QUEEN'S PULSE

SPRING 2021

QUEEN'S ELECTROPHYSIOLOGY

We Keep Your Heart In Rhythm

Queen's Electrophysiology Team:
From left to right: Kyleen Lopez (PPA), Michelle Tusa (RN), Sara Hamele (APRN), Beverly Uclusin (RN), Tiffnie Li (Coordinator), David Singh (MD, Chief of Cardiology and Electrophysiology), Kailie Wong (APRN), Tate Kusatsu (APRN), Caitlin Belle Farinas (PPA), Alamelu Ramamurthi (MD, Electrophysiology), Gautham Kanagaraj (MD, Electrophysiology)

QUEEN'S HEART INSTITUTE

THE QUEEN'S HEALTH SYSTEMS

Message from Director, Center for Heart Rhythm Disorders

I am proud to share some of the highlights from our electrophysiology (EP) program in this edition of Pulse. I am delighted to be working with two new outstanding electrophysiologists, Dr. Gautham Kanagaraj and Dr. Alamelu Ramamurthi. We have significantly expanded our team so that we can meet the increasing demand for EP services around the State. Our EP team provides cutting-edge disease management using the latest technology to ensure the delivery of the best care for our patients with heart rhythm disorders. Over the next year we have several ambitious initiatives including: the establishment of a lead management program that will include lead extraction, the establishment of a comprehensive atrial fibrillation clinic, the opening of two state-of-the-art EP labs, and expansion of EP services to other areas in the State. Please reach out to us if we can be of any help to you and your patients.

A Brief History of Atrial Fibrillation

Few diseases have impacted the field of cardiology more than Atrial Fibrillation. Also known as "AF," it has transformed the field of cardiology and lifted the field of electrophysiology from relative obscurity to one of the fasted growing fields in modern medicine. Several decades ago, only a few drugs were available for the treatment of AF. Today, there are a dizzying array of treatment options for AF including, lifestyle modification, drug therapy, oral anticoagulation, catheter ablation, surgical ablation, occlusion of the left atrial appendage, cardioversion, neuromodulation, pacemaker implantation, and more. A literature search for the term "Atrial Fibrillation" between the years 1960-1970 revealed 1,886 indexed publications. That same search between the years 2010-2020 revealed 53,145 indexed publications. Today there are scientific journals and meetings solely devoted to the management of AF. It has emerged as one of the most common and most challenging diseases in modern medicine.

AF is far and away the most common heart rhythm disorder in adults and is largely related to aging. An increasing elderly population is expected to impact AF prevalence. In the United States alone, it is expected that 12 million people will have AF by the year 2030 [1]. The world-wide prevalence of AF is estimated to be 33 million[2]. The financial burden of AF also has continued to rise. One study suggests that in the United States, the direct and indirect healthcare costs due to AF may be as high as 26 billion dollars annually. [3]

The British cardiologist Sir Thomas Lewis is credited with one of the earliest descriptions of AF in modern medicine. His lecture on "Auricular Fibrillation" at the University College Hospital in London was published in the British Medical Journal in January of 1912[4]. His remarkable powers of observation and the written word are evident in his description of AF. "I refer to an affection of the heartbeat which is now spoken of as auricular fibrillation...The confused and wavy impulses which hurry from interspace to interspace; the swollen legs and belly... the utter disorder of rhythm, the hopeless jumbling of strong pulsations with quick runs of almost imperceptible beats, the changing length of intervening pauses are all characteristics." [4] Here Lewis describes many features of AF that can be discerned by physical exam: his reference to "the swollen legs and belly" describes a patient with heart failure, a common sequelae of the disease. His reference to the changing length of pauses, is the hallmark of AF – by simply feeling a patient's pulse, a clinician will observe an irregular and chaotic rhythm.

The human heart beats approximately 100,000 times each day and 3 billion times over the course of an 80-year life span. Although a human heart rate can vary depending on an individual's state (e.g. sleeping versus exercise) the average heart rate ranges between

60-80 beats per minute. One of the cornerstones of cardiovascular physiology is electro-mechanical coupling. That is, electrical events lead to mechanical events. Every heartbeat is the result of a complex array of ion exchanges through a vast network of voltage gated channels located in muscle fibers throughout the heart. The final stage in this sequence involves the activation of two key proteins: actin and myosin. Configurational changes in the myosin protein leads to binding with the actin protein resulting in shortening of the muscle filament and ultimately, cardiac muscle contraction.

