to save yourself another 100 calories. And for a heartier meal, go for breakfast tacos, but switch your tortilla out for lettuce. It still gives you the taco feel, with a little crunch and a whole bunch of saved calories. What a difference for the same delicious food!

In addition to diet, exercise should be a very important part of your lifestyle to protect your heart, muscles and overall health. You may find yourself feeling less fatigued, discover you have much more energy, and even have a better mood! Exercise also relieves stress, improves memory and helps you sleep better. So, find physical activities that you enjoy and commit to them.

Often varying the daily activity makes it more interesting. I’ve found a partner system works best for me. That might mean

(Continued on page 24)
The prevalence of osteoporosis, a condition of weak and brittle bones that predisposes you to fractures from commonplace trauma like coughing, twisting and falling from standing height, continues to be a serious public health concern. The “silent” condition doesn’t usually cause pain until you experience a fracture.

Worldwide, one in three women over age 50 will experience osteoporotic fractures, as will one in five men over 50 years of age. Fractures in older adults, especially hip fractures, can lead to prolonged hospital and nursing home stays, which in turn can predispose a person to life-threatening infections. A recent study found that the annual cost of caring for osteoporotic fractures exceeds that of caring for breast cancer, myocardial infarction, or stroke in women aged 55 years and older.

Why does it occur?

Formation of new bone and removal of old bone is a lifelong process. Osteoporotic bone can be thought of as resembling Swiss cheese, but with more holes as you age, and hence is weaker. Most adults reach peak bone strength and density in their 20s and 30s. After this, bone strength declines slowly with age.

The amount of maximum bone strength you reach influences your risk for osteoporosis. This, in turn, is influenced by both genetic and environmental factors. For example, if you are thin, Caucasian and female, with a family history of thin bones, you will probably achieve less peak bone mass. Long-standing calcium or vitamin D deficiency can lead to slow weakening of the skeleton. Plus, other medical conditions can predispose you to develop thin bones, among them kidney and liver disease, use of steroids (such as prednisone), rheumatoid arthritis, cancer and chemotherapy, and hormone issues like an overactive thyroid, parathyroid, or adrenal gland. Moreover, environmental factors that make you likelier to fall (such as stroke, impaired vision or hearing and muscle weakness) also increase your risk for fractures.

How is osteoporosis diagnosed?

A bone density study or DEXA (Dual-energy X-ray absorptiometry) assesses your bone mineral density, also known as BMD, which typically is very low in osteoporosis. Also, it can be diagnosed if you have a low-trauma fracture even without a DEXA study. A diagnosis of osteopenia on a DEXA study indicates "pre-osteoporosis" status, which is serious as well since the majority of osteoporotic fractures occur before a DEXA scan reveals osteoporosis. In this case, doctors may use an online risk calculator called FRAX® (fracture risk assessment tool), which estimates your risk of having a fracture. If fracture risk is high, then treatment is recommended, even for osteopenia.

What can you do for your bone health?

Guidelines created by experts from the American Association of Clinical Endocrinologists (AACE) recommend certain fundamental measures to ensure good bone health, including:

- Calcium intake (1200mg daily)
- Adequate vitamin D supplements (1000-2000 international units daily)
- Limiting alcohol intake
- Quitting smoking, and
- Regular exercise, including weight-bearing or resistance training. If you are older, physical therapy may be needed to help, especially with fall-proofing your home to make it safer.

What medications are used to treat osteoporosis?

Medications used to treat osteoporosis work by either preserving your bone (bisphosphonates, denosumab, estrogen, calcitonin or raloxifene), or increasing the formation of new bone (teriparatide).
Bisphosphonates (alendronate, risedronate, ibandronate, zoledronic acid) can be used as a pill or as an injection and are usually the first medications suggested since they are excellent at preventing both hip and spine fractures. However, they should not be prescribed to patients with kidney disease, or those who cannot tolerate bisphosphonates due to gastrointestinal acid reflux or allergic reactions. Denosumab is an every-six-months subcutaneous injection that prevents both hip and spine fractures and can be prescribed to those who are unable to take bisphosphonates. Another prescription treatment option is teriparatide (Forteo®, daily injection) which helps to stimulate the formation of new bone and may be particularly helpful in people with very low bone mass or those intolerant to other forms of therapy. However, it should not be prescribed to patients who have had radiation therapy to the bone or certain other bone diseases.

