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EPCMS Mission:
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Recently the FDA approved consumer direct 23 and me genome testing to determine disease risk. As I read the new research related to CRISP-R, gene replacement in single gene conditions; I could not help but wonder what the future of medicine will look like, who will be interpreting the genetic test results, and more importantly, how will we manage the influx of patients that will inundate our offices trying to not only understand their results, but also determine risk and intervention strategies for a specific disorder. In my opinion, the approval provided by the FDA, was ill-advised and will add an increase burden to our already exhausted profession.

There are many considerations to be wary of the new FDA recommendations.

1. The genome, as we know it, includes only 3% of the 3.2 billion bases on the DNA; the other 97%, the research community considers it “Junk DNA” which may be true or not. Also, we have not addressed the metabolic steps between the genetic DNA and the many intermediaries before the DNA’s expression.

2. We have neglected to understand the many factors of genetic translation, epigenetics or even the interface between our own individual microbiome and the genetic makeup. For example, how come “identical twins” are so frequently discordant in their outcomes?

3. What is the economic cost resulting in the increased number of people accessing this technology? What are the ethical ramifications, when someone recognizes that they have the genes that are statistically related to Alzheimer’s, for example?

4. This may result in increased number of individuals requesting genetic therapy; how will clinicians be trained to interpret and assist patients? As practicing physicians, how will we respond when we are presented with the awkward situation where the patient who has concentrated on one condition will know more than the physician who will be required to know a little about many?

According to Dr. Plotnikoff the determination of an individual’s genome could provide a better understanding of specific metabolic pathways for individuals which could be important in metabolizing medications, supplemental dietetic needs, determining which mediators may help individual’s general condition or neurotransmitter production, and facilitate therapies.

New research on CRISP-R, that has the capacity to replace defective genes with healthy SNP, could cure single gene defects, but it will be a lot harder in complex multigene syndromes.

It is my estimation that this recommendation will result in people with inappropriate background becoming partially trained and providing “advice” without the complex understanding of how the genome interfaces with so many factors or an understanding of the clinical conditions, as frequently the findings do not correlate with the outcomes as has been demonstrated by extensive research on the genetics of bipolar syndromes or autism.

I hope we monitor closely the results of this FDA approval. As the recommendations and implementation strategies have not only ethical and economic concerns, but also possibly false expectations from many patients. In addition, will require to enhance the training in genetics and epigenetics for all of us as we will be confronted with a large number of patients looking for answers once they have their genome.
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I begin my work day driving with the radio on a morning talk show. I don’t always agree with the topics, but the DJs will inevitably have me laughing by the time I arrive at my office. That laughter makes sure that my day starts pleasantly. It creates a feeling that nothing can go wrong that day. I try to carry that feeling through to my staff and patients. I tell funny stories to get the staff in a happy mood and I try to get each patient with whom I deal smiling and laughing. One of the benefits of working in pediatrics is that almost all my patients prefer to laugh rather than cry or be angry. Babies who are cranky get silly faces from me, older kids who are scared of shots get jokes or encouragement to tell me a funny story. If a child won’t or can’t take a deep breath when I am examining his/her lungs, I tickle them to get them laughing—both as an ice breaker, but also when they take a deep breath after laughing, I can hear the lungs easily.

Having a sense of humor in one’s life is helpful; having a sense of humor in medicine is imperative. How else can we recover from treating patients who are chronically or terminally ill? How else can we work with families or are stressed about their next meal or whether they can care for their children effectively? How can we protect both them and ourselves from the health effects of depression? It is not always possible or appropriate to laugh at everything we encounter, but having a good, deep laugh that reaches both your belly and your eyes is a sure way to shake off the stress of a difficult day.

I first learned about Patch Adams and his philosophy of humor and play, which he has promoted as essential to physical and emotional health, in medical school. As an exercise for the medical students, we were required to come up with a comedic routine to use at our next outpatient clinic day. I will admit, I am not the greatest comedian and I am horrible with remembering jokes. But I do know how to laugh at myself and I do know how to act silly to build rapport with patients. It was one of the most psychologically difficult, yet enjoyable, exercise in medical school. I encourage all physicians to attempt this at least once.

As we see our next generation of student and residents graduate this summer, please make sure to teach them about this concept as well. These days, we need laughter and humor more than ever.

“Always laugh when you can. It is cheap medicine”.
— Lord Byron

“I have always felt that laughter in the face of reality is probably the finest sound there is and will last until the day when the game is called on account of darkness. In this world, a good time to laugh is any time you can.”
— Linda Ellerbee, American Journalist and news correspondent
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Introduction
Pneumatosis cystoides intestinalis (PCI) is a rare complication of mixed connective tissue disease and scleroderma. PCI is defined as the presence of intramural accumulation of gas filled thin-walled multi-locular cysts in the submucosal or sub-serosal layers of the gastrointestinal tract. Typically, PCI develops during scleroderma flare-ups associated with mechanical insult or bacterial overgrowth. Imaging studies are used for diagnosis with CT or plain film radiography showing gaseous cysts in the bowel walls. Conservative treatment is usually all that is required for PCI, however with signs and symptoms of bowel ischemia or perforation, immediate surgical intervention is needed. In addition, the appearance of apparently unexplained free air under the diaphragm suggests surgery is needed for the working diagnosis of perforated viscus, peptic ulcer disease, or diverticulum. In this case series we describe two patients, both presenting with PCI as a complication of scleroderma.

Case Presentation
Case 1
A 41-year-old woman was transferred to our hospital from another facility due to unresolved abdominal pain, nausea, emesis worsened by oral intake for the past month. Her history was significant for a long standing mixed connective tissue disease, systemic scleroderma, with severe gastrointestinal involvement diagnosed 7 years ago, and reliance on total parenteral nutrition (TPN). In addition, she had been diagnosed with systemic lupus erythematosus (SLE), Raynaud’s phenomenon, autoimmune hepatitis, Sjogren’s syndrome, and polymyositis. She had chronic abdominal pain with intermittent nausea and vomiting since 2013. Those symptoms were controlled with domperidone, a prokinetic, and antiemetic until about a month ago. In order to relieve her abdominal pain, she required fentanyl patch 100 mg every 3 days and hydromorphone 2 mg three times a day as needed. Prior to her current admission, she had stopped outpatient TPN for approximately 1 month. She resumed oral intake on her own, noting that her overall intake was poor since it would trigger nausea, vomiting and abdominal pain that gradually worsened. Her transfer records included abdominal computed tomography (CT) images which revealed extensive pneumatosis intestinalis in the distal ileum, and colon and pneumoperitoneum with free air under the diaphragm (Figure 1). Ruptured viscus was suspected. She underwent immediate diagnostic laparotomy revealing intact bowel with no evidence of perforation. No resection or intervention took place at that time. It was her second diagnostic laparotomy, with the first one being performed 6 months prior in the same clinical setting of free air under the diaphragm resulting in similar negative findings. On admission to our hospital she complained of diffuse abdominal pain, 8 out of 10 in intensity, associated with nausea and occasional vomiting of solid food contents 3 hours post meal intake. During physical examination, the patient was afebrile, blood pressure 148/98 mmHg, and heart rate 87 beats per minute. She appeared cachectic and weighed 44.3 kg. On physical examination she had facial and digital skin tightening typical of systemic scleroderma, epigastric tenderness, her abdomen was soft and nondistended without rebound or guarding. The laboratory investigations revealed normal white blood cell count, hemoglobin 8.3, hematocrit 25.6, mean corpuscular volume 91.4, platelets 212, sodium 141, potassium 3.3, chloride 110, bicarbonate 21, blood urea nitrogen 6, creatinine 0.68, albumin 1.4, total protein 8.7, glucose 66, calcium 7.3, total bilirubin 0.3, AST 30, ALP 56. Patient was placed on in-
travenous fluids (IV) and bowel rest (NPO). An abdominal ultrasound with Doppler flow showed no evidence of celiac artery compression. A glucose breath test for small intestinal bacterial overgrowth (SIBO) was also positive. A gastric emptying scintigraphy study was limited secondary to less than 75% consumption of the radiotracer tagged meal, with retention of the tracer within dilated distal esophagus. Upper gastrointestinal endoscopy revealed atonic esophagus, chronic gastritis, dilated first and second portions of duodenum. Pathological specimen was positive for *H. pylori*. The patient was restarted on TPN while being treated with antibiotics for SIBO and *H. pylori*, in addition to metoclopramide IV for gastrointestinal dysmotility. In order to overcome dependence on TPN with all its complication and costs, the patient underwent laparoscopic J-tube placement with antral smooth muscle biopsy revealing a few inclusion bodies, mild to moderate fibrosis in between the muscle bundles, and 15 interstitial cells of Cajal per high power field, which is within normal limits. Patient tolerated J-tube feeding very well during her remaining hospital stay. Nutrition formula (1.5 cal/cc Jevity®, Abbott) feedings were administered over 8-10 hours at night at a rate of 60-80 cc per hour. The patient would try oral feeding during the day when there was no J-tube feeding. TPN was discontinued prior to discharge. She was discharged home on antibiotics, azathioprine (for her scleroderma) and medication to manage pain. As an outpatient, she continued with nocturnal J-tube feeding and soft mechanical diet during the day as tolerated. During her follow-up appointments with a gastrointestinal motility expert one month later, the patient’s condition remained stable. Her appetite improved, but she had not gained weight since discharge. Since her tolerance for oral food intake improved, the goal for the following visit was to gradually increase caloric intake.