The normal electrical sequence in the heart is highly regulated. A collection of specialized cells located at the top of the right atrium comprise the sinus node. The sinus node is the master pacemaker of the heart. Along with the autonomic nervous system, it is responsible for the regulation of heart rate. Every time the sinus node fires, the impulse travels toward the center of the heart. Along the way, atrial muscle tissue is activated resulting in atrial contraction. Toward the bottom of the right atrium, the electrical impulse arrives at a structure known as the AV node. Here, the signal is transmitted to the ventricles via a network of specialized conduction fibers known as the HIS-Purkinje system. The result is ventricular contraction. Under ordinary circumstances, there is a 1:1 relationship between atrial contraction and ventricular contraction – that is, every sinus discharge results in one atrial contraction followed by one ventricular contraction.

In AF, this physiology shifts dramatically. In the heart, “Fibrillation” refers extremely rapid contraction of a chamber. In AF, the atria are beating 400-600 times per minute. When a chamber fibrillates, it is literally quivering and the contractile apparatus of the involved chamber is compromised. In AF, the atria function as passive conduits for blood flow as no substantial atrial contraction occurs. Importantly, the AV node protects the ventricles from transmitting every impulse arising in the atria. If there was as 1:1 relationship between the atria and ventricles during AF (i.e. the ventricles are beating at 400-600 times per minute) the result would be ventricular fibrillation, otherwise known as death. The AV node is endowed with an important property known as “decremental conduction.” The more the AV node is bombarded by impulses, the fewer impulses are transmitted to the ventricles. In the case of AF, the AV node may only transmit every third or fourth impulse to the ventricles. Thus, while a patient’s heart rate may be rapid in AF, it will rarely exceed rates greater than 200 beats per minute.

Although our understanding of AF and its treatments have expanded significantly, the cornerstones of AF management have remained remarkably consistent: stroke prevention, rate control or rhythm control. It has long been established that AF is associated with higher stroke rates. Patients with atrial fibrillation are known to have sluggish blood flow or “stasis,” one of the key risk factors for blood clot formation. Anatomically, an embryological remnant of the left atrium known as the left atrial appendage (LAA) provides an ideal environment for clots to form. Its narrow neck and deep muscular ridges can promote severe stasis creating an ideal environment for clot formation. For this reason, clots that form in the heart due to AF frequently do so in the LAA[5]. When a blood clot that has formed in the heart is ejected and occludes a blood vessel supplying a region of the brain, oxygen rich blood can no longer reach the affected area leading to cell death. Interruption of blood supply to the brain due to a blood clot arising from the heart is referred to as a “cardioembolic stroke.”

For several decades, the cornerstone of stroke prevention was the medication Warfarin. It emerged in the marketplace as a rat poison in 1948. As it was tasteless and odorless it could be mixed with bait food. Rodents would continually return to the bait and

Continued on next page.

A Brief History of Atrial Fibrillation

Continued from previous page.

Dr. Singh performing atrial fibrillation ablation on a patient in the EP Lab.

feed until lethal doses of the drug was consumed. The result was death from catastrophic hemorrhage. In 1951, a member of the armed services attempted suicide with warfarin, but fully recovered after being treated with Vitamin K in the hospital. Research to evaluate its potential as a therapeutic anticoagulant ensued. Warfarin inhibits an enzyme responsible for activating Vitamin K throughout the body. Because the hepatic synthesis of clotting factors II VII IX and X are dependent on Vitamin K, warfarin effectively inhibits systemic blood clot formation. Until recently, it was the only medication that was shown to reduce mortality among patients with AF[6]. In addition, it has been shown to reduce stroke risk in patients with AF by 64%[6]. For these reasons, warfarin remains an important drug in the management of AF.

Despite its efficacy, warfarin has many limitations. It is known to have a narrow therapeutic index. Too little warfarin is associated with reduced efficacy, too much warfarin is associated with bleeding. For this reason, patients taking warfarin have to have their blood levels checked frequently which in turn can negatively impact their quality of life. Perhaps the most feared consequence from warfarin is intracranial hemorrhage (ICH), a complication that often leads to death or severe disability. The annual risk for ICH among patient taking warfarin ranges from 0.3%-3.7%[7]. It is also associated with a plethora of interactions. Foods with high levels of Vitamin K can reduce its efficacy. In addition, there are many drugs that can raise levels of warfarin resulting in an elevated bleeding risk.