The second-line therapies approved in the U.S. are raloxifene, which reduces spine fractures although the effect on hip fractures is not as certain, and calcitonin, which reduces risks of spine fracture but not clearly in the hip and may also help relieve pain from acute spinal fractures.

How is treatment measured and monitored?

The goal of monitoring osteoporosis therapy is to identify patients who continue to have bone loss despite therapy. Ideally, monitoring should be done at the same center using the same machine and technologist as the previous DEXA scans and should include both hips and the spine.

- **Good response:** DEXA scan shows a stable or increasing BMD
- **Poor response:** DEXA scan shows significant decrease of BMD or a fracture while on treatment. If this happens, your doctor will make sure you are taking your osteoporosis medication adequately and will rule out any other conditions that might be affecting your bone health.

Usually, your doctor will check a DEXA scan every one to two years after starting the treatment. Once your BMD has been stable, you doctor may repeat the DEXA at longer intervals.

**How long should I be treated?**

Your FRAX score will determine the duration of the osteoporosis treatment.

- If your fracture risk is high, guidelines recommend continuing treatment with osteoporosis pills such as alendronate or risedronate for 10 years, or yearly infusions of intravenous osteoporosis medication (zoledronic acid) for six years. Based on current knowledge, it's yet unclear what the risks and benefits of treatment beyond 10 years.
- If your risk is low, your doctor may consider taking you off the medication after five years of BMD stability on osteoporosis pill treatment or three years of yearly infusion osteoporosis treatment (known as a “drug holiday”).

**What are the risks of osteoporosis medication?**

There are mainly two adverse events from osteoporosis medications that are of concern.

- **Osteonecrosis of the jaw** – Some cases have been reported previously that in high doses of osteoporosis medications, sudden decay of the bone cells of the jaw has occurred that does not heal. The probability of this complication happening now on the current doses is very low. In comparison, the risk of fracture in untreated osteoporosis is much higher than that of osteonecrosis of the jaw. Risk factors for this complication include dental disease, dental procedures, and poor oral hygiene. Your doctor may examine your teeth before prescribing osteoporosis therapy. If significant dental issues are detected, your doctor will consider delaying...
Fighting Every Pound: An Endocrinologist Gets Candid About Her Own Struggle With Weight Issues

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that you take walks during your lunch break with a coworker or colleague. For those of us who cannot find motivators at work to spare the shared time, find them among your friends and family who are struggling to do the same. For me, that looks like sending a text to my workout buddy after each activity, knowing that if she gets to her exercise first, it will only push me harder to do something as well. I know the biggest challenge of the gym is just getting there. It is so easy to make an excuse, so sometimes knowing that you just need to get it done no matter what can be the perfect amount of motivation.

So, if the weather is too rainy, too cold or too hot (as it often is here in Texas), find a nearby mall or storefront that can provide a comfortable place to do your daily walk. No stopping at those bargain sales bins, though!

As a busy physician, I understand that sometimes making time outside of work is, in itself, not possible. But I do find it.

As for exercise, I do my best, but on those inevitable days where I finish work late and can't even think about any sort of activity, I allow myself permission to target the next day as an “absolute, must-do” and take the night off. I understand that life does not always stick to a schedule, so to be successful I must find positive solutions instead of only worrying about the problems.

Regardless of how much weight you want to lose, or to keep off, set a realistic goal, contact your health care provider to make a plan, and stick to it. If your plan doesn’t work after a few weeks or you just can’t keep up, rethink your plan and try again. I have. Trust me, I am in the trenches with you!

AACE Guidelines- An Update on Osteoporosis Recognition, Treatment

(Continued from page 23)

osteoporosis therapy until these dental issues are addressed. For patients who are already receiving treatment and need dental procedures, there is no evidence that stopping the treatment will reduce the risk.

