# OHIO PA Insider

Special August 2023 Issue

## Dr. V's Benefit Concert

*By Jakob Barzak, PA-S & Melissa Ratzel, PA-S*

On Friday, April 14th, students from the Ohio University Physician Assistant Program visited the McConnell Center for the Arts in Worthington for a benefit concert. Students from the Cohorts of 2023, 2024, and 2025 were in attendance to celebrate the nearing end of the spring semester with a night of classic music from their talented professor, Dr. Jeff Vasiloff.

The night began with a performance from a local artist, Adena Brooks, who played a combination of original songs and American classics like “Jolene” by Dolly Parton. Brooks started playing the guitar in high school and has since gone on to play at numerous notable venues in the Columbus area and beyond.

At the conclusion of her set, students from the Class of 2025, Melissa Ratzel and Jakob Barzak, took the stage to introduce their professor, “Dr. V.” Ratzel joked that Dr. Vasiloff was unable to get his good friends, Harry Styles and Dua Lipa, to attend the concert. Barzak followed by reading the poem “The Laughing Heart" by Charles Bukowski. Vasiloff (vocals and mandolin) then took the stage with fellow band members Jake Vasiloff (drums/percussion), Joe Brenneman (woodwinds and vocals), Stan Smith (lead guitar and music director), Taylor DeVault (lead and acoustic guitar), and former student Tom Nasr (bass guitar). Vasiloff, who has written numerous original songs, presented some of his own music alongside some well-known classics.

After a brief intermission, several students participated in a comedic skit with Dr. Vasiloff. Class of 2024 and 2025 students Justin Kowalski (God), Reid Katz (Tall Man), and Will Lawson (Strong Man) acted alongside Vasiloff and his son, Jake (Young Man). After the audience recovered from a good chuckle, three student awards were presented, and the band retook the stage to close out their set.

Figure 1: Dr. Vasiloff, Tom Nasr (Class of 2023), performing at the McConnell Center for the Arts

## Inside This Issue...



Figure 2: Jakob Barzak, PA-S & Melissa Ratzel, PA-S

On behalf of the Ohio University Physician Assistant Program, we would like to thank everyone that attended the concert for supporting our program. It has allowed us to plan future events in the community as well as support local charity groups. We also want to thank Adena Brooks, Dr. Jeff Vasiloff, and the band members for their excellent performances. We look forward to next year's concert and encourage everyone to attend!

Figure 3: Students from all three cohorts were in attendance for Dr. V's concert

# Ohio PA at AAPA 2023

Four Ohio PA students from the class of 2023 were selected to present their posters at the American Academy of Physician Associates in May, 2023. Katelynn Kinley, PA-C presented her case study of, "An Unusual Case of Lymphadenopathy." Hannah Barker, PA-C presented her case study of, "An Unusual Case of Neck Pain." Kaylee Ramsey, PA-C presented her case of, "Persistent Hyperparathyroidism." Nick Musico, PA-C was unable to attend in person, so Dr. Vasiloff presented their case study on, "Hip Fracture, Stroke, and a Mystery Embolus."


Figure 4: Katelynn Kinley, PA-C presenting at AAPA 2023

## An unusual case of lymphadenopathy: case study

Katelynn Kinley, PA-S; Jeffrey Vasiloff, MD, MPH

### Background

Localized lymphadenopathy in and near the head and neck has a wide differential diagnosis including **common benign conditions and less common serious diseases.** Common causes include reactive lymphadenopathy from a nearby infection **(strep throat, mono, CMV, toxoplasmosis, acute HIV, cat scratch disease).** TB and atypical mycobacteria can also cause cervical lymphadenopathy, as well as systemic lupus erythematosus, as well as lymphoma and **cancers of the head and neck**. We present a case of patient with **localized lymphadenopathy and pain for 6 weeks.**

### Case

A 52-year-old male presented to his primary care office with swelling in his right neck with associated pain x 6 weeks. Patient was **concerned for a pulled muscle** and had tried ibuprofen and ice without relief. His wife was concerned about a thyroid problem and suggested he see a doctor. With his PCP, he **denied sore throat, fever, cat scratches or bites, weight loss, and had no history of malignancy.** He had a 31-year history of smoking.

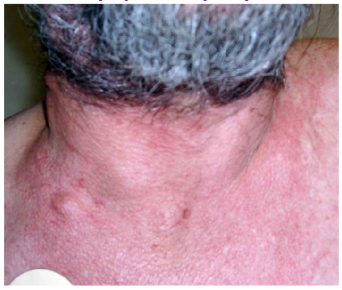
### Physical Exam / Imaging

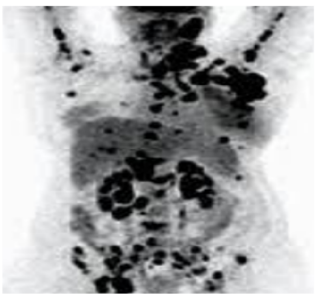
On exam he was a middle-aged man who **appeared well**. T 98.9, P75, R14, BP118/79. HEENT exam was unremarkable, but he had a 1.5 x 1 cm right supraclavicular mass that was extremely tender, soft, but not moveable. Thyroid exam was normal. He had no lymphadenopathy elsewhere nor any hepatosplenomegaly. **The rest of the exam was unremarkable.** Bloodwork was unremarkable. Chest X-ray was normal. **CT of the neck and chest revealed lymphadenopathy involving multiple thoracic compartments.** He underwent fine-needle aspiration of the mass. Pathology revealed **adenocarcinoma.** The specimen was positive for TTF-1, cytokeratin AE 1-3, Ber-EP4, and MOC-31. PET scan revealed more extensive lymphadenopathy in the following areas: **cervical, internal mammary, right subpectoral, mediastinum, and hila, as well as some bone metastases.** No primary lung mass was seen.