For these reasons, the search for an alternative oral anticoagulant was the subject of intensive research for many years. In 2010, the Food and Drug Administration approved the direct thrombin inhibitor dabigatran for the reduction of stroke in patients with non-valvular AF. This development changed the landscape of AF management in a monumental way. Dabigatran was shown to be equally efficacious with respect to stroke reduction as compared to warfarin[8]. Perhaps more importantly, it was associated with fewer bleeding risks including intracranial hemorrhage, fewer drug interactions, and did not require blood monitoring.

Several other oral anticoagulants emerged onto the

market thereafter including rivaroxaban (approved 2011), apixaban (approved 2012), and edoxaban (approved 2015)[9-11]. In each trial, these anticoagulants were found to be at least as effective as warfarin in reducing stroke and safer with respect to intracranial bleeding. As a result, clinicians have many more choices when it comes to prescribing medications to reduce stroke risk in patients with AF.

The range of symptoms in patients with AF is somewhat perplexing. Many patients are completely asymptomatic and may only learn about their condition from an Apple watch or a screening ECG. On the other hand, some patients find the condition so disabling that they are unable to perform their activities of daily living. For patients who are symptomatic, management is primarily aimed at improving or eliminating their symptoms. To this end, clinicians have employed two main strategies: rate control, where the chief aim is to limit the exaggerated heart rates often encountered in AF and rhythm control where the goal is to maintain sinus rhythm. Historically, drugs that target AV nodal conduction have been used in a rate control strategy. These include beta blockers, calcium channel blockers and digoxin.

There are times when AF patients cannot be adequately rate controlled despite drug therapy. It was one of these patients that changed the field of electrophysiology permanently. Mr. Paul Anderson was a retired oil refinery worker who developed pulmonary edema due to AF with rapid rates resistant to drug therapy[12]. On April 9, 1981 Dr. Melvin Scheinman and his team at the University of California, San Francisco used a catheter to deliver DC energy to the AV node[12]. This resulted in AV block such that impulses from the atria could not transmitted to the ventricles. It also ushered in a new era of electrophysiology where catheter ablation became the standard of care to treat a wide array of electrical disorders. Today, we still perform AV nodal ablations for patients with AF who cannot be adequately rate controlled with drugs.

EP Nurse Beverly Uclusin provides expert clinical coordination.

Rhythm control is a strategy that centers around maintaining sinus rhythm. For many decades this was only achievable through the use of anti-arrhythmic medications. Early on, drugs like quinidine and procainamide were employed. These medications were marginally effective and associated with significant side effects. Later on, drugs like flecainide and propafenone were employed but were not without side effects as well including life-threatening arrhythmias[13]. Amiodarone is perhaps the most effective anti-arrhythmic medication with respect to AF but is associated with multiple side effects including severe pulmonary toxicity. Despite these limitations, anti-arrhythmic medications can be a highly effective rhythm control tool for appropriately selected patients.

If the first catheter ablation in humans was a landmark moment in medicine, another key development occurred when a group from Bordeaux, France published a seminal paper in the New England Journal of Medicine[14]. In it they noted that the pulmonary veins were frequently the source of rapid firing resulting in atrial fibrillation. In other words, the pulmonary veins could be implicated as the trigger for AF in many patients. Moreover, they documented that ablation around the pulmonary veins resulted in electrical isolation such that impulses in the veins could not reach the surrounding atria. The result was a significant reduction in AF among the patient who underwent ablation.

Continued on next page.

A Brief History of Atrial Fibrillation

Continued from previous page.

This single observation transformed the field of electrophysiology from as a relatively obscure field comprised of "nerdy" practitioners obsessed with electrograms to a mainstream sub-discipline of cardiology. Ablation for AF has become the most common procedure practiced by modern day electrophysiologists. Indeed, AF management has come to dominate most modern-day electrophysiology practices. The demand to treat patients with symptomatic AF has paralleled the high prevalence of the disease. While ablation is far from perfect (indeed we constantly remind our patients that "it is not a cure") it is our most effective tool and can be a life-changing intervention. It is not uncommon to have a patient come to the office following ablation and thank us for "giving them their life back". There is also a large body of data on AF ablation that supports its use in appropriately selected patients[15]. Whereas there is conflicting data on whether AF ablation has a significant impact on mortality, it has been shown to significantly reduce the burden of AF in patients who undergo the procedure.