- **Atypical femur fractures** – These are fractures of the middle of the long bone of the thigh that happen with little or no trauma. It may happen after many years of treatment or after abruptly stopping medications without proper continuation of care. The probability of this complication happening is low, but should be suspected if sudden onset of groin pain is experienced, especially if treatment was stopped abruptly.

- **Atrial fibrillation** – While the benefit of bisphosphonates greatly outweighs the risk of developing atrial fibrillation, patients with pre-existing heart conditions like congestive heart failure or valvular heart disease should be aware of this possibility.

- **Esophageal cancer** – A rare potential side effect of oral bisphosphonates is esophageal cancer. However, nationwide, multiple large-scale studies have failed to provide evidence for any increased risk of esophageal cancer with the use of oral bisphosphonates. Oral bisphosphonates, if not taken appropriately (sitting upright, with a glass of cold water, and not lying down for 30 minutes after), can stick to the food pipe and cause ulcers. Hence, patients with preexisting conditions like esophageal ulcers, Barrett’s esophagus, or narrowing of the esophagus may be better served by IV bisphosphonates.

**Is treatment more effective using two or more osteoporosis medications in combination rather than using only one?**

Guidelines currently do not recommend the combined use of two osteoporosis medications. There is no proven benefit in either fracture risk or improvement in DEXA scan. Moreover, costs are increased and adverse effects could be higher.

**When should I be referred to a clinical endocrinologist or osteoporosis specialist?**

Your doctor may consider referral to a specialist if you have:

- Normal bone density on DEXA scans but sustained fractures without significant trauma
- Recurrent fractures
- Continued bone loss while receiving therapy
- Other associated diseases causing your bones to be brittle
- Severe or unusual cases of osteoporosis
- Other associated diseases (such as renal disease) which may complicate treatment
Looking for Resources to Assist With Your Prescription Medication Costs?

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PrescriptionHelp.aace.com

Presented as a public service by the American Association of Clinical Endocrinologists and the American College of Endocrinology
WILE THYROIDOLOGY, the study of the thyroid gland, may be perceived as a relatively new area of endocrinology specialty, references to the thyroid can be found as far back as the literature of the ancient Greeks.

Thus, the long history of thyroid disease and its treatment is also central to the history of much of the practice of endocrinology in general, particularly in early initial descriptions of its anatomy, physiology and associated pathologies, as well as the more recent identification of its molecular mechanisms of action, which have resulted in the identification and purification of thyroid replacement hormone.

We begin with goiter (enlarged thyroid), named after gutter, which is Latin for “throat.” Goiter has been long identified in iodine-deficient regions such as the mountainous regions of Europe and China and northern India (where it was known as galaganda) and subsequently by various writers including...
Hippocrates, Pliny and Galen. As early as 1600 BCE, Chinese writers described goiter treatment using burnt seaweed and sponge, as did European physicians, including Roger of Palermo and Arnold of Villanova in the 12th and 13th centuries, respectively. Goiter was not linked specifically to the thyroid gland, however, until Fabricius did so in the 17th century. Earlier writers had used various terms for goiter, including struma, scrofula, and bronchocele, without necessarily differentiating between tumors of the thyroid and those of other neck structures.

Galen, in the first century AD, was the first to describe the thyroid gland, but it wasn’t until the late Middle Ages that more complete descriptions of the thyroid gland itself emerged, first in China by Wang Hei, around 1475, and Vesalius in 1543. Vesalius, in fact, described the thyroid gland as two glands, one on either side of the larynx, but Eustachius a decade later noted that the thyroid was, in fact, a singular gland with the two lobes connected by an “isthmus.” The term “thyroid” (referring to the Greek for an oblong shield, thyreos) was first used by Thomas Wharton in 1675.