### Diagnosis and Treatment

**He was diagnosed with adenocarcinoma of unknown primary;** however, it was speculated that the origin was most likely the lung. He received cycles of carboplatin, Taxol, and Avastin.

Example of Supraclavicular Lymphadenopathy





Widespread disease on PET/CT scan typically found at diagnosis in patients with CUP.

### Final Disposition

His clinical course was complicated by DVTs and PEs, and he ultimately succumbed from his disease 18 months after diagnosis. Carcinoma of unknown primary (CUP) is a catastrophic illness that is poorly understood. CUP is a heterogeneous disease of varying subtypes including **adenocarcinoma** (70% of cases); undifferentiated cancer (20-25%), squamous cell carcinoma (5%); and neuroendocrine cancers (1%). Treatment is directed toward the suspected primary. **This identification is made better by advances in immunohistochemistry as well as molecular cancer classifier assays**. Going forward, earlier diagnosis is paramount, and fortunately, with screening for lung cancer in heavy smokers, and the recent development of liquid biopsies, the tide will hopefully be turning in these cases.

Figure 5: Katelynn Kinley, PA-C & Dr. Vasiloff, M.D. poster from AAPA 2023

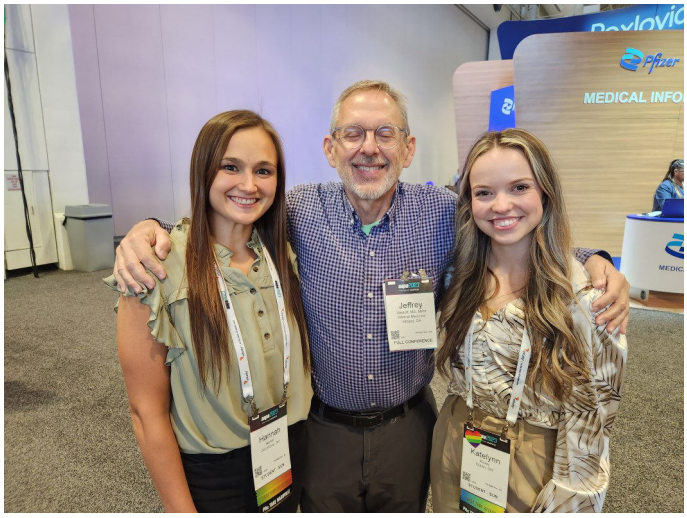



Figure 6: Hannah Barker, PA-C, Dr. Jeffery Vasiloff, M.D. & Katelynn Kinley, PA-C at AAPA 2023

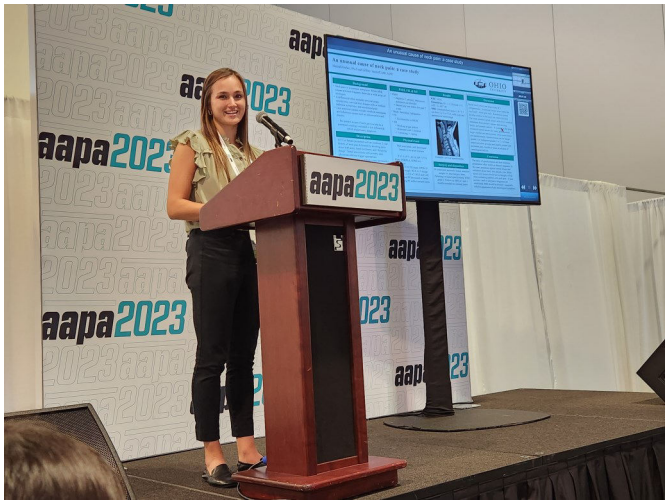



Figure 7: Hannah Barker, PA-C, presenting at AAPA 2023

## An unusual cause of neck pain: a case study

Hannah Barker, PA-S and Jeffrey Vasiloff, MD, MPH

OHIO UNIVERSITY

### Background

- Neck pain is a common complaint. While MSK causes are most frequent, there are a wide array of etiologies.
- A differential often includes cervical strain, spondylosis, cervical disc disease with or without radicular symptoms, and myofascial pain.
- Less common causes include vertebral metastases and infectious causes such as osteomyelitis and discitis.

We present a case of neck pain not due to an etiology commonly included in the differential,

### Description

A 33 yo female presented with an insidious 2 year history of neck pain followed by shooting pains down both arms, hand numbness, and "dropping things." Initial encounters with primary care physicians did not trigger appropriate investigation—as her symptoms were attributed a past history of substance abuse, anxiety and depression, and chronic hepatitis C. Eventually plain films were done, which showed only mild spondylosis. EMG revealed L C5, C6, C7, and C8 radiculopathies, as well as on the R side at C7 and C8. Pain and hand weakness persisted so she was referred to a specialist.

### PMH, FH, & SH

- PMH
  - Hepatitis C, anxiety, depression, substance use disorder
  - Denies any use within the past 2 years
- Meds: Sertraline, Gabapentin
- FH
  - Glioblastoma in MGM
- SH
  - Working at gas station
  - Widowed with 3 children
  - + smoking, + vaping

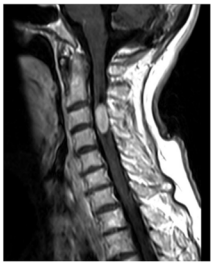
### Physical exam

Well nourished, well developed female in no acute distress.