Case 2
A 59-year-old female with history of scleroderma and gastroparesis, presented to the emergency room with nausea, vomiting, abdominal pain, decreased food intake, constipation for the past four days and sharp left-sided chest pain of one day duration. She had three previous admissions with similar symptoms at a different facility. Her past medical history was significant for breast cancer, status post bilateral mastectomy in 2005 and 2006 followed by chemoradiation treatment. She was diagnosed with scleroderma in 2012. She was seen by a gastrointestinal motility expert at our institution a few months prior to the current admission. At that time, she reported significant weight loss during the past 6 months. Treatment with the prokinetic/antiemetic agent, domperidone was initiated by way of FDA IND protocol. On admission to our medical center, the patient had a temperature of 36.6 degrees Celsius, heart rate 103 beats per minute, blood pressure 91/56 mm Hg (mean arterial pressure 68 mm Hg). On physical exam she was cachectic, with facial skin tightening characteristic of systemic scleroderma. Her abdomen was distended and tender with hypoactive bowel sounds. Laboratory work-up revealed hemoglobin 6.2, hematocrit 18, mean corpuscular volume 88.2, platelets 287, sodium of 124, potassium 3.7, chloride 86, bicarbonate 21, blood urea nitrogen 29, creatinine 0.92, glucose 153, calcium 7.3. Computed tomography (CT) of the chest showed cavitated necrotizing pneumonia in the left lung base, likely due to aspiration, with no evidence of significant pulmonary fibrosis (Figure 2). Her abdominal CT revealed a diffuse dilated small bowel with pneumatisis intestinalis and...
pseudocystoides intestinalis (PCI) was described during autopsy by DuVernoi in the 18th century. PCI is an extremely rare occurrence in scleroderma and mixed connective tissue disease. PCI is defined as the presence of intramural accumulation of gas filled thin-walled multi-locular cysts in the submucosal or subserosal layers of the gastrointestinal tract, sometimes resulting in pneumoperitoneum. The signs and symptoms of PCI can range from nonspecific abdominal discomfort, pain, nausea, vomiting, weight loss, diarrhea, to radiological signs consistent with abdominal perforation. These manifestations were well represented in both patients in this case series.

Scleroderma results in a multitude of pathologies including microangiopathic changes, atrophy of the intestinal layers, fibrosis, intestinal dysmotility, bacterial overgrowth, increased intraluminal pressure, and degradation of the intestinal wall. The small intestine is the part of the gastrointestinal tract most commonly involved. Cysts measuring 0.5-10 cm are most commonly found in the terminal ileum, less commonly the large intestine and rarely the stomach. Multiple theories exist for the pathogenesis of PCI, with the two predominant mechanisms being a mechanical disruption versus bacterial overgrowth. The mechanical theory states that PCI development in scleroderma results from a pseudo-obstruction or compression state resulting in gas passing through the submucosal or subserosal layers of the intestines and small cysts forming subserosally. The bacterial overgrowth theory of PCI development in scleroderma results from intestinal hypomotility which creates bacterial overgrowth in the intestines and subsequent bowel distention and intraluminal pressure caused by increased bacterial fermentation production of hydrogen and/or methane being forced into submucosal or subserosal tissues, and breakdown of mucosal integrity.

Less common theories for development of PCI include chronic long term use of corticosteroids used in the course of treating scleroderma, which results in atrophy of the intestinal mucosa and relocation of bowel gas to the submucosal/subserosal area. However, it should be noted that neither of our patients had received corticosteroid treatment for any extended periods. A previous, less supported theory of PCI development is pulmonary gas from ruptured alveoli resulting in air traveling down the mediastinum and tracking into the retroperitoneum eventually leading into the bowel mesentery, or in the setting of emphysema, intra-abdominal pressure, or pulmonary obstruction. Pre-existing pulmonary disease (i.e., pulmonary fibrosis, emphysema) was not evident in our patients.

Imaging studies are important for the diagnosis of PCI. The advent of CT has decreased the incidence of late stage disease complications such as intestinal perforation or pneumoperitoneum, such that most PCI in scleroderma is detected earlier while relatively asymptomatic. CT is sensitive for detecting PCI, but cannot distinguish between benign and life threatening causes. Although X-ray had been used in the past to detect air cysts in the intestinal wall, CT has become the gold standard. Imaging should show “radiolucent linear or circular air bubbles in the bowel wall with or without subdiaphragmatic free air.” In this setting, a laparoscopy for “perforation of the bowel” is not indicated. This finding could be explained by diffusion of gas from the cysts rather than “true perforation.” As illustrated in our first case, two laparoscopies were performed for presumed perforation.

However, bowel wall gas seen submucosally or in muscle is also observed in other conditions. Differential causes include gas forming organisms (e.g., Clostridium), necrosis, ischemia or perforation can complicate toxic megacolon in inflammatory bowel disease, ischemic bowel. Conservative treatments include bowel rest, oxygen therapy, supportive care (fluids and electrolytes), total parenteral nutrition, bowel decompression, octreotide infusion and antibiotics. Increased oxygen can reduce submucosal/subserosal gas cysts due to the decreased partial pressure of nitrogen which decreases fermentation of intestinal bacteria.

Complications of PCI are rare, but can include pneumoperitoneum, volvulus, true complete obstruction, or bowel ischemia leading to hemorrhage. Ischemia leading to gangrene should be suspected when elevation of lactate is detected, in which case immediate surgical intervention is necessary. Although usually benign, patients need to be watched carefully for bowel ischemia or perforation. PCI is usually a late stage disease presentation in scleroderma.

PCI as a radiographic finding may be present in patients with a wide spectrum of disease processes in addition to systemic sclerosis such as pulmonary causes (emphysema, pulmonary fibrosis, cystic fibrosis), intestinal causes (pseudo-obstruction, peptic ulcer disease, IBD, diverticulitis), and mesenteric vascular disease, colitis, and trauma. Although PCI can resolve with watchful waiting as in our first patient, clinicians should be aware of other complications of scleroderma where PCI can be a secondary finding, as illustrated in our second case, where we presume that gastroparesis due to scleroderma led to aspiration, pulmonary abscess, respiratory and circulatory failure, and death.

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Abstract
Self-monitoring of blood glucose can contribute usefully to management of diabetes mellitus. There are several products for self-monitoring of blood glucose to choose from. Although there is no overall single best blood glucose monitoring system, different systems offer different combinations of features that might be better for some patients. Seven features were identified as having the greatest impact on SMBG meter selection: (1) cost, (2) meter-related factors, (3) test strip-related factors, (4) environment, (5) physiological, (6) medication-related, and (7) human factors. Taking a patient's financial resources, dexterity, and comfort with technology into consideration to individualize selection of a blood glucose monitoring system can improve compliance and accuracy of home blood glucose testing.