To these medical pioneers, the function of the thyroid was unclear. Various functions were ascribed to the thyroid, including a lymphatic function (the transportation of lymph, a fluid containing infection-fighting white blood cells, throughout the body), detoxification, a holder for worms, filling free spaces around the larynx, and guarding “the female system from the influence of the more numerous cases of irritation and vexation of mind to which they are exposed than the male sex (Rush, 1820).” However, the predominant theory for many years was from Roman Empire physician and philosopher Galen, who described the thyroid as a source of laryngeal lubrication fluid. Subsequently, the thyroid was thought to function as a “vascular shunt,” whereby the thyroid was thought to hold excess blood from the brain and thus prevent adverse consequences to neurologic tissue.

In the mid-to-late 19th century, physiologists such as Berthold, Bernard and Brown-Sequard identified the concept of the bloodborne “chemical messenger” enabling regulation of the functions of distant organs as well as general metabolic effects. In doing so, they established the foundation of modern endocrinology. “Organotherapy,” the identification of deficiencies of unknown “internal secretions” produced by “ductless” glands, coupled with treatment of these conditions by extracts of the corresponding gland, became more popular. The use of thyroid extract for hypothyroidism in the late 1800s was one of the earliest successful applications of this concept.

In 1905, British physiologist Ernest Starling described the term “hormone,” first used by his colleague Sir William B. Hardy and derived from the Greek for “to set in motion,” to describe such bloodborne chemical messengers.
Sir John Simon (1844) noted that the structure of the thyroid suggested a secretory function.

Studies of thyroidectomies in animals in the mid-to-late 1800s supported the vital role of the thyroid for sustaining life. Emil Theodor Kocher (1883), in work that won him the 1909 Nobel Prize in Physiology and Medicine, noted poor outcomes in patients who had undergone thyroidectomies for goiters in Alpine areas of Switzerland. Conditions such as cretinism – consisting of bone growth delay and small bones, dwarfism, mental deficiency (congenital cretinism), and goiter (with endemic cretinism); myxedema – a condition characterized by an increase in mucous in connective tissues and edema, first described by William Gull in 1874; and cachexia strumipriva, noted by dry skin, decreased mental abilities, decreased energy, facial and extremity edema, and muscle weakness, were increasingly thought to have a common cause.

In the 1880s, a trio of physicians linked the loss of the thyroid gland as the common underlying cause of these conditions based on anatomical studies and the effects of thyroidectomies in animals such as monkeys, sheep and dogs. However, thyroid hormone deficiency as the underlying cause was not yet known.

Improvements in the treatment of hypothyroidism followed. After studies in the late 1890s attempting grafting of thyroid tissue after thyroidectomy had produced temporary improvements, English physician George Redmayne Murray demonstrated in 1891 sustained and significant clinical improvement in a myxedematous woman injected with sheep thyroid extract, as did Portuguese investigators Bettencourt and Serrano in 1890. After oral thyroid extract was also noted to be effective by H. W. G. MacKenzie and E. L. Fox in 1892, oral treatments became the predominant form of thyroid extract administration.

The essential link between iodine and thyroid disease also became increasingly evident with time. Swiss physician Jean-François Coindet identified iodine as the probable agent in sponge that could treat goiters and started using it for this purpose in 1820. French chemist Jean Baptiste Boussingault, based on his experience in Columbia where he observed locals preventing goiter with salt iodized from a nearby mine, in 1833 suggested iodized salt as a goiter preventative. Unfortunately, initial attempts using iodine to prevent goiter were complicated by adverse effects such as Jod-Basedow phenomenon (high thyroid levels in persons with goiter using excess doses of iodine).

In 1853, French physician Gaspard Adolphe Chatin linked the lack of environmental iodine content with goiter, although his findings were initially disregarded. At that time, goiter was thought to originate from water-related causes or ex-

This anatomy drawing by Leonardo da Vinci in 1510 is considered the first drawing of the thyroid gland.
cess dietary magnesium. The iodine-dependent biochemistry of thyroid function also became clearer about this time. In the late 19th century, German chemist Eugen Bau- mann noted high iodine levels in the thyroid gland, and, in 1896, identified “thyreo-iodin” as the active agent in thyroid. In 1907, Dr. David Marine identified the requirement of iodine for normal thyroid function. The use of iodine was first demonstrated for goiter treatment in animals in 1913 and for greatly decreasing goiter incidence in schoolgirls living in an iodine-deficient region in 1917. The subsequent introduction of iodized salt, initially in Switzerland in 1920 and in the U.S. in 1924, greatly reduced iodine deficiency and the subsequent incidence of iodine deficiency-associated goiters.