T 97.9 | P 71 | R 18 | BP 117/74
**Eyes**: PERRLA, EOMI w/o nystagmus
**Neuro**: CN II-XII intact, DTRs 2+
**MSK**: Strength: RUE 4+/5 except hands 4/5; LUE 4/5; RLE and LLE 5/5; LT and PP decreased in hands.
Remaining PE within normal limits.

### Results

**WBC**: WNL
**Chemistries:** K+: 3.2, Glucose: 110, AST: 72, ALT: 80
**MRI:** intramedullary tumor 1.8 * 3.9 cm from C2-3 to C5-6, with cysts and peritumoral hemorrhage



Example: cervical ependymoma

### Surgery and disposition

Pt underwent successful tumor resection surgery by neurosurgery team. Pathology revealed ependymoma, WHO grade 2. Follow-up MRI at 3 and 6 months revealed no residual tumor.

### Discussion

Spinal cord tumors can arise in any of 3 compartments. Intramural tumors arise within the cord itself—from glial cells. Extramural tumors develop exterior to the cord, but within the dura. The most common spinal tumors arise from vertebral metastases from primary cancers such as lung, breast, and prostate cancers. In contrast to the brain, intramural spinal tumors are rarely glioblastomas. A larger number are astrocytomas, while most tumors are ependymomas. Ependymomas can be benign (grade 1) or malignant (grades 2 or 3). All these tumors are slow growing and usually present with persistent pain, months or years in advance of neurological

### Conclusion

The rarity of ependymomas and their insidious presentation often results in delayed diagnosis. In this case, prejudices against mental illness and substance abuse likely also played a role. While spinal cord tumors should not be thought of first when evaluating patients with neck pain—if pain persists—and no alternative diagnoses are established, MRI should be pursued—especially with the appearance of any neurological symptoms.

Figure 8: Hannah Barker, PA-C & Dr. Vasiloff, M.D. poster from AAPA 2023

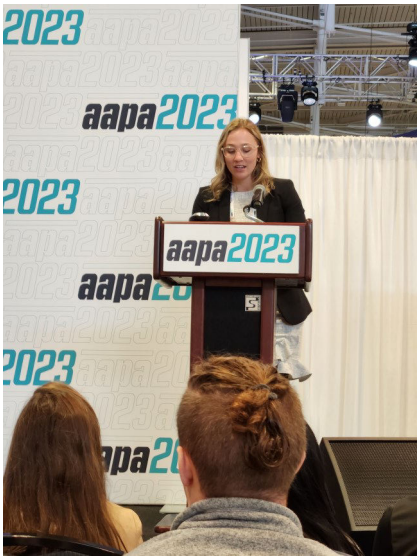



Figure 9: Kaylee Ramsey, PA-C presenting at AAPA 2023

Figure 10: Dr. Jeffery Vasiloff, M.D. & Kaylee Ramsey, PA-C at AAPA 2023

## A Case of Persistent Hyperparathyroidism

Ramsey K and Vasiloff J
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**Background**

Hyperparathyroidism is the third most common endocrine disorder diagnosed in the US and one of the most common causes of hypercalcemia. Most cases are due to a single adenoma or hyperplasia of the glands. Aberrant production of parathyroid hormone (PTH) leads to loss of feedback system in the PTH-calcium loop and causes elevated levels of calcium in the blood via increased kidney, GI, and bone resorption. This can cause symptoms including osteoporosis, nephrolithiasis, GI upset, and mood changes. We present an unusual case of persistent hyperparathyroidism.

**Description of Case**

JD is a 50 yo male who presented to new PCP to establish care. Initial laboratory studies were ordered and returned unremarkable, except for elevated calcium. Subsequent labs were drawn to investigate etiology and demonstrated elevated PTH and decreased 25-OH-Vitamin D. Review of systems (ROS) was positive for fatigue and headache, but the remainder of ROS was negative. Most likely diagnosis was primary hyperparathyroidism. Patient was referred to endocrinologist and ENT surgeon for removal of culprit parathyroid. Two surgeries failed to locate parathyroid adenoma or hyperplasia and laboratory evidence continued to demonstrate primary hyperparathyroidism. Patient was referred to tertiary medical center for evaluation. Specialized work-up failed to locate adenoma or hyperplastic tissue.

**Lab Evaluation**

**Initial:**

Calcium: 11.2 mg/dL

Intact PTH (Normal Values 14-71 pG/mL): 119.6 pg/mL

25-OH-Vitamin D (Normal values 30.0-100.0 ng/mL): 29.6 ng/mL

**Current:**

Ca 10.9 mg/dL

PTH: 105 pg/mL

24-hour Urine Calcium: 420 mg

**Imaging**

11/17/20 Sestamibi (Without SPECT-CT)
Negative
6/14/21- Sestamibi/SPECT-CT
Intense uptake in mid to inferior right thyroid
DEXA
Normal
1/28/22 Sestamibi (Without SPECT-CT)
Negative
8/16/22 4D Parathyroid Scan
Negative
9/13/22 Venous Sampling
Venography demonstrated left sided IVC, the internal jugular veins did not demonstrate distinct thyroid veins to sample. Due to abnormal anatomy, procedure was aborted prior to completion of sampling. Of sampling completed, parathyroid hormone ranged from 105-220 pG/mL. The 220 pG/mL collection was located in the mediastinum, indicating possible adenoma there.

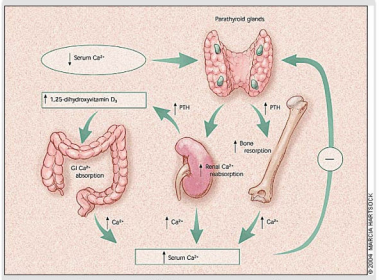



Figure 1. Parathyroid Hormone Feedback Loop

**Treatment**

Medications:
Vitamin D3 1,000u PO QD
3/25/21 Parathyroid Exploration, Left thyroid lobectomy
Left lower parathyroid not located, the others had normal appearance. Left thyroid lobectomy performed due to missing parathyroid in which biopsy demonstrated no parathyroid tissue.