Introduction
Self-monitoring of blood glucose (SMBG) is a necessity for all insulin-treated patients, and can be useful in monitoring and adjusting treatment of non-insulin-treated patients with diabetes mellitus. About 29.1 million people in the United States have diabetes mellitus. The high prevalence diabetes mellitus within the United States has propelled an expanding diabetes management equipment industry. Currently, there are numerous blood glucose monitoring systems marketed in the United States.

The aim of this paper was to evaluate factors of relevance in selecting glucose monitoring system for patients, highlight the importance of patient education in proper blood glucose test site preparation and technique for blood sampling, and review sources of error and interference with blood glucose readings that might be clinically relevant in particular circumstances.

Literature Search Strategy
A literature search was conducted using PubMed and MedLine databases using Boolean combinations of search terms: self-monitoring blood glucose, blood glucose meters, blood glucose test strips, diabetes mellitus, glucose monitoring, interference, limitations, review, oxygen content, skin preparation, glucose dehydrogenase, glucose oxidase, best practices. Studies from 1987 to the present were retrieved, reviewed and selected based on their quality and relevance to the topic under investigation.

Regulatory Background
SMBG has been used for over twenty years to monitor glycemic control, adjust medication and especially insulin dosing in diabetic patients. Early blood glucose monitoring systems relied heavily on patients being able to apply the blood sample directly to the test strip, accurately time the reaction, wipe the blood from the test strip, and compare the color of the test strip to a reference chart printed on the strip container. Several steps of that procedure were sources of error. Newer monitoring systems have minimized sources of error by removing the need to manually wipe blood off the test strips, and automating the timing and reading steps. In 1987, the American Diabetes Association convened a consensus panel to improve quality control practices for self-monitoring of blood glucose practices. The panel identified essential components of quality control which included: (1) meter calibration to ensure adequate performance; (2) glucose control solutions for reagent strip performance evaluation; (3) comparison of glucose monitoring system performance with a reference laboratory; and (4) periodic retraining and correction of user technique by a healthcare provider or qualified diabetic educator.

In January 7, 2014 the Food and Drug Administration (FDA) released more stringent requirements for blood glucose monitor performance to approximate laboratory reference ranges such that 95% of measurements must be within ±15% and 99% of measurements within ±20%. Trained technicians obtain better reliability and accuracy of glucose monitoring equipment. FDA guidelines also suggest documenting performance of glucose monitoring systems in the hands of untrained people to better simulate “real-world” conditions. Additional glucose monitor design features might be attractive and useful to some patients.

Physician Considerations in Glucose Monitor Selection
Physicians could help individualize the choice of glucose monitoring system for their patients. The choice of monitor will not supersede the importance of proper skin preparation and blood sampling technique, but choices do have potential to provide more accurate and reliable results in patients who cannot avoid sources of interference such as drugs, icosadextrin-containing peritoneal dialysate solutions, or supplemental oxygen. Ease of use and added design features might better engage some patients in management of their disease, and allow a physician to track patients’ glycemic control between follow-up visits to assess and adjust management accordingly. Some of the considerations in blood glucose monitor selection will represent trade-offs in cost, meter-related features, test strip-related features, and comfort.

Continued on page 12
and requirements for SMBG to perform in circumstances outside the usual spectrum of environmental conditions, physiological states, medication-related interferences, and human operator limitations of dexterity, vision, and intellectual capacity.

Cost Considerations
For some patients, cost will be the principal limiting factor in selection of a SMBG meter.3 Unless patients are willing to incur out-of-pocket costs for an alternative device and corresponding test strips, physician and patient discretion in SMGB selection might be constrained to the extent that insurance companies will only cover specific meters under their plans.3 The cost of the meter is seldom the issue. Some manufacturers give away their monitors, because the profit for the manufacturer resides with the strips. The retail cost of different brands ranges between $0.40 to $2.10 per test strip, but cost $0.08 to $0.12 to manufacture.7 Each type of monitor requires the manufacturer’s proprietary brand of glucose test strip. Differences in the price of the test strips compounded over one year can cost a patient testing their blood glucose four times per day ranging $584 to $3,066 annually. Physicians can help patients understand how some features might justify additional cost of the strips for a particular monitor in such circumstances as needing large digital readout to compensate for impaired vision, or ease of operation to compensate for arthritis or diabetic neuropathy affecting hands.

Meter-Related Features
The basic technology of SMBG meters is that of an amperemeter. They measure the change in current as a function of electrochemical conversion of sample glucose to gluconolactone between the carbon, palladium or gold electrodes of disposable strips.

Every SMBG meter has inherent imprecision and bias, even when used by trained personnel, but generally, those differences between meters are not clinically meaningful.8 Features more likely to be worthy of practical consideration include the size, lighting of strip port and the display screen; the ease of handling of the unit itself; the ability to transfer and store organized information from the meter to external memory such as a computer, SD card or smartphone; as well as the adaptability of the meter for alternate site testing (AST). AST, by reducing the volume of blood required for each test allows for shallow lancing and results in less pain and improved convenience. AST can be an attractive option for patients with aversion to the recurrent finger pricks required for monitors that require a larger volume of blood than AST devices.8

Some recent SMBG meters have built-in safeguards such as test strip expiration detection, alerts for inadequate blood sample size, exposures to out-of-range temperatures, and no requirements for coding or calibration. Those innovations can mitigate potentially substantial errors glucose test results.8

Test Strip-Related Factors
All SMBG strips are comprised of a permeable membrane that separates blood sample from an oxidoreductase reagent layer that converts glucose to gluconolactone.13 There are two distinct oxidoreductase reagent systems: glucose oxidases or glucose dehydrogenases. Glucose oxidase reactions are sensitive to differences in oxygen tension. With the exception of mannose, glucose oxidase is highly specific for glucose. There are multiple glucose dehydrogenases with different specificity to distinguish glucose from other sugars, and proprietary differences in catalytic co-factors used to mediate and accelerate the conversion of glucose to gluconolactone, each with slightly different vulnerability to error from endogenous and exogenous interference. Table 1 provides comparison of current blood glucose monitoring reagent systems.13

Within regulatory quality control requirements, lot-to-lot, vial-to-vial, and strip-to-strip variability in blood glucose test strip affect capillary blood glucose readings.3 Manufacturing of strips within very narrow tolerance is key to achieving accuracy reliably and rapidly from very small blood samples < 1 microliter. Minute variations in well size or capillary diffusion separation between sample layer and chemistry layer will influence accuracy.3

Environmental Conditions
Altitude, temperature, humidity and electromagnetic radiation from mobile phones have been shown to affect SMBG test results.8

Altitude
Although the percentage of individual inspired gases (i.e., nitrogen, oxygen, carbon monoxide) is the same at all altitudes, ambient and blood oxygen concentration decline proportionally with declining atmospheric pressure at increasing altitude. To the extent that changes in elevation and changes in the fraction of inspired oxygen (i.e., patients on supplemental oxygen) alter the partial pressure of capillary blood oxygen, altitude and supplemental oxygen have potential to interfere with performance of SMBG systems with glucose oxidase reagent systems. Glucose and oxygen are the natural substrates for glucose oxidase, and hydrogen peroxide is the reduced product. At altitudes >2000 meter above sea level (El Paso International Airport is 1200 m; Ruidoso, NM is 2110 m; Cloudcroft, NM is 2640 m), bias attributed to lowered

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<th>interference</th>
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<tr>
<td>glucose oxidase</td>
<td>FAD</td>
<td>changes in partial pressure of oxygen, mannose, uric acid, bilirubin, acetaminophen, aspirin, ascorbic acid, heparin, warfarin, metabolites of icodextrin</td>
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<tr>
<td>glucose oxidase</td>
<td>peroxidase; FAD; hexacyanoferrate</td>
<td>changes in partial pressure of oxygen, mannose</td>
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<td>glucose dehydrogenase</td>
<td>PQQ; phenyldiaminequinone</td>
<td>maltose, icodextrin and metabolites xylose (e.g., hand residue from sugar-free gum)</td>
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<td>NAD; phenyldiaminequinone</td>
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<td>glucose dehydrogenase</td>
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FAD = flavin adenine dinucleotide
NAD = nicotinamide adenine dinucleotide
PQQ = pyrroloquinolinequinone

Continued on page 13
PaO₂. Patients planning trips to high altitudes should not rely on glucose oxidase-based SMBG reagent systems.