In terms of thyroid hormones themselves, crystallized thyroxine (T4) – the main hormone produced by the thyroid gland – was isolated by American physicist Edward Cal- vin Kendall of the Mayo Foundation in 1914. And in 1927, chemist Charles Robert Harrington characterized the molecular structure of T4. The following year he and colleague George Barger synthesized T4, after which it became more widely available both in quantity and cost. It was observed, however, that T4 alone did not fully explain thyroid activity. In 1928, Kendall, noting that desic- cated thyroid extract, which is pig or cow thy- roid glands, dried and powdered for therapeutic use – had more effect than thyroxine, suggested the possibility that a more “active” form existed. This agent, T3, with much higher biologic activity than T4, was identified by biochemist Rosalind Pitt-Rivers and endocrinologist Jack Gross in London and Dr. Jean Roche in Paris in 1952.

Thyroid stimulating hormone (TSH), secreted by the pitu- itary to regulate thyroid hormone production, was identified in 1935 by J.B. Collip and E.M. Anderson. The presence of a thyrotropic substance (having an influence on thyroid gland secretions) of anterior pituitary gland origin was noted as early in 1916 by two young biologists, Bennet M. Allen and Philip E. Smith, working independently on tadpole models, and in 1929 independently by American physician Leo Loeb and Frenchman Max Aron, who demonstrated thyroid enlargement in guinea pigs given pituitary extract from cattle.

Subsequently, the concept of “feedback control” as applied to biologic systems was described, including the “pituitary-thyroid axis” in the 1940s and the “thyrostat” as described by R.G. Hoskins, Harvard Medical School Director of Neuro-Endo- crine Research, in 1948. This concept of “feedback” inhibition, using TSH to assess the adequacy of thyroid replacement doses, is central to current models of treatment for hypothyroid- ism resulting from thyroid function deficiency. More recently, TRH (thyrotropin-releasing hormone), originating from the hypothalamus in the brain and which regulates TSH produc- tion, was identified by Nobel Prize recipients Andrew V. Schally and Roger Guillemin in 1970.

The most common cause of hypothyroidism in the United States, Hashimoto’s thyroiditis (also known as Hashimoto’s disease), was first reported in 1912 by its eponymous discoverer, Japanese surgeon Hakaru Hashimoto. He described “struma lymphomatosa,” which he identified as goiters infiltrated by lymphocytes, and oftentimes hypothyroidism. His discovery, however, was not fully accredited until 1938, likely due to factors such as the original paper being in German and Hashimoto’s private practice in Japan being outside the confines of academics. We now know that Hashimoto’s thyroiditis is an autoimmune disorder and can be characterized both by enlarged, as well as small, thyroid glands. The identification of Hashimoto’s as an autoimmune disease was first suggested in 1956 when the associated autoantibodies were identified by clinical immunologists Ivan Roitt and Deborah Doniach.

(Continued on page 30)
Hyperthyroidism

Mayo Clinic co-founder Dr. Charles Mayo first used the term “hyperthyroidism” in print in 1907, but the distinction between the two major causes of toxic adenoma (a thyroid nodule producing excess thyroid hormone) and Graves’ disease was not clearly made until 1913, by endocrinologist Henry Plummer and Dr. Walter Boothby. Ophthalmopathy (eye protrusion) is a characteristic feature of Graves’ disease, although not always present.

Goiter associated with eye disease has been noted as far back as the 6th century AD. Persian physicians such as Avicenna also described it in the 11th century and Sayyid Ismail Al-Jurjani in the 12th century. Subsequently, the association of goiter, hyperthyroidism and eye disease as a singular disease didn’t occur until the early 1800s when multiple descriptions of goiter and exophthalmos (bulging of the eye out of the orbit) were published.