Biopsy Parathyroid glands: Normocellular parathyroid tissue

Post-op PTH: 162

7/29/21 Parathyroid exploration, right thyroid lobectomy
Ectopic tissue, possibly ectopic parathyroid, near cricoid cartilage. This tissue was dissected and demonstrated probable hypercellular parathyroid tissue. Right thyroid excised and benign.
Post-op labs: PTH 130

**Discussion**

Hyperparathyroidism, although often asymptomatic, can have injurious effects on the body, including osteopenia to osteitis fibrosa cystica and fragility fractures to kidney failure. When left unmanaged, these outcomes can be devastating and have significant morbidity and mortality. Most cases of hyperparathyroidism occur due to single adenoma or area of hyperplasia and surgical excision will cure the illness. However, in our case, exhaustive diagnostics have been done and the causative agent has yet to be found, therefore classifying as persistent hyperparathyroidism. Currently, our patient's final management is uncertain due to the unclear etiology of aberrant parathyroid hormone production. Familial syndromes have been ruled out. Our patient continues to undergo specialized workup.

**Conclusion**

It is important to thoroughly evaluate patients with primary hyperparathyroidism to avoid devastating manifestations including bone loss, fractures, and kidney disease. This is especially true in patients of younger age without documented bone or kidney disease to prevent progression with age. Therefore, providers must be vigilant in detecting and pursuing primary hyperparathyroidism.

**Acknowledgements**

Fig. 1- Hartsock , M. (2004). *Hyperparathyroidism.* American Family Physician .

Figure 11: Kaylee Ramsey, PA-C & Dr. Vasiloff, M.D. poster from 2023

# Hip Fracture, Stroke, and a Mystery Embolus: Case Study

Nicholas Musico, PA-S, Jeffery Vasiloff, MD, Melissa Bowlby, PA-C
Ohio University Physician Assistant Program

## Background

Stroke is a common cause of death and disability. Common mechanisms include: a) subarachnoid hemorrhage; b) intraparenchymal hemorrhage; c) carotid or intracranial thrombosis; and d) cerebral embolism. Cerebral emboli commonly arise from left atrial thrombi associated with atrial fibrillation. Less common sources include mural thrombi in patients s/p MI, or vegetations in patients with left-sided endocarditis.

We report a case of a cerebral embolus that did not originate in any of these sites.

## Case Description

A 75 y/o woman fell but did not seek medical attention for left hip pain for 24 hrs. Imaging revealed a L femoral neck fracture. PMH was positive for paroxysmal atrial fibrillation, HTN, hyperlipidemia, hypothyroidism, and s/p R THA. Medications included lisinopril, atorvastatin, L-thyroxine, and ASA. T 98.8; P 104; BP 142/94; O2sat 98%. PE was consistent with L hip fracture, but was otherwise unremarkable. She underwent successful L ORIF. Postoperatively, she became tachypneic and hypoxemic—and stated she could not move her left arm.

## Stroke Assessment

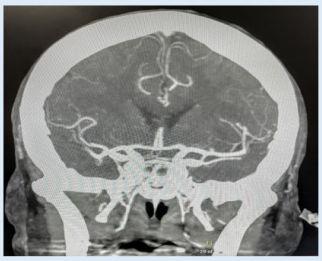
NIH-SS score of 11. Her presenting symptoms included left arm paresthesia, weakness, left facial palsy, left sided neglect and dysarthria. She was oriented to person and time but not to place.

Stroke evaluation included negative CT for bleeding, but CTA revealed occlusion at M2 of the right middle cerebral artery. ECG was negative for atrial fibrillation, and echocardiography failed to reveal a source of the presumed cerebral embolus. Emergent catheter-directed thrombectomy was successfully accomplished. After the procedure, the patient again had hypoxemia. Further work up revealed a DVT in the left iliac vein as well as pulmonary emboli.

## Conclusion

Transcranial Doppler ultrasonography with bubble test was positive for a communication between the two sides of heart, consistent with a patent foramen ovale (PFO). Repeat CT of the brain revealed contrast staining versus minimal bleeding, thus, anticoagulation was not begun. Rather, the patient underwent percutaneous placement of an inferior vena cava filter. The patient had no further pulmonary or cerebral emboli and regained some strength on the left side prior to transfer to inpatient rehabilitation.

## Picture showing the M2 artery occlusion



## Discussion

A patent foramen ovale is an opening between the atria that fails to close on its own after childbirth. It has been shown to remain in 25% of people. While strokes derived from a PFO are still uncommon, those with a cryptogenic stroke have been shown to be four times more likely to have a PFO. Because 40% of ischemic strokes are cryptogenic, it is important to investigate this possibility in appropriate patients, such as our case, who appears to have suffered a stroke from a "so-called" paradoxical embolus—beginning in the venous circulation. This is why it is called a paradoxical embolus, because venous clots "should only" embolize to the lungs—not the brain.

Once a PFO is identified, it can be closed, decreasing the risk of stroke. The 2017 RESPECT study showed closing a PFO could greatly reduce the risk of further strokes in patients with histories of cryptogenic strokes. Ten year follow up of a series of patients who underwent percutaneous implantation of a PFO closure device showed 45% reduction in rate of strokes. In our case, added protection was afforded by the inferior vena cava filter, which would decrease the risk of emboli to the lungs or paradoxically. While rare, it is paramount for patients deemed high risk to be evaluated for this as a cause of an acute ischemic stroke.