**Temperature**

The blood glucose monitor reading does not require complete conversion of the glucose in the sample to gluconolactone by the enzyme reagent in the test strip. Although that may seem counter-intuitive, but the standard 5-second reading is extrapolated from the concentration of gluconolactone generated in that time, achieved by controlling the rate of diffusion of glucose from the sample layer to the reagent layer containing excess enzyme. Diffusion and enzyme velocity are temperature dependent. Newer SMBG meters have temperature sensors that correct for ambient temperature within a finite range (typically 10-40°C, 50-104°F), but out-of-range temperatures will result in uncorrected bias. Falsely high results will be obtained at temperatures above that range, and falsely low results when a monitor is used at temperatures below the recommended range.

Bear in mind that the monitor senses temperature, but it is the temperature of the test strip that affects the reading, so taking a monitor out of a hot vehicle to perform a SMBG measurement requires wait time for the monitor to cool down to the ambient temperature. The same concept applies to carrying a meter indoors in winter.

**Humidity**

Most systems perform reliably over a wide range of 10% to 90% humidity, but the strips must always be stored in their sealed container with desiccant in a cool (8-30°C, 46-85°F) dry environment. Patients should be advised that taking a few strips out of the container to carry along in purse or pocket for use during an outing will be compromised by ambient humidity, even in dry days of summer. In the case of multi-strip cartridges or wheels, each strip compartment has a 0.5 mm bead of desiccant, so a strip should be used within a few minutes once the foil covering is breached. A bathroom is not ideal.

**Electromagnetic radiation**

Studies have demonstrated electromagnetic interference from mobile phones on blood glucose results. Mobile devices are ubiquitous. Conscientious patients and practitioners might intentionally run SMBG samples away from mobile devices. The Agamatrix Inc. One Drop™ SMBG system is designed to pair with iOS or Android™ device. Asked, “To what extent does electromagnetic radiation affect OneDrop™ device performance?”, the company replied, “If not used correctly, the One Drop Meter may interfere with your TV, radio, or other electronic devices that receive or transmit RF signals. With the exception of your iOS or Android™ device, other electronic wireless devices that are in use nearby, such as another cell phone or a wireless network, may prevent or delay the transmission of data from your One Drop Meter.”

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- Surgical wound care
- Vascular elevation

Pain Management
- Pain clinic
- Pain procedures
- Pain injections
- Trial and permanent stimulator placement

Ancillary Services
- MRI
- CT Scan
- Ultrasound
- DEXA Scan
- Full service laboratory

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Luis Ayo, MD, FAAP

I met my friend, Dr. Luis Ayo, July 1st 1975, as a brand new assistant Professor of Pediatrics-Infectious Disease in El Paso at what was the Regional Campus of Texas Tech University School of Medicine; Luis was just beginning his first year of residency in Pediatrics at Thomason. Those few months were tough, as we had one faculty and four first year and four second year residents. But, we persevered, albeit busy, with 45 beds, over 3000 deliveries, and a busy pediatric clinic.

Luis from day one he demonstrated his adoration for smaller babies and the difficult to manage newborns. Luis’s devotion was an inspiration to watch; in those years Thomason did not have a NICU—instead we changed oxygen tanks as their alarms sounded when their PSI dropped. For many, this would be a stressful time because in an instant the ventilators could malfunction and the infant could become hypoxic. But, not for Luis, he thrived in this environment—it was as if he was programmed by some divine intervention to care for these children. It was no surprise to me that he would become a neonatologist.

Following his residency, Luis left for Florida to train as a neonatologist. However, he later returned to El Paso. We could not have been more blessed that he decided to come back. He had an innate ability to cultivate and build teams through his compassionate nature and love for the profession. This was particularly felt at Providence Memorial Hospital, where he served as part of the Board—and also at Las Palmas. At Texas Tech, over the course of only a couple of years Luis had effectively won the hearts and minds of all the residents and nurses and was rated as the best teacher his second year with the University. Although his obligations did not allow for him to continue with Texas Tech he had left a lasting impression on the faculty and residents at large. Even years later, residents who knew him often commented about his expertise and his commitment to save every baby that was admitted to the Unit. Luis’s legacy as a phenomenal neonatologist is without question, the last infant we saw together required an extensive stay in the Unit—I am glad I did not bet him on the outcome, as I would have lost. In spite of the plethora of challenges, because of Luis’s expertise, commitment, and care the baby was eventually discharged home.

Luis’s accomplishments are too great to detail simply put, he was remarkable.

Although it is difficult to not mention neonatologist when we speak of Luis because it was such a large part of his being—he was more than that. He was one of the most compassionate people I had the privilege to know. He was a true gentleman, and never spoke ill of anyone. Luis was a visionary, frequently anticipating changes to systems and political waves ahead of the storm.

Even more, Luis was a true and trusted friend. And I and many of you here are blessed to have not only been his friend but also to have witnessed what true dedication and commitment as a physician is. Even after his myocarditis he would not stop caring for his children, his love for the profession, nor did he ever lose his sense of humor.

Simply saying that we are missing Luis is an understatement. For all of us he was and will always be more than a colleague, teacher, board member, and section head. He was an indefatigable physician and a true friend.

Gilbert A. Handal, MD
Physiological Factors
Various physiological states such as capillary perfusion, hematocrit, endogenous biomolecules and metabolites (e.g., triglycerides, bilirubin and uric acid), and blood oxygen content can all bias blood glucose test results.

Decreased peripheral perfusion, as in Raynaud, hypovolemic and cardiogenic shock, hyperosmolar-hyperglycemia, can yield lower-than-expected blood glucose test values due to increased tissue glucose extraction. Venous or arterial sampling will provide accurate blood glucose measurement in such cases. Correlation with the patient’s clinical condition must always be considered in instances of questionable or unexpected laboratory values.

Hematocrit and blood glucose are inversely related. Hematocrit <35% bias toward higher blood glucose readings, and high hemconcentration or polycythemia bias toward SMBG under-estimation of blood glucose relative to measurements by reference laboratory methods. Some, but not all newer SMBG systems include ‘multi-well’ technology that separately measures and corrects for hematocritin the glucose calculation.

Triglycerides in hypertriglyceridemia and chylomicronemia/lipemia comprise a higher proportion of blood volume and thus decrease plasma glucose per unit volume, leading to falsely low estimation of blood glucose. High bilirubin can result in falsely low blood glucose readings. Uric acid concentrations above approximately 20mg/dL can result in falsely high blood glucose readings to the extent that uric acid in sampled blood is oxidized by hydrogen peroxide in glucose oxidase-based reagent systems that do not include peroxidase as a second enzyme. Since uric acid is a DNA degradation product, this has the most clinical importance among patients with gout and cancer patients undergoing chemotherapy or radiation treatments resulting in high cell turnover.

The relationship between high blood partial pressure of oxygen status (PaO2 ≥ 150mmHg) and underestimation of blood glucose concentration with glucose oxidase test strips is understood. Oxygen and glucose are both glucose oxidase substrates, and compete for electrons, so excess oxygen drives the reaction away from gluconolactone in favor of hydrogen peroxide production, resulting in falsely low blood glucose estimation. The opposite is true in low oxygen states such as may be seen in patients with chronic obstructive pulmonary disease, where low oxygen states (PaO2 < 45mmHg) can lead to falsely elevated blood glucose estimation. Glucose dehydrogenase-based test strip reagents are not biased by variations in blood oxygen concentration.