Caleb Hillier Parry, an English physician in Bath, described a series of thyrotoxic patients in 1786 (but published in 1825) and described the association of goiter, cardiac enlargement, and palpitations, including eye protrusion. Ultimately this disease was called “Graves’ disease” after Robert Graves, an Irish physician who described the condition in 1835. Although both Parry and Graves identified patients with Graves’ disease in their descriptions, neither identified Graves’ disease as a separate entity from other forms of hyperthyroidism. In Germany, Karl Adolph van Basedow described bulging of the eye, goiter, and palpitations in 1840, which he termed the “Merseburg Triad.” In Europe, Basedow’s name is the one associated with this disease. In 1856, Charcot also noted associated tremor. The suggestion of Graves’ disease being caused by excess thyroid production was expressed by Plummer and Boothby in 1924. However, the discovery of the cause, “long-acting thyroid stimulator (LATS)” didn’t occur until the late 1950s (D.D. Adams and J.M. McKenzie). This substance was noted to be an antibody in 1965, and the target of that antibody, the TSH receptor, was identified in 1978.

Early treatment of Graves’ disease mainly utilized iodide treatment or surgery. Iodide use for Graves’ disease was first described in 1820, by Swiss physician Jean-François Coindet. Nine years later, French physician Jean Lugol created the iodine solution that is named for him and still used in modern treatment. In 1948, Drs. Jan Wolff and Israel Lyon Chaikoff at the University of California described the mechanism of iodide exposure leading to inhibited organification of thyroid hormone in abnormal thyroid glands, thereby leading to decreased thyroid production. Subtotal thyroidectomy (surgical removal of the thyroid), sometimes accompanied by the tying off of thyroid arteries (ligature), was also described as early as 1823, although this practice became less common over time, while iodide treatment was increasingly utilized before surgery after Plummer at Mayo started doing so in 1922. In the 1850s and 1860s, cervical sympathetic chain resection was also tried, following the concept that Graves’-related palpitations were associated with increased sympathetic activity.

By 1940, thyroid surgery techniques had been perfected, including by Dr. William Halsted at Johns Hopkins. Halsted also identified techniques to keep parathyroid glands intact during surgery, noting the complications of postoperative muscular spasms that otherwise resulted due to a deficiency of calcium as a consequence of parathyroid removal. At Mayo, Plummer’s approach of using iodine (Lugol’s) solution preoperatively for Graves’ disease surgery resulted in mortality rates less than 1 percent by 1925. However, surgical approaches were replaced in the 1940s by nonsurgical approaches, including use of radioiodine treatment and thiouracil.

The potential use of radioiodine for hyperthyroidism was first described in 1938 by Dr. Saul Hertz, and MIT physicists Robley Evans and Arthur Roberts, from rabbit studies showing radioiodine was specifically taken up by induced hyperplastic (enlarged by tissue) and goitrous (swollen) thyroid glands. The same year, at the University of California Berkeley, Iodine-131 (I-131), with a longer half-life, was generated by chemist Glenn Seaborg at the behest of friend and colleague Dr. Joseph Hamilton. In 1939, Hamilton and Soley first used I-131 for diagnostic thyroid uptake tests. In 1941, hyperthyroidism treatment with radioiodine using Iodine-130 (I-130) was first described at MIT by Hertz and Roberts, and later that year by Hamilton and Lawrence using I-131. This is the isotope that we use today. Treatment of hyperthyroidism with I-131 became increasingly widespread after 1946, when the substance became available in quantity from the Manhattan Project facility at Oak Ridge, Tennessee.

The association between goiter and eye disease has been described as early as the 6th century AD, as well as by the famous Persian physician Avicenna (pictured above) in the 11th century.
Dr. Emil Theodor Kocher (1841 – 1917)
Swiss physician and recipient of the 1909 Nobel Prize, pioneered thyroid surgery and reduced the mortality of thyroidectomies to below 1% in his operations.

Dr. Rosalind Pitt-Rivers (1907 – 1990)
A British biochemist who along with Jack Gross helped discover the thyroid hormone T3.