Despite all of the advances in our understanding and treatment of AF there is still more to learn. While the atria require a trigger to initiate fibrillatory activity, we still are trying to grasp why this activity is sustained in patients with AF. Not all atria are capable of sustaining AF. A healthy heart should not fibrillate even in the presence of a trigger. Usually, patients in the early stages of AF have short episodes of AF that are discrete and contained. As the disease progresses, there are a myriad of changes that occur in the atria that provide the substrate for longer durations of AF. This progression eventually results in a state where the AF is sustained and does not stop on its own.

How to modify this "substrate" is currently the subject of intense research. If the mechanisms that contribute to AF substrate could be elucidated, they could potentially serve as targets for ablation.

EP Nurse Practitioner Sara Hamele reviews pacemaker tracing with patient.

Many candidate mechanisms have been proposed as potential targets. Terms like "rotors," "spatio-temporal dispersion," and "complex fractionated atrial electrograms" are just some of the terms used to describe various mechanisms underlying AF as well as targets for ablation[16, 17]. It is beyond the scope of this discussion to describe these mechanisms in detail, but they have all provided considerable insight with respect to our understanding of AF.

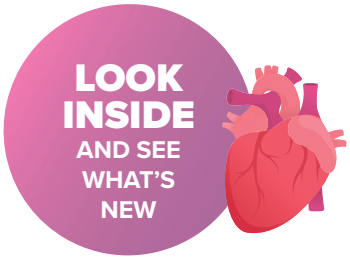
In the future, more emphasis will be placed on the prevention of AF and addressing modifiable risk factors associated with AF and AF progression. We now know that addressing issues like obesity, sleep apnea, and alcohol consumption are vital for the prevention and management of AF[18, 19]. As our understanding of AF continues to evolve, so too will the technology that guides our interventions. We are in an unprecedented era of growth with respect to electrophysiology and our ability to treat heart rhythm disorders like AF. It is plausible that in the near future we may make the most important discovery of all – a cure for AF.