Continued on page 18
Medication-Related Interferences
Several drugs and medical therapies can interfere with blood glucose readings. Acetaminophen, dopamine, ascorbic acid, ibuprofen, epidraine, salicylate, and tetracycline are a few drugs known to cause interference within therapeutic range of concentrations.10,11 Drug formulations containing maltose, galactose, and xylose have the potential to enter the bloodstream during certain diagnostic tests and therapies, causing abnormally high blood glucose readings, particularly readings obtained with glucose dehydrogenase–PQQ reagent systems, somewhat less with glucose dehydrogenase—FAD reagent systems, and not with glucose oxidase—peroxidase reagent systems.12 Recall that glucose and oxygen are the natural substrates for glucose oxidase, and hydrogen peroxide is the reduced product capable of non-specific oxidation of drugs and metabolites (e.g., bilirubin, uric acid), and interfere with electron transfer in glucose conversion to gluconolactone. Glucose oxidase reagent systems that include peroxidase as a second enzyme react with hydrogen peroxide to produce water and oxygen and thus mitigate that mechanism of interference.

Icodextrin is a glucose polymer and a component of some peritoneal dialysis solutions. Up to 30% of this compound can be absorbed into systemic circulation during the dwell time. Although icodextrin directly cross-reacts with glucose dehydrogenase-based SMBG reagent systems, maltose is a metabolite of icodextrin. Both glucose dehydrogenase-based reagent systems, and to a lesser degree glucose oxidase-based reagent systems have been shown to cross-react with metabolites of icodextrin, causing falsely high blood glucose readings.8 Over-estimation of glycemia has potential to motivate excess dosing of insulin and undertreatment of hypoglycemia. It is imperative that patients are properly educated on how to use their meters, understand how to store test strips, and know not to use expired test strips and control solutions. Patient understanding of the importance and procedure of coding BG meters impacts reliable readings; if incorrectly done, coding could lead to measurement errors of ±30%.8 Patient dexterity and level of education and training factor into the reliability of blood glucose results.

Dexterity often plays a role in blood glucose testing. Patients often have physical limitations (such as peripheral neuropathy or arthritis) in being able to manually manipulate and insert small test strips into their meter properly. SMBG systems are available with preloaded test strips in ‘drums’ or daisy wheels (for example, Breeze2® – BayerTM) may circumvent the need for patients to handle individual test strips.9

Although there are several operator-dependent factors that can compromise consistent accuracy of blood glucose results, proper collection of the blood sample is of utmost importance. The capillary blood sample itself needs to be uncompromised. Currently there is no consistent and well-researched information regarding proper preparation of the sample site prior to blood glucose collection. It is likely that a considerable portion of patients fail to wash their hands prior to blood glucose sample collection.6 The amount of blood needed for most current SMBG meters ranges from 0.3μL to 0.1μL; therefore, even the slightest contamination of the testing site by sugar-containing foods or fluids (i.e., fruits or fruit juices, respectively) could drastically bias the sample blood glucose reading.8 Improper lancing depth might compel the user to aggressively milk the finger in order to expel a sufficient volume of blood. Doing so will dilute the capillary blood with interstitial fluid of lower glucose concentration so monitor readings tend to erroneously lower estimation of blood glucose concentration.

Human Factors
Over 90% of blood glucose test results inaccuracies are attributable to inappropriate operator technique.8 It is imperative that patients are properly educated on how to use their meters, understand how to store test strips, and know not to use expired test strips and control solutions. Patient understanding of the importance and procedure of coding BG meters impacts reliable readings; if incorrectly done, coding could lead to measurement errors of ±30%.8 Patient dexterity and level of education and training factor into the reliability of blood glucose results.

Table 2: Summary of Factors Influencing Blood Glucose Monitor Selection and Accuracy

<table>
<thead>
<tr>
<th>Cost</th>
<th>Type of reagent</th>
<th>Amount of blood required</th>
<th>Manufacturing process (lot-to-lot, strip-to-strip, and vial-to-vial variation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meter-Related</td>
<td>Ease of use and handling</td>
<td>Readability of display</td>
<td>Data transfer from internal memory to computer or external device</td>
</tr>
<tr>
<td></td>
<td>Built-in safeguards (i.e., test strip expiration, under-dosing of blood sample, exposure to out-of-range temperature notifications)</td>
<td>Alternate-site testing</td>
<td>Coding requirement</td>
</tr>
<tr>
<td></td>
<td>Complete, comprehensible, and clear labeling and operating instructions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test Strip-Related</td>
<td>Environmental</td>
<td>Physiological</td>
<td>Medication-Related</td>
</tr>
<tr>
<td></td>
<td>Peripher al blood perfusion</td>
<td>Hematocrit</td>
<td>Icodextrin and other sugars (maltose, galactose and xylose) used in therapies and diagnostic tests</td>
</tr>
<tr>
<td></td>
<td>Temperature</td>
<td>Oxygen content</td>
<td>Drugs (i.e., Acetaminophen, ascorbic acid, dopamine, mannitol)</td>
</tr>
<tr>
<td></td>
<td>Electromagnetic radiation</td>
<td>Metabolites (i.e., triglycerides, bilirubin, uric acid)</td>
<td></td>
</tr>
<tr>
<td>Human</td>
<td>Patient dexterity (i.e., arthritis, peripheral neuropathy)</td>
<td>Patient training and education on proper use</td>
<td>Use of expired test strips, glucose control solution</td>
</tr>
<tr>
<td></td>
<td>Inappropriate storage of equipment</td>
<td>Incorrect interpretation of test results, and out-of-range values</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Incomplete test strip insertion into meter</td>
<td>Correct preparation of and obscuring of test-site to be sampled from</td>
<td></td>
</tr>
<tr>
<td></td>
<td>If coding is required, patient understanding of how to accurately code</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Discussion/Conclusion
SMBG monitoring continues to play an important role in diabetes management, and places the patient in an active role, with potential to motivate actions that lower HbA1c and decrease hospitalization complications related to diabetes.3 SMBG provides

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feedback on the impact that lifestyle behaviors such as exercise and nutrition can have on glycemic control, which bears importantly on preventing, delaying or mitigating potentially devastating retinopathy, neuropathy, nephropathy, micro- and macro-vasculopathies, infectious, orthopedic and other complications associated with poor glycemic control in diabetes.\(^3\) The actual utility of SMBG depends on whether patients or their care providers do something useful with the readings by adjusting diet, activity or medication.\(^1\) SMBG results can guide the health team’s ascertainment of abnormal blood glucose values between office visits.\(^3\) Diabetes is prevalent, and a large profitable competitive market for SMBG has driven remarkable advancements in SMBG technology.

This literature survey has identified and reviewed several factors for consideration by physicians assisting patients selecting a blood glucose self-monitoring system [summarized in Table 2]. Differences in the cost of the monitor itself is rather inconsequential, but each monitor requires use of its proprietary blood glucose test strips. Differences in the prices of test strips amortized over months and years can be very substantial. Some patients’ circumstances such as moving to very different altitude, supplemental oxygen, use of certain peritoneal dialysate solutions or use of certain medications favor one SMBG reagent system over another.

The majority of diabetic patients will manage just as well with any system of their choosing. Several studies have contrasted substantial errors made by users as opposed to well-trained personnel. A SMBG system cannot compensate for carelessness in skin preparation and operation. Ensure sufficient education and training to consistently perform SMBG measurements properly with whichever system they have at their disposal. Advancements in ease of use and features available with some SMBG systems help compensate for the impaired vision, dexterity and cognition that are frequent manifestations of advanced diabetes. Five manufacturers dominate the market with almost 90 SMBG choices. Table 3 summarizes a small selection of those with potentially helpful features. According to Walgreens, CVS and Walmart, OneTouch Ultra2—LifeScan, OneTouch Verio IQ—LifeScan, TrueMetrix Air—Trividia, and ReliOnUltima—Abbott are among the most often dispensed SMBG systems in El Paso.

### Description of Contribution to the Project
My contributions included searching and reviewing current literature pertaining to glucose monitoring, researching current and emerging capillary blood glucose monitoring systems, and formulating the outline of this written work. I also wrote the final version and all drafts of the paper in collaboration with Dr. Quest.
I independently created and presented a poster based on this paper at the 2017 Scholarly Activity & Research Program Spring Symposium on April 5th.

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Courtney Miller, MD/MPH Candidate, Class of 2017, Texas Tech University HSC, El Paso Paul L. Foster School of Medicine.