## Example of a PFO closure

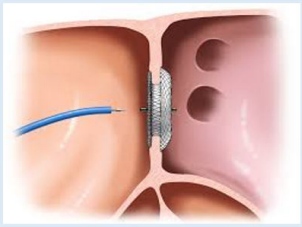


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Figure 12: Nicholas Musico, PA-C, Dr. Vasiloff, M.D. & Melissa Bowlby, PA-C poster from AAPA 2023

# Class of 2023 Hooding Ceremony


Figure 13: Class of 2023 Hooding Ceremony


Figure 14: Dr. Cacchillo, M.D. & Jennifer Vu, PA-C

## Congratulations to the Class of 2023 on their graduation and having a 100% pass rate on their recent PANCE examination!

Figure 15: Left to right, Justina Syu, PA-C, Andi Lamb, PA-C, Emily McCulloch, PA-C, & Meredith Mayfield, PA-C


Figure 16: Cassie Nemeth, PA-C providing the closing remarks

# Highlight on Environmental Diseases

*By Dr. Jeffrey Vasiloff, M.D., MPH*

Fortunately, homeostasis protects us from many environmental challenges such as high or low ambient temperatures. Nevertheless, lethal hyperthermia and lethal hypothermia do occasionally occur. But coping with climate and weather conditions is only one of many environmental exposures that can lead to illness and death.

Besides temperature extremes, other potentially harmful environmental exposures include: a) air pollution; b) water pollution; c) chemicals; d) toxins; e) poison gases; f) heavy metals; g) ultraviolet light; h) ionizing radiation; i) high altitude; j) high atmospheric pressure (in diving); and so on.

Of literally hundreds of exposures, the following will be discussed: a) carbon monoxide poisoning; b) mercury poisoning; c) lead poisoning; and d) ionizing radiation.

**A. Carbon monoxide poisoning**

Almost all fuels consist of hydrocarbons (gasoline, oils, "jet fuel," "natural gas," propane, and so on). The main products of fuel combustion are: a) energy (heat and light); b) CO2; and c) water vapor. However, depending on the purity or contamination of the fuel, variable levels of carbon monoxide (CO) are also produced.

A "bon fire" poses little risk of CO poisoning because the relatively small quantities of CO are diluted by the atmosphere. However, when CO is produced in an enclosed space, such as in a house, CO poisoning kills more than a thousand Americans per year. Most cases of CO poisoning have the following sources: a) house or other structural fires; b) furnaces; c) fireplaces; d) space heaters; e) wood-burning stoves; or f) automobiles "idling" in garages. As many as two-thirds of fatal cases of CO poisoning occur in suicides.

Carbon monoxide is, like most gases, invisible. And it has no odor. It is easily "breathed-in" without knowing it. In structural fires, if a fire begins in the basement, CO that rises up into the bedrooms can sicken or kill sleeping residents before any flames from the fire reach the upper floor or floors.

Carbon monoxide leads to failure of arterial blood to deliver adequate oxygen to cells throughout the body. This can lead to cell injury and death that appears first and foremost in the myocardium, leading to myocardial infarctions, and to the brain. The reason for the inadequate delivery of oxygen is that when CO is "breathed-in" it competes with oxygen to be carried in the blood by the red cells.

The electrochemical attraction of CO for the iron moiety in hemoglobin is more than 200 times greater than the attraction for oxygen. Thus, CO "outcompetes" O2, and hemoglobin carries more CO and less O2. This is also true because the CO binding to the first of hemoglobin's four binding sites, changes the shape of hemoglobin so that it is not a good "deliverer-unloader" of O2 by the other three binding sites.

Also, the CO that is delivered throughout the body is an actual poison to the mitochondria of all cells. Thus, even the small amount of O2 that "makes it to" a body cell, cannot be used to produce ATP. Low O2 in the blood and poor utilization of O2 by body cells leads to lactic acidosis, which further complicates CO poisoning.

It is easy to suspect CO poisoning in patients rescued from a fire, but others may be missed if they had lesser exposures in the absence of a major fire. In those exposed via a poorly-vented wood burning stove the symptoms may not be severe and can be nonspecific—if their exposure was not severe or prolonged. These nonspecific symptoms include: a) headache; b) dizziness; c) malaise and weakness; d) nausea; e) difficulty concentrating; and d) shortness of breath.

Severe complications include seizures, lethargy, or coma, as well as the delayed appearance of neurological and psychiatric manifestations in survivors, for example, after 20 days. Cardiac complications include myocardial ischemia, infarction, and arrhythmias. Finally, lactic acidosis occurs. More than 1000 people in the US die of CO poisoning per year.

**B. Mercury poisoning**

Mercury in any form is toxic. The three main forms are: a) elemental mercury (only mercury); b) inorganic mercury (mercury cation with an anion, a mercury salt); and c) organic mercury (mercury that is covalently bonded to a hydrocarbon). Elemental mercury is found in traditional thermometers and other measuring devices, as well as fluorescent bulbs. Exposures to elemental mercury occur most often in those with occupational exposures, including recycling of mercury containing products.

Mercury salts (inorganic mercury) were used in the past as a component of oral medications. Also, they are sometimes used today in cosmetics and remedies from other countries. However, occupational exposure is the major means of inorganic mercury poisoning.

Organic mercury can be ingested by eating contaminated fish. Many cases of organic mercury toxicity were first reported in Minamata Japan around the time of the Second World War. Mercury was used in a chemical plant with the mercury waste discarded into Minamata Bay. This was absorbed by algae, which in turn, was eaten by small fish and other small marine species. These organisms have very little ability to excrete ingested mercury. Thus, the levels accumulate. Then bigger fish eat the smaller fish, and organic mercury levels rise higher and higher.