1. Colilla S, Crow A, Petkun W, Singer DE, Simon T and Liu X. Estimates of current and future incidence and prevalence of atrial fibrillation in the U.S. adult population. Am J Cardiol. 2013;112:1142-7.
2. Chugh SS, Havmoeller R, Narayanan K, Singh D, Rienstra M, Benjamin EJ, Gillum RF, Kim YH, McAnulty JH, Jr., Zheng ZJ, Forouzanfar MH, Naghavi M, Mensah GA, Ezzati M and Murray CJ. Worldwide epidemiology of atrial fibrillation: a Global Burden of Disease 2010 Study. Circulation. 2014;129:837-47.
3. Kim MH, Johnston SS, Chu BC, Dalal MR and Schulman KL. Estimation of total incremental health care costs in patients with atrial fibrillation in the United States. Circ Cardiovasc Qual Outcomes. 2011;4:313-20.
4. Lewis T. A Lecture ON THE EVIDENCES OF AURICULAR FIBRILLATION, TREATED HISTORICALLY: Delivered at University College Hospital. Br Med J. 1912;1:57-60.
5. Blackshear JL and Odell JA. Appendage obliteration to reduce stroke in cardiac surgical patients with atrial fibrillation. Ann Thorac Surg. 1996;61:755-9.
6. Hart RG, Pearce LA and Aguilar MI. Meta-analysis: antithrombotic therapy to prevent stroke in patients who have nonvalvular atrial fibrillation. Ann Intern Med. 2007;146:857-67.
7. Steiner T, Rosand J and Diringer M. Intracerebral hemorrhage associated with oral anticoagulant therapy: current practices and unresolved questions. Stroke. 2006; 37:256-62.
8. Connolly SJ, Ezekowitz MD, Yusuf S, Eikelboom J, Oldgren J, Parekh A, Pogue J, Reilly PA, Themeles E, Varrone J, Wang S, Alings M, Xavier D, Zhu J, Diaz R, Lewis BS, Darius H, Diener HC, Joyner CD, Wallentin L, Committee R-LS and Investigators. Dabigatran versus warfarin in patients with atrial fibrillation. N Engl J Med. 2009;361:1139-51.
9. Giugliano RP, Ruff CT, Braunwald E, Murphy SA, Wiviott SD, Halperin JL, Waldo AL, Ezekowitz MD, Weitz JI, Spinar J, Ruzyllo W, Ruda M, Koretsune Y, Betcher J, Shi M, Grip LT, Patel SP, Patel I, Hanyok JJ, Mercuri M, Antman EM and Investigators EA-T. Edoxaban versus warfarin in patients with atrial fibrillation. N Engl J Med. 2013;369:2093-104.
10. Patel MR, Mahaffey KW, Garg J, Pan G, Singer DE, Hacke W, Breithardt G, Halperin JL, Hankey GJ, Piccini JP, Becker RC, Nessel CC, Paolini JF, Berkowitz SD, Fox KA, Califf RM and Investigators RA. Rivaroxaban versus warfarin in nonvalvular atrial fibrillation. N Engl J Med. 2011;365:883-91.
11. Granger CB, Alexander JH, McMurray JJ, Lopes RD, Hylek EM, Hanna M, Al-Khalidi HR, Ansell J, Atar D, Avezum A, Bahit MC, Diaz R, Easton JD, Ezekowitz JA, Flaker G, Garcia D, Geraldes M, Gersh BJ, Golitsyn S, Goto S, Hermosillo AG, Hohnloser SH, Horowitz J, Mohan P, Jansky P, Lewis BS, Lopez-Sendon JL, Pais P, Parkhomenko A, Verheugt FW, Zhu J, Wallentin L, Committees A and Investigators. Apixaban versus warfarin in patients with atrial fibrillation. N Engl J Med. 2011;365:981-92.
12. Scheinman MM. Every journey begins with the first step: Reflections on the first catheter ablation in humans. Heart Rhythm. 2021;18:145.
13. Echt DS, Liebson PR, Mitchell LB, Peters RW, Obias-Manno D, Barker AH, Arensberg D, Baker A, Friedman L, Greene HL and et al. Mortality and morbidity in patients receiving encainide, flecainide, or placebo. The Cardiac Arrhythmia Suppression Trial. N Engl J Med. 1991;324:781-8.
14. Haissaguerre M, Jais P, Shah DC, Takahashi A, Hocini M, Quiniou G, Garrigue S, Le Mouroux A, Le Metayer P and Clementy J. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. N Engl J Med. 1998;339:659-66.
15. Asad ZUA, Yousif A, Khan MS, Al-Khatib SM and Stavrakis S. Catheter Ablation Versus Medical Therapy for Atrial Fibrillation: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. Circ Arrhythm Electrophysiol. 2019;12:e007414.
16. Cheniti G, Vlachos K, Pambrun T, Hooks D, Frontera A, Takigawa M, Bourier F, Kitamura T, Lam A, Martin C, Dumas-Pommier C, Puyo S, Pillois X, Duchateau J, Klotz N, Denis A, Derval N, Jais P, Cochet H, Hocini M, Haissaguerre M and Sacher F. Atrial Fibrillation Mechanisms and Implications for Catheter Ablation. Front Physiol. 2018;9:1458.
17. Seitz J, Bars C, Theodore G, Beurtheret S, Lellouche N, Bremondy M, Ferracci A, Faure J, Penaranda G, Yamazaki M, Avula UM, Curel L, Siame S, Berenfeld O, Pisapia A and Kalifa J. AF Ablation Guided by Spatiotemporal Electrogram Dispersion Without Pulmonary Vein Isolation: A Wholly Patient-Tailored Approach. J Am Coll Cardiol. 2017;69:303-321.
18. Du X, Dong J and Ma C. Is Atrial Fibrillation a Preventable Disease? J Am Coll Cardiol. 2017;69:1968-1982.
19. Chung MK, Eckhardt LL, Chen LY, Ahmed HM, Gopinathannair R, Joglar JA, Noseworthy PA, Pack QR, Sanders P, Trulock KM, American Heart Association E, Arrhythmias C, Exercise CR, Secondary Prevention Committee of the Council on Clinical C, Council on Arteriosclerosis T, Vascular B, Council on C, Stroke N, Council on L and Cardiometabolic H. Lifestyle and Risk Factor Modification for Reduction of Atrial Fibrillation: A Scientific Statement From the American Heart Association. Circulation. 2020;141:e750-e772.