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Chilaiditi Syndrome: A Rare Entity with Critical Presentation

Majd Michael, MD
Richard W. McCallum, MD

Abstract
Chilaiditi syndrome is a rare entity in which interposition of bowel between the liver and right hemidiaphragm manifests with abdominal pain, nausea, vomiting, anorexia, and constipation that can be easily misdiagnosed as bowel perforation or intestinal obstruction. Most patients with radiologic evidence of hepatodiaphragmatic interposition of bowel remain asymptomatic (i.e., Chilaiditi sign). We report the case of a patient with Chilaiditi syndrome who suffered from acute on chronic abdominal pain accompanied by nausea and vomiting, and underwent exploratory laparoscopy based on a working diagnosis of small bowel obstruction.

Case
50-year old Hispanic male presented to our hospital with severe 8/10 abdominal pain, localized to the right upper quadrant, and associated with nausea, vomiting and anorexia. His medical history included chronic kidney disease, rheumatoid arthritis, hypertension, and previous hospitalizations for unexplained abdominal pain. Initial evaluation included an abdominal ultrasound which revealed cholelithiasis without evidence of cholecystitis. This was followed by a CT of the abdomen without contrast that revealed dilated loops of small bowel, mostly of the jejunum and ileum, concerning for a small bowel obstruction. Subsequently, the patient underwent an exploratory laparoscopy with a preoperative diagnosis of small bowel obstruction. Laparoscopic exploration of the bowel did not reveal any obstruction or ischemia. However, the surgeons described segments of dilated small bowel and evidence of increased peristalsis.

Symptoms persisted after the exploratory laparoscopy and a new CT of the abdomen, and pelvis was obtained on post-operative day 5 that showed diffusely dilated colon without any evidence of mechanical obstruction suggesting colonic ileus or Ogilvie syndrome. In addition, a colonic loop at the hepatic flexure was interposed anterior to the liver and between the superior margin of the liver capsule and the diaphragm [figures 1-2]. This displaced colonic bowel loop is consistent with Chilaiditi sign, and explains the patient’s symptoms. Our patient was stable during ongoing observation post-operatively, but continued to have significant abdominal pain and food intolerance that recovered gradually and was able to be successfully discharged.

Discussion
Chilaiditi sign is a rare radiologic finding in which hepatodiaphragmatic interposition of the colon or small bowel occurs.

This sign was first described in 1910 by Demetrius Chilaiditi, a Greek radiologist who practiced in Vienna, Austria. He described three cases of patients with bowel interposition between the liver and diaphragm.1 Chilaiditi sign is a radiographic term, as most patients with this anomaly will remain asymptomatic throughout their lives. Those who become symptomatic will develop Chilaiditi syndrome, which manifests with intermittent abdominal pain, distention, vomiting, anorexia, and constipation that may require surgical intervention.2

The ability to recognize Chilaiditi sign is crucial, as it is commonly misinterpreted as free air under the diaphragm, pneumoperitoneum, which is an indication for immediate surgical exploration based on a working diagnosis of a perforated viscus; e.g. peptic ulcer or colonic diverticulum. The prevalence of Chilaiditi sign is around 0.025 to 0.28% of general population, more prevalent in males than females, and the incidence increases with age. The bowel segments, most commonly found interposed between the liver and diaphragm or abdominal wall are the colonic hepatic flexure and transverse colon, although interposition of the small bowel has been reported.3 Recognition of this condition becomes especially important when

Continued on page 23
Performing interventional procedures such as hepatic biopsy and percutaneous gallbladder drainage.

Suspensory ligaments and fixation of the colon normally prevent interposition of the colon between the liver and diaphragm. Hence, any variations in normal anatomy can lead to the interposition of the colon. Anatomic distortions may predispose patients to the development of Chilaiditi sign, including decreased liver dimensions, elongation of the ligamentous suspension of the liver, and redundancy of the colon. Other described anomalies associated with this sign are right hepatic lobe segmental agenesis, relaxation or genesis of the mesentery suspensory ligaments, and hypermobile transverse mesentery and transverse colon. Another predisposition to Chilaiditi sign is elongation of the lower thoracic cage in chronic obstructive pulmonary disease, leading to a broader space where colonic interposition can occur. The prevalence of Chilaiditi sign in cirrhotic patients without ascites is much higher than in the general population, possibly explained by the smaller volume of the cirrhotic liver. A retrospective study of the relationship between Chilaiditi syndrome and obesity concluded that obesity may be a contributing factor in the etiology of Chilaiditi syndrome, and the difference in fat deposition between men and women may account for the increased prevalence in men, who are more likely to have “central” or visceral organ obesity. Mental illness and schizophrenia may also increase incidence of Chilaiditi sign.

**Figure 2.** Diffusely dilated colon with interposed colonic loop behind and superior to the liver, and no evidence of mechanical obstruction.
Chilaiditi sign has been associated with a variety of functional disorders, such as chronic constipation (colonic elongation and redundancy), aerophagia, cirrhosis, diaphragmatic paralysis, chronic lung disease, obesity, and multiple pregnancies.

Patients with Chilaiditi syndrome usually present with gastrointestinal symptoms followed by respiratory distress, and less frequently angina-like chest pain. Gastrointestinal symptoms include anorexia, nausea, emesis, abdominal pain, distension, potentially with serious complications including volvulus and bowel perforation. Our patient presented with these symptoms requiring hospitalization in the past for recurrent vomiting and abdominal pain episodes, which had no explanation. A working diagnosis of cyclic vomiting syndrome was being considered. Intestinal pseudo-obstruction which can overlap with Ogilvie syndrome, has also been described in a patient with Chilaiditi syndrome.

Diagnosis of Chilaiditi syndrome is based upon clinical findings and signs observed on plain radiographs and CT scans. CT scans of the abdomen enables clinicians to exclude diaphragmatic hernia and differentiate subphrenic fluid, true pneumoperitoneum, and air within the bowel lumen. This differentiation is of a critical importance because perforation can also complicate Chilaiditi syndrome when the involved bowel segment strangulates, and eventually perforates. The radiologic differential diagnosis is established by observing an elevation of the right hemidiaphragm due to caudal displacement of the liver, haustral markings between the liver and diaphragmatic surface, and the absence of image displacement with changes in the patient’s position. Pneumoperitoneum and subdiaphragmatic fluid collections have different characteristics being more mobile on lateral decubitus radiographs, and can be accompanied by pulmonary findings such as ipsilateral pleural effusion and basilar atelectasis.

In most cases of Chilaiditi syndrome, management is conservative and consists of bowel decompression, bowel rest, and aggressive fluid rehydration. Patients who fail conservative therapy should undergo an exploratory procedure. Colonoscopy should be performed with great caution considering the risk of gas being trapped in the acutely angulated and interposed bowel, potentially leading to perforation. Administration of carbon dioxide as the insufflating agent for colonoscopy is appropriate for decreasing this risk because it is nonexplosive, rapidly absorbed, and increases colon blood flow.

REFERENCES


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Many patients receive medical care, but do not take the necessary action to improve their health due to a lack of knowledge or motivation. This can be quite frustrating for physicians, who want to see their patients’ health improved but unfortunately don’t have the time to devote more than 30 minutes on a single healthcare topic, such as screenings or nutrition. This can be especially difficult in free clinics, where the line to see a physician is out the door.

Consequently, the Baptist Clinic teamed up with Paul L. Foster medical students to create the Baptist Clinic’s Community Education Outreach Program. Initiated in 2016, the program’s purpose is to both educate patients about their diseases as well as teach prevention strategies, but do so in an open discursive environment. This allows patients to ask all their questions, however many there may be, and gain the education necessary to drive out previous misconceptions. Without a good understanding, patients will keep coming back with the same preventable disease processes, like uncontrolled hypertension or diabetes. Additionally, they will inadvertently share these poor practices with their family and friends.

Currently, the program hosts around 5 sessions per month, in a small group setting of 12-15 patients each session. Medical students create a demonstration to teach patients about a topic such as healthy cooking, diabetes, hypertension, heart-healthy lifestyle, or the importance of screening exams such as colonoscopies and mammograms. Educational material is obtained from nationally wide accepted references, including the CDC & U.S Preventative Services Task Force. The target population is given the opportunity to learn the importance of and to practice new lifestyle behaviors. Additionally, these sessions are opportunities for patients to ask detailed questions relating to their individual choices. Patients are given brochures, recipes, and reading material (ex MyPlate Food balance table) to help them adjust at home and report feeling better equipped to care for their own health after attending an educational course about their disease or life circumstances. Not only are the patients empowered to better care for their own health, the medical students that are teaching learn about the psychosocial impact these health conditions have on the patients’ lives. These experiences will help the medical students to be more attuned to the challenges and misconceptions patients have about their health care, and ultimately better communicators with their own patients.