Fish from Minamata Bay was a major source of food for those living nearby. Humans also have no effective mechanism to excrete mercury. Many Minamata residents developed severe neurological signs and symptoms due to high organic mercury blood and brain levels. Before the condition was found to be due to high levels of methyl mercury, it was simply called Minamata disease.

All forms of mercury are cellular toxins, and methyl mercury is an especially powerful neurotoxin. Common manifestations include: a) paresthesias; b) tremor; c) ataxia; d) deafness; e) visual field loss; and f) cognitive deficits; among others.

**C. Lead poisoning**

Lead is another “heavy metal” that is toxic, especially to neurons. The toxicity of lead has been known for thousands of years. As a result of this knowledge, severe lead poisoning is only infrequently seen in the US, except for occasional occupational exposures. This includes those who work in lead mining, lead processing, plumbing, auto repair, and construction, and especially in those who do demolition and building rehabilitation.

In the past, lead was used in paints (up to the 1970s) which exposed painters and all those who live in older homes. When these paints age, flakes can drop off the walls and onto the floors, where young children may eat them. Also, paint dust, containing lead dust, can be inhaled. In addition, anyone who uses ammunition can be exposed to lead, such as firing range instructors, law enforcement officers, firefighters, and hunters are at risk.

Gasoline contained lead in the past—and it is still used in some countries. Immigrants and refugees often have been exposed to lead. Finally, older water pipes were made of lead, and have contaminated drinking water.

In 2014, city leaders of Flint Michigan sought to lower the cost of purchasing water from Detroit by using local water from the Flint River. Unfortunately, the old pipes to the Flint River were made of lead. From April to October 2014, almost 100,000 residents in Flint were exposed to very high levels of lead.

Lead from the environment can easily enter the body by: a) ingestion from drinking contaminated water; b) ingestion by small children by eating “old paint flakes;” c) ingestion by use of antiquated lead-containing pottery or glassware; d) inhalation of lead fumes and dusts—including leaded gasoline in the past; e) and inhalation or ingestion of lead-contaminated dusts and soils—especially in those living in older structures in lower-income urban areas.

Young children are more susceptible to lead poisoning because the blood-brain barrier is not fully developed, which allows a greater uptake by the central nervous system. Those with very low levels of exposure may be asymptomatic or manifest one or more of the following: a) decreased learning and memory; b) decreased verbal ability, including impaired speech; c) hearing loss; d) early onset symptoms of "attention deficit hyperactivity disorder" (ADHD); and e) lowered IQ, among others. In those with slightly greater exposure to lead, possible manifestations include the following additional symptoms: a) fatigue; b) irritability or drowsiness/lethargy; c) muscle aches; d) paresthesias; and nonspecific gastrointestinal complaints.

Acute exposures of high levels of lead can cause severe multi-system illness, but these cases are primarily seen in accidental occupational exposures. Most cases of lead poisoning are due to chronic exposure of lower levels. Unfortunately, there is no "safe" blood level of lead.

**D. Ionizing radiation**

Energy is easily transferred from one object to another. For example, when a ball is kicked, kinetic energy from the moving leg and foot is directly transferred to the ball. But kinetic energy is only one form of energy—other forms of energy are not directly transmitted, but, rather "conveyed" through liquids or gases. For example, sound is transmitted through (and conveyed by) air on its journey to our ear drums. In other words, this energy requires a "medium" for its movement. On the other hand, radiation is a form of energy that not only can move through various media, but also through "nothingness"—like outer space.

There is no single form of radiation. Some types of radiation consist of electromagnetic waves; others consist of streams of particles. Further, different frequencies of electromagnetic waves and different types of particles carry different quantities of energy. For example, infrared radiation, which carries warmth from the sun, is of relatively low energy. Forms of radiation that convey lower levels of energy are classified as "non-ionizing radiation." In contrast, X-rays have such high levels of energy that they can destroy malignant tumors. These forms of radiation are classified as ionizing radiation.

What does "ionizing" mean? It means that this radiation has "so much energy" when it interacts with ("strikes") tissues such that electrons are "knocked out" of their orbits. The "ripping away" of molecular electrons from their positively charged nuclei dramatically destabilizes these molecules, setting into motion a host of chemical reactions. These reactions ultimately lead to tissue inflammation, destruction, and genetic mutations.

On the other hand, ionizing radiation can be used to treat some cancers. This is often called "radiation therapy." One of the fundamentals in this modality of treatment is to avoid "hitting" nearby healthy tissues. Thus, "shielding" techniques are employed; however, some normal tissue is always affected. In contrast to the beneficial effects of ionizing radiation on tumors, when normal tissue is hit, there are many serious consequences. These include: a) inflammation and scarring; b) death of normal cells; c) mutations in the DNA of cells that are not killed.
When a mutation of DNA does not kill a cell, it is sometimes called a "sub-lethal" mutation. Because these mutations are "sub-lethal," the cell "continues on" but carrying the mutation or mutations. As these cells undergo normal replication, additional mutations can occur, sometimes sufficient to transform the cells into cancer. While it is not common, it is a great irony that ionizing radiation that cures a patient of cancer, can, by affecting healthy tissues, contribute to the later development of a second cancer.

In summary, while the deadly properties of ionizing radiation can be "used" or "exploited" to treat existing cancers, ionizing radiation exposure is an important cause of cancer—especially in the much larger number of people who are not being exposed intentionally to high dose radiation as part of radiation therapy.