Dr. Henry Stanley Plummer (1874 – 1936)
American endocrinologist who in 1913 helped distinguish between the major causes of toxic adenoma and Graves’ disease; also improved mortality rates for Graves’ disease surgery to less than 1% by 1925.

Ernest Henry Starling (1866 – 1927)
A British physiologist who in 1905 described the term “hormone” after studying pancreatic secretions with colleague William Bayliss.

Dr. Edward Calvin Kendall (1886 – 1972)
An American chemist and Nobel Prize recipient who in 1914 isolated thyroxine (T4), the main thyroid hormone.

Dr. Hakaru Hashimoto (1881 – 1934)
A Japanese surgeon who in 1912 was the first to describe the disease that would later be named Hashimoto’s thyroiditis.

Dr. William Stewart Halsted (1852 – 1922)
An American surgeon who helped perfect thyroid surgery techniques and keep parathyroid glands intact during surgery.

Dr. Saul Hertz (1905 – 1950)
An American physician who in 1938 discovered the use of radioactive iodine for the treatment of hyperthyroidism.
These nonsurgical approaches revolutionized treatment for Graves’ disease. In the early 1940s, compounds such as thiouracil were also developed as treatments for hyperthyroidism. Propylthiouracil (PTU) was developed around the same time and, having less side effects than thiouracil, was used widely within a decade. It was soon joined by methimazole and carbimazole, which is used in Europe but not approved in the U.S. PTU and methimazole are the mainstay oral agents of current Graves’ disease treatment.

Thyroid Cancer

As early as the first century AD, Chinese physician Tshui Chih-Thi distinguished non-curable (malignant) from curable (benign) neck masses. Many centuries later, thyroid cancer was first described in 1811. From early on, the differentiated nature of most cases of thyroid cancer was noted. Dr. Humphry Rolleston, in “Endocrine Organs in Health and Disease, (1936), notes thyroid cancer “...may closely resemble normal thyroid tissue, and the metastases may do the same.” Furthermore, “...carcinoma rarely supervenes in the thyroid of toxic goitre.” However, unlike in the modern day, Rolleston noted that the prognosis was “gloomy,” and that “...when the diagnosis is definite, the condition is often beyond the reach of surgery...” However, it is not necessarily true that the cancer cases he was describing were similar to the generally nonaggressive cancers we generally treat nowadays.

The use of radioiodine for treating thyroid cancers was first described in New York in 1941, and subsequently described through the first half of the 1940s, using Iodine-130. Iodine-131 was subsequently available from after 1946. Recent events such as the release of radioiodine from Chernobyl and Fukushima have increased public awareness of thyroid cancer.

More recent approaches to thyroid cancer diagnosis and treatment include the use of fine needle aspirations. Needle biopsy was first described in 1930, by Martin and Ellis, but the technique, by then using finer needle diameters, was not widely adapted in the U.S. until the 1980s. Recent developments in thyroid cancer treatment include the increasing use of ultrasound for diagnostic purposes, the use of rTSH for treatment and diagnostic purposes in the U.S. (after FDA approval in 1998), as well as the recent development of tyrosine kinase inhibitors for treatment of advanced-stage thyroid cancers. Current “third generation” TSH assays have very high sensitivity for detection of thyroid dysfunction, but also have raised the possibility of detecting subtle changes in thyroid function that are of debatable clinical relevance.

Advance preparation is a key defense for chronic disease management during emergencies.

Being caught unprepared during natural disasters and emergency situations can be potentially life-threatening to a person with diabetes. The My Diabetes Emergency Plan is a convenient checklist that contains all of the essential items those with diabetes need to have readily available in the event of an emergency.

On the website, you can download the plan in English or Spanish and view a step-by-step video of how to create your kit.
You can become an active participant in protecting your well-being by visiting www.thyroidawareness.com.

The site features in-depth content about thyroid disease risk factors, symptoms and treatment options, as well as downloadable flyers about the range of thyroid conditions.
Thank You

The American College of Endocrinology (ACE) and the American Association of Clinical Endocrinologists (AACE) would like to thank Lilly Diabetes for its support of the EmPower initiative.