In the future, the organization desires to increase the number of sessions and to incorporate educational input from physicians, nutritionists, and other healthcare providers. Additionally, the medical students hope to investigate the effects of these sessions on patients’ preventative health strategies, lifestyle behaviors, and overall health. The goal is to achieve long-term improvement of quality of life for the patients and provide them with the correct resources to achieve their goals. If you are interested in becoming a collaborator with this organization, please contact Sumana Reddy at sumana.reddy@ttuhsc.edu.


Impact of Medical Student Interventional Radiology Symposium

Nabih Diab, BS; Haley Swanson, BS; Nassim Akle, MD

Introduction: In 2012, the formation of an integrated Interventional Radiology (IR) residency, now a primary specialty, was approved by the ACGME. Medical student IR exposure during preclinical years is minimal. The goal of “IR Week at Texas Tech” was to increase medical student awareness and interest in IR.

Methods: A cross-sectional survey of the 25 attendants at the symposium was conducted using Survey Monkey and data was analyzed to evaluate the symposium’s effectiveness.

Results: 20 attendants, 7 females and 13 males, responded to the survey. 85% were preclinical. Prior to the symposium, 60% were considering IR as a career and 55% have been to an IR event. After the symposium, 80% of the students were considering IR as a career choice and 100% were interested in attending more IR events.

Conclusion: “IR Week at Texas Tech” successfully introduced Paul. L. Foster medical students to the field of IR. Attendance at the three-day symposium resulted in a 33% increase in the number of students considering IR as a career and generated interest in future IR events. We can conclude that medical student symposiums are useful tools to recruit future IR applicants.
Zika Field Support Assigned to the Sun City

Flor Puentes, MPH

Thank you for the opportunity to introduce myself and my new role with the City of El Paso Department of Public Health (DPH). I represent the Centers for Disease Control and Prevention (CDC). I came on board in April of this year to join forces with the City in the fight against Zika virus. From 2,800 eligible local health departments in the U.S., only 25 were selected to host a CDC field appointee. Fortunately, El Paso was selected along with three other cities in the state of Texas. An El Paso native, I graduated from UT Houston School of Public Health-El Paso Regional Campus in 2007 with a Masters in Public Health. I bring to the position a unique mix of border health research experience and a sincere concern for everyone in our community.

My role as a CDC Field Epidemiologist will be to focus on the following Zika virus related activities:

- Expanding clinical outreach and communication to community healthcare providers
- Assisting in proper testing of at-risk pregnant women
- Sharing Zika information with the community at-large through community meetings, events, and other forums
- Collecting data on pregnant women with laboratory evidence of Zika virus infection and their infants with emphasis on birth defects
- Developing and implementing protocols for case management of Zika positive pregnant women and their infants to ensure proper follow up.
- Providing current educational tools to at-risk pregnant women-following CDC guidelines during outreach activities
- Linking patients to care and other appropriate services

During the Zika and other arboviruses season, I will assist with community and healthcare provider outreach, a core component of our local Zika plan. Educational materials and training will be offered to all local health care providers including private and academic healthcare providers, Federally Qualified Health Centers (FQHC), promotoras (community health workers), the El Paso County Medical Society and its members, the Association of Professionals in Infection Control (APIC), area hospitals through grand rounds, academic health centers, March of Dimes, WIC and other interested agencies. Educational packets will include information on updated CDC Zika Interim Response Plan, CDC US Zika Pregnancy Registry, Healthcare provider toolkit for Health Departments, Zika pregnancy registry and birth defect surveillance, Testing, diagnosing and reporting, and information on Zika Care Connect. Most of these educational materials are currently available on the DPH website: www.EPHealth.com and will be updated on a regular basis as new information becomes available.

I look forward to meeting you during my outreach activities, and welcome the opportunity to provide guidance and educational resources that will benefit our community, particularly those that are most vulnerable during this coming mosquito season. Together we can educate and empower our community to adopt a culture of prevention to minimize complications from mosquito-borne diseases. I can be reached via email at PuentesFX@elpasotexas.gov or at (915) 474-5523.

Together we can fight the bite both day and night.

Flor Puentes, MPH, CDC Field Support Epidemiologist, City of El Paso Department of Public Health.

Student Survey Project on Physician Volunteerism in El Paso

MS3 student Jennifer Nielsen at Paul Foster School of Medicine is doing a research project seeking to analyze the perceived benefits and barriers of volunteerism among physicians in free clinics in El Paso. The hope is that by establishing what motivates physicians, possible pathways through policy may be advocated for in order to better enable physician volunteerism in free clinics. (For example, the AMA might be able to advocate that physicians should receive CME credits or tax credits based on number of hours a physician volunteered at free clinics.) Please consider taking the following survey in order to participate in this worthy cause. It shouldn’t take more than 5 minutes. The survey link is posted on the EPCMS website home page: www.epcms.com

Thank you for your participation and service to medicine. Feel free to contact Jennifer Nielsen with any questions or need of assistance at 971-409-2258 or Jennifer.nielsen@ttuhsc.edu.

https://docs.google.com/forms/d/e/1FAIpQLSdzavdf52UGxTsaB78xPCI1OfGCxGJTb ок 2vCs/viewform?c=0&w=1&usp=mail_form_link
This closed claim study is based on an actual malpractice claim from Texas Medical Liability Trust. This case illustrates how action or inaction on the part of the physicians led to allegations of professional liability, and how risk management techniques may have either prevented the outcome or increased the physician’s defensibility. This study has been modified to protect the privacy of the physicians and the patient.

TMLT has seen an alarming increase in the number of claims filed related to Wernicke’s encephalopathy (WE) following bariatric surgery.

These claims involve allegations of failure to monitor thiamine levels in post-bariatric surgery patients and failure to treat symptoms of nausea, vomiting, visual disturbances, and motor impairments with thiamine supplementation. Specialties included in these claims are general surgery, emergency medicine, internal medicine, and gastroenterology.

A high index of suspicion is required when treating patients with a history of bariatric surgery presenting with symptoms suggestive of WE. Surgeons, emergency physicians, internists, gastroenterologists, and all health care professionals should be aware of the risk factors and symptoms associated with nutritional deficiencies to minimize any adverse effects. Early recognition of symptoms is important for ALL providers caring for this patient population.

Physicians treating these patients could be subjected to litigation if they do not order labs checking thiamine levels when a patient who has recently undergone bariatric or gastric surgery exhibits symptoms detailed in this risk alert.

Closed claim study: failure to diagnose thiamine deficiency
Presentation
A 22-year-old woman came to a bariatric surgery center for treatment of her morbid obesity. She underwent extensive diagnostic testing and education before being scheduled for a sleeve gastrectomy. A general surgeon performed the procedure on June 25.

Physician action
The patient’s postoperative course was complicated by persistent dysphagia and gagging when eating (as opposed to vomiting). On August 6, the general surgeon ordered multiple studies that ruled out a structural cause for the patient’s symptoms. Nutrition/vitamin levels were not checked.

The patient claimed that on August 20, her mother called the bariatric surgery center and reported that the patient was unable to drink protein drinks or take her multivitamins. The mother asked the nurse if the patient could switch to a gummy vitamin that would be easier to swallow. Purportedly, the nurse said that a gummy multivitamin would be fine.

No one spoke to the general surgeon about this change or about the patient’s inability to keep protein shakes down. The general surgeon later testified that she would not have recommended the change to a gummy vitamin.

The patient’s mother called the surgery center on August 23 to report that the patient had continued nausea, vomiting, diarrhea, and was now experiencing double vision. The nurse from the surgery center advised that she had spoken to the general surgeon who said the symptoms should go away within three months of the surgery.