How individuals are exposed to radiation

How are "regular" people exposed to ionizing radiation? First, living on earth exposes us to low levels of radiation. This is called natural background radiation. We are "hit" by radiation from the sun and space. These electromagnetic waves are called "cosmic" radiation. We are also "hit" by electromagnetic waves and particles from the radioactive decay of isotopes in the earth's crust, such as radon.

"Breathing in" radon gas leads to exposure of not only electromagnetic waves ("X-rays" and "gamma rays"), but also particulate forms of radiation, such as alpha and beta particles. Alpha particles are actually the nuclei of helium atoms; that is, 2 protons and 2 neutrons—without the normal 2 electrons. Alpha particles have a +2 charge. Beta particles are actually electrons, which have a -1 charge.

Thus, all of us are exposed to some ionizing radiation just by living on the earth. The quantity of natural background radiation is about 0.3 mSv per year. What is a Sv? Sv is the abbreviation of for the unit, Sievert. This is the unit of the "effective dose" of ionizing radiation that a person absorbs. Further, an mSv is 1/1000 of a Sievert.

In addition to background radiation, almost all Americans are exposed to radiation via diagnostic imaging. The average estimate of exposure from these "studies" is 2-3 mSv per year. This quantity is 6-9 times higher than the natural background radiation. Some do not get any imaging in a year or many years. But there are others who require ten or more imaging studies in a single year.

Older versus new forms of imaging and their radiation exposures

The first imaging was done using X-rays. In general, "regular" X-ray imaging exposes patients to relatively low levels of radiation. But after the 1970s—due to advances in "radiology" technology—other forms of imaging were developed. These new forms of imaging were both better and worse than the "old X-rays." "Computed tomography" was the name given for what are referred to now as "CT scans" or just "CTs". Sometimes, the older "CAT scan" is still spoken.

CTs provide amazing details that are totally invisible on "old X-rays," which are sometimes referred to as "radiographs" or "plain films" or simply, "X-rays." CTs have improved diagnosis immensely, diagnosed cancers earlier, and saved many lives. However, whereas the routine "chest X-ray" is 0.12 mSv; an abdominal chest CT is 8mSv, and CT angiography (CTA) to diagnose or rule out a pulmonary embolus is 15 mSv.

Since some patients require repeated CTs, they can be exposed to very high doses, and the concern is that diagnostic X-rays may be causing or contributing to the development of some cancers. What is the risk of cancer from medical imaging? This is difficult to know for many reasons. First, the doses received from medical imaging—even if many CTs are done—is much lower than the doses received in radiation therapy.

For example, in the treatment of lung cancer, the dose of radiation—broken up into several sessions—is about 60 Gy. Gy stands for Gray, which is a unit used to express the "energy" or "magnitude" of radiation. 1 Gy = 1 Sv for the most common types of radiation therapy. Thus, if we assume that a patient treated for lung cancer absorbs 60 Sv—that is dramatically greater than the radiation absorbed by a CT of the lung (8 mSv), or even ten CTs (80 mSv).

Say a person receives 10 lung CTs over a few years. This would equal an overall effective dose absorbed of 80 mSv. The key questions is this—how close is this magnitude of radiation absorbed in several diagnostic CTs compared to that absorbed by a patient being treated for lung cancer?

Converting Sv to mSv, the following is obtained: 60 Sv * 1000 mSv/1 Sv = 60,000 mSv. Thus, even in someone who was given ten CTs, the absorbed dose would be only 80 mSv compared to 60,000 mSv. Further calculations reveal that the person who received 10 CTs —a great number—still received only about 1/1000 of the radiation dose of the patient getting radiation therapy.

The complex relationship between radiation exposure and cancer

We know that the high doses used in radiation therapy can—in a small minority—cause the later development of a new cancer. How do we know this? The best evidence for radiation causing cancer was not determined by any planned experiment. This would be unethical and illegal. However, an "accidental" study of the effects of high dose radiation was done by investigating those who were subjected to huge doses of radiation by the nuclear bombs of World War II detonated at Hiroshima and Nagasaki.

Many who lived near the epicenter of detonation died instantly. But there were many survivors who lived some distance from the epicenter. In a study conducted over more than 60 years, it was possible to compare estimated doses absorbed (based on distances from the blasts) and the later occurrence of cancer.

This provided incontrovertible evidence that high doses of radiation can cause cancer. Interestingly, many of those exposed to very high doses did not get cancer--for reasons that will be discussed below. On the other hand, many did develop cancer far more frequently than those in Japan living far from the explosions. Even more important, the higher the dose of radiation absorbed, the greater was the occurrence of cancer.

But is this data relevant to the risk of developing cancer from far lower levels of exposure—that is, via medical imaging? What we do know is that when the level of exposure is very high, the cancer rates are high. When the level of exposure is moderately high, the cancer rates are moderately high. When the level of exposure is modestly high, the cancer rates are relatively low. Thus, at these levels, the greater the exposure, the greater the incidence of cancer. In fact, there is a linear relationship.

We do not know if the very low levels of radiation exposure provided by imaging follows the linear relationship we see at higher and ever higher levels of radiation. However—for reasons beyond the scope of this discussion--the consensus of relevant experts is that the relationship is probably linear even at the medical imaging levels. Using this assumption, the following estimates have been made concerning cancer risk at doses in the range of imaging. In a person who receives an exposure of 10 mSv (about as much as a single CT), the risk of developing cancer is about 1 in 1000, overall, not a frequent occurrence.

However, radiation exposure is especially hazardous in children and adolescents. Also, different tissues react differently to radiation. Some of the most radiation-sensitive tissues include: a) embryonal (that is, during pregnancy); b) blood; c) bone marrow; d) lymph tissue; e) testes; and f) ovaries. Less sensitive tissues include: a) skin; b) eyes; c) organs of the digestive tract; d) uterus; and e) bladder. The least sensitive tissues include the central nervous system and muscles. Some of this variability of risk in different tissues has to do with whether cell replication is common (very sensitive tissues) or not (least sensitive tissues).

In any individual, radiation exposure may or may not result in cancer. Why is this? Because no two people are exactly alike. Among other variables, individuals vary in: a) genetics; b) immune system function; c) DNA repair processes; d) age; e) sex; and f) expo-sure to other carcinogens and various medications such as immunosuppressive medications or previous chemotherapy for cancer.

These factors contribute to the apparent randomness in which a low dose radiation (like 10 mSv) will cause cancer in only 1 of 1000 exposed people. And we cannot identify which individual of the 1000 will be the unlucky one. The likelihood of developing cancer from a certain dose of radiation—expressed as only a probability--is referred to as the stochastic effect of radiation exposure.

What is important in medical imaging is to always keep in mind that while the risk of cancer is low—it is NOT zero. Thus, exposure to ionizing radiation via imaging needs to be minimized. CT scans are responsible for about half of the radiation exposure related to imaging. Another fourth is due to “nuclear medicine” imaging. In these studies, radioactive molecules are infused into the patient’s bloodstream. These “tracers” that can be “seen” by “detectors” are called “radionuclides.”

Acute radiation poisoning

Turning from the risks of low dose radiation, what happens to people exposed to massive amounts of ionizing radiation, such as in occupational accidents at nuclear reactors, such as in the Chernobyl reactor “melt-down.” These exposures are usually lethal, and the illness that results in called “acute radiation poisoning.”

Why is high dose radiation so damaging? By “ionizing” several molecules in cells, new chemicals are produced that have had one or more electrons “knocked out.” Thus, these chemicals have “unpaired” electrons. And molecules with unpaired electrons “can think” of nothing other than “hooking up” with other molecules to repair their ‘lonely’ unpaired electrons.” These molecules are like hungry sharks attacking innocent schools of fish.
Thus, ionizing radiation “sets into motion” these “so-called” free radicals, that react with and damage and destroy lipids of cell membranes, essential cellular enzymes and proteins, and DNA itself. At high doses, massive cell death predominates. At lower doses, DNA mutations can occur, which can persist and lead to cancer, as discussed earlier.

How much radiation does it take to cause radiation sickness? Recall that 10 mSv was the effective exposure of one CT scan. A whole-body dose of 1 Gy (~ 1 Sv) will cause radiation sickness. A dose of 4.5 Gy—given to 100 people will kill 50 of them in the ensuing days or weeks. A dose of 10 Gy—given to 100 people will kill all of them.

Thinking about the exposure of one CT (10 mSv), it would take 100 CT scans done one-after-another to result in 1 Gy (~ 1 Sv). Acute radiation sickness has three phases: a) prodromal phase (0-2 days after exposure); b) latent phase (2-20 days); and c) manifest phase (21-60 days). The prodromal phase includes: a) gastrointestinal symptoms; b) headache and dizziness; and c) fatigue and lethargy.

During the latent phase, prodromal symptoms improve and can completely remit. During this period, however, bone marrow cells and lining epithelial cells are dying. When these cells perish, pancytopenia ensues with anemia, thrombocytopenia, and leukopenia. This results in massive hemorrhage and sepsis. The source of sepsis is often the gastrointestinal tract as its barrier function has been destroyed. Finally, there is cerebral edema and alterations of mental status. There is no effective treatment for acute radiation poisoning.

# Alumni News

**Jessica Porter, PA-C**, ’21, currently working in orthopedic surgery and in the emergency department in Milford, MI and is getting married on September 8, 2023.

**Brook Ray, PA-C**, ’21, working in Psychiatry in Westerville, OH and recently had a baby girl, Edith on April 11, 2023.

**Reilly Candow**, PA-C, ’21, (Pictured below) working in Family Medicine with the Baptist Medical Group in Pensacola, FL. and added a little girl, Reagan, to the family in April, 2023. They are moving to their next station in San Diego, CA s

**First Clinical Positions Accepted by Class of 2023**

**Alex Ancona, PA-C**
Mid-Ohio Emergency Services, Columbus, Ohio

**Katie Boehmer, PA-C**
Mid-Ohio Emergency Services, Columbus, Ohio

**Haily Cisneros, PA-C**
Emergency Physicians Professional Association of Minnesota

**Joseph Delatore, PA-C**
Emergency Medicine at Ashtabula County Medical Center

**Haley Guggenheim, PA-C**
Pediatrics, Upper Arlington Office, Nationwide Children's Hospital

**Kate Kinley, PA-C**
Surgical and Medical Oncology Fellowship, The James Cancer Hospital, Ohio State University

**Andrew Kraly, PA-C**
Surgical Residency, Johns Hopkins Medical Center, Baltimore

**Meredith Mayfield, PA-C**
Emergency Medicine, Elkhart General Hospital, Indiana

**Natalie Molnar, PA-C**
Family Medicine, Colorado Springs Family Practice, Colorado

**Miranda Moore, PA-C**
Primary Care, OhioHealth Primary Care, Hilliard, OH

**Nicholas Musico, PA-C**
Neurosurgery, OhioHealth, Grant Medical Center, Columbus, OH

**Thomas Nasr, PA-C**
Hospital Medicine Fellowship, Mayo Clinic Medical Center, Arizona

**Molly Quinn, PA-C**
Surgical Oncology Fellowship, The James Cancer Hospital, Ohio State University

**Kaylee Ramsey, PA-C**
Ocular Oncology, Ohio State Wexner Center

**Jennifer Vu, PA-C**
Dermatology, Veterans Administration Medical Center, Chillicothe, OH

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