On August 25, the patient came to the emergency department of a local hospital with symptoms of blurred and double vision. She was discharged and told to follow up with the general surgeon. The patient’s mother called the surgery center on August 26 to report that her daughter was seeing double. The general surgeon prescribed Pedialyte popsicles, Gatorade, and protein shake supplements. She also scheduled another EGD for August 27. This study did not reveal any cause for the nausea and vomiting.

Over the next week, the patient and her mother repeatedly called the surgery center to report nausea, vomiting, and blurry/double vision. The general surgeon prescribed metoclopramide and promethazine.

On September 6, the patient reported weakness, difficulty walking, and blurry/double vision. The general surgeon ordered additional labs and all results were reported as normal. Nutritional studies were not ordered.

The patient went to the ED on September 8 and was seen by a neurologist. He diagnosed Wernicke’s encephalopathy, and the patient was treated for thiamine deficiency. She recovered, but her visual disturbance is permanent and she has been determined to be legally blind. She also walks with an ataxic gait.

Allegations
Lawsuits were filed against the general surgeon and the bariatric surgery center. The allegations included:

- failure to recognize thiamine deficiency (general surgeon);
- failure to treat the patient’s nausea, vomiting, visual disturbances, and motor impairments with thiamine supplementation (general surgeon);
- failure to test the patient’s thiamine levels (bariatric surgery center).

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Wernicke-Korsakoff Syndrome (WKS) classically, but not always, presents with the clinical triad of confusion, ataxia, and nystagmus. Eighty-five percent of the survivors of the acute phase of Wernicke encephalopathy who remain untreated go on to develop Wernicke-Korsakoff Syndrome.”

In the case presented, there were several instances where a breakdown in communication and patient care occurred.

- The bariatric surgery center nurse did not consult with the general surgeon regarding the patient’s symptoms and the request for a medication adjustment. Had this step been taken, the physician could have had an opportunity to agree with the patient’s request or implement a different treatment plan.
- The surgery center did not follow the guidelines for monitoring patients’ post-operative nutritional status, as stated in their Patient Education Manual.
- Not ordering nutritional studies when the patient reported neurological symptoms was another missed opportunity.
- The physician performing the removal of the IUD administered 5% dextrose intravenously in a patient with a history of bariatric surgery, which is contraindicated.
- The patient did not tell the general surgeon about the IUD removal procedure.
- A high index of clinical suspicion is required when treating patients with a history of bariatric surgery presenting to any health care provider with symptoms suggestive of WE.
- While not an issue in this case, there have been cases in which the physician defendants contend that it was someone else’s responsibility to check the patient’s vitamin levels. Ultimately, there was a delay in getting the needed lab work and this led to catastrophic patient outcomes. Some of these physicians stated that they felt the patient seemed to be eating well, so checking nutrition was not necessary.
- Cases have also been documented in which the appropriate lab work was ordered, but not completed. Developing and consistently following procedures for monitoring and acting on test results may prevent these results — or lack of results — from being overlooked.

Sources


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The following is a list of new/re-instated members of the El Paso County Medical Society. Congratulations to all new members!!!

AKLE, NASSIM, MD
R
Catholic University of Louvain, 2004
5001 El Paso Drive, CSB-A02, El Paso, TX 79905
(915) 215-6000

ANEKWE, EMMANUEL C., MD
HOS
University of Nigeria College of Medicine, 1996
7500 N. Mesa, Ste. 210, El Paso, TX 79912
(915) 307-2631

BENITEZ, ADOLFO L., MD
FM
Facultad de Medicina de la Univ del Valle, 1999
3270 Joe Battle, Ste. 100, El Paso, TX 79938
(915) 504-6900

CAJAS, OSWALDO C., MD
GP
University of Guayaquil, Ecuador, 1967
1721 Lee Trevino, El Paso, TX 79936
(915) 590-9424

CHAVEZ, EVA P., MD
PS HSP
UT Health Science Center at San Antonio, 1999
1517 N. Mesa, El Paso, TX 79902
(915) 533-0269

GURMENDI, ALFREDO F., MD
TS GS
UT Health Science Center at Houston, 1992
1416 George Dieter Dr., El Paso, TX 79936
(915) 849-5127

HWANG, RICHARD Y., MD
OPH
Duke University School of Medicine, 2010
1700 Curie Dr., Ste. 3800, El Paso, TX 79902
(915) 532-3912

JIMENEZ, MAURICIO E., MD
IM
Universidad Federal de Espirito Santo, 1981
1501 N. Mesa, Ste. 2, El Paso, TX 79902
(915) 532-1222

JOHNSON, OWEN N. III, MD
PS PSP
University of Virginia School of Medicine, 2004
5005 N. Piedras St., El Paso, TX 79920
(915) 742-3444

JONES-ALLEN, ANGELA S., MD
FM
West Indies Medical School, 1992
4824 Alberta Ave., ANNEX 4th Floor, El Paso, TX 79905
(915) 521-7866

KASUGA, DANIEL T., MD
OPH
Georgetown University School of Medicine, 2010
1700 Curie Dr., Ste. 3800, El Paso, TX 79902
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LOIZEAUX-WITTE, JOHN N., MD
DR MSR
University of Southern California School of Medicine, 1990
2100 N. Oregon St., El Paso, TX 79902

LOPEZ, Raul J., MD
APM AN
Ohio State University College of Medicine, 2008
3215 Gateway Blvd West, El Paso, TX 79903
(915) 598-7246

MARZOUK, ISSAM EL-DEAN, MD
PCC IM
University of Alexandria, 2002
4305 N. Mesa, Ste. A, El Paso, TX 79902
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OMAR, ASAD, MD
IM PUD
St. Matthew’s University , British West Indies, 2010
4849 N. Mesa, El Paso, TX 79912
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Continued on page 31
OMAR, FAHAD, MD
IM PCC
Ross University School of Medicine, 2009
1011 Montana Ave., El Paso, TX 79902
(915) 702-0030

PADILLA, LUIS A., MD
GO
Facultad de Medicina Universidad Anahuac, 1992
4532 N. Mesa St., 3rd Floor, El Paso, TX 79912
(915) 317-5077

SAFDAR, AMAR, MD
ID IM
Dow Medical College, 1989
3270 Joe Battle Blvd, Ste. 315, El Paso, TX 79938
(915) 206-2150

SIMPSON, BRENDA M, MD
D
Oregon Health and Science University School of Medicine, 2011
1700 Murchison Dr., Ste. 215, El Paso, TX 79902
(915) 544-3254

SUNDRANI, SHANKER S., MD
NS N
Sarojini Najdu Medical College Agra University, 1977
3028 Trawood, Ste. C, El Paso, TX 79936
(915) 590-1890

TEEGARDEN, ERNEST A., MD
AN PM
College of Osteopathic Medicine, 1967
7300 Remcon Cir Ste 200, El Paso, TX 79912
(915) 532-3600

THOMAS, MARK D., MD
U
Texas Tech University HSC-Lubbock, 1982
4687 N. Mesa, Ste. 100, El Paso, TX 79912
(915) 532-3119

WRIGHT, RACHEL H., MD
NPM PD
UT Medical Branch, 2005
4845 Alameda Ave., El Paso, TX 79905
(915) 298-5444

YATES, MARIANA M., MD
IM END
Universidad Central de Venezuela, 2004
4800 Alberta Ave., El Paso, TX 79905
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**Bariatric Surgery**

**Benjamin L. Clapp, MD, PA, FACS**  
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El Paso, TX 79902  
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**Obstetrics / Gynecology**

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**Ophthalmology**

**Louis M. Alpern, MD**  
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### Ophthalmology (Continued)

**David R. Schecter, MD**  
**Daniel G. Blumenfeld, MD**  
Diplomate, American Board of Ophthalmology  
1220 N. Oregon  545-1484  
1200 Golden Key, Ste 163  593-1226  

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**Javier De La Torre, MD**  
**Ahmed Soliman, MD**  
**James Cole, MD**  
**Stephen Purdy, OD**  
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### Pediatrics

**Roberto Canales, MD**  
Pediatric Hematology, Oncology and Intensive Care  
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### Pediatric Ophthalmology

**Violeta Radenovich, MD, MPH**  
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