QUEEN'S HEART INSTITUTE

THE QUEEN'S HEALTH SYSTEMS

1301 Punchbowl Street • Honolulu, Hawaii 96813



MEET THE TEAM

QUEEN'S ELECTROPHYSIOLOGY

David Singh MD
Dr. David Singh specializes in cardiac electrophysiology. His clinical interests center on catheter ablation of supraventricular and complex arrhythmias including atrial fibrillation and ventricular tachycardia. Dr. Singh earned his medical degree at Georgetown University School of Medicine, and completed his Internal Medicine residency training at the University of California, San Francisco (UCSF). He continued his training in Cardiovascular Diseases at Cedars Sinai Medical Center in Los Angeles, where he served as Chief Cardiology Fellow. In 2009, he returned to UCSF to undertake training in Electrophysiology. Dr. Singh has been involved in a number of clinical research efforts including the development of novel techniques for better recognition of cardiac arrhythmias, risk factors for sudden cardiac death, and ablation techniques for atrial fibrillation.

Dr. Singh also has helped to spearhead a number international health initiatives, and maintains a passion for working with underserved populations. He is currently part of an electrophysiology team that conducts teaching and electrophysiology procedures in Cambodia. In 2009, he founded the Northern Mariana Islands Cardiology Technical Assistance Project which focused on developing access to cardiology care to this region. As a cardiology fellow, he co-founded, the Los Angeles Free Cardiology Clinic, devoted to providing cardiology care to the individuals lacking adequate access to health care. He has served as Chief of the Department of Cardiovascular Diseases for the Queen's Medical Center since 2019.

Board Certifications: Cardiovascular Disease, Clinical Cardiac Electrophysiology

Alamelu Ramamurthi MD
Dr. Alamelu Ramamurthi was born in India. She grew up in New Delhi and went to medical school in Chennai. Her interest in valvular heart disease brought her to Boston in 2011 for a research fellowship in non-invasive cardiovascular imaging at Tufts Medical Center. After spending 2 years as a research fellow, she completed her internal medicine residency at Tufts Medical Center. Following this, she completed her fellowships in Cardiolvascular Diseases and Electrophysiology at Lahey Clinic in Burlington, MA. She and her husband are avid hikers and nature enthusiasts.

Board Certifications: Internal Medicine, Cardiovascular Disease, Clinical Cardiac Electrophysiology

Gautham Kanagaraj MD
Dr. Gautham Kanagaraj was born in India and raised in Bahrain, New Zealand and California. Dr. Kanagaraj completed his internship and residency in internal medicine at the State University of New York at Stony Brook. Following this, he completed his cardiology fellowship at the University of Hawai'i in Honolulu and his electrophysiology fellowship at Harbor UCLA/Good Samaritan in Los Angeles. His clinical interests include complex ablation (AF, VT) and device management (implantation, extraction). Dr. Kanagaraj married his medical school sweetheart and has two daughters. Outside of cardiac electrophysiology, interests include art, travel, history, economics, surfing, and snowboarding.

Board Certifications: Internal Medicine, Adult Comprehensive Echocardiography, Cardiovascular Disease, Clinical Cardiac Electrophysiology

Kailie Wong APRN
Kailie was born and raised on Kauai and has lived on Oahu since 2013. Kailie completed a Master of Science in Nursing at the University of Hawaii at Manoa in 2016. She began her career in Electrophysiology following graduation. Kailie has played an active role in developing the Queen's AF management program. She is also currently involved in several research and quality improvement projects pertaining to heart rhythm disorders. Kailie has been recognized by Queen's as a compassionate provider who consistently receives high marks from her patients. She enjoys educating patients on how lifestyle modifications can improve their heart health. She enjoys yoga, hiking, and indoor cycling.

Sara Hamele APRN
Sara Hamele has worked as an APRN in electrophysiology since 2012. She earned her Bachelor of Sciences in Exercise Science (kinesiology) from Western Washington University, and Master of Science in Nursing from Pacific Lutheran University. Her clinical interest and expertise includes: cardiac device rhythm management, arrhythmias, and congenital heart disease; she also has experience in general cardiology and interventional cardiology. In her spare time she enjoys hanging out at the beach with her husband and three girls.

Tate Kusatsu APRN
Tate is one of the newest members of our EP team. She was born and raised in Hawaii and graduated from Kalani High School. She completed her graduate program in Nursing in 2017, and her Doctorate in Nursing from the University of Hawaii Manoa, in May 2020. In her spare time, she enjoys reading ECG's and spending time with family.

Beverly Uclusin EP Nurse Coordinator
Beverly Uclusin is the Electrophysiology Program Patient Care Coordinator for the Queen's Heart Physician Practice (QHPP). She was born in the Philippines. She completed a Bachelor of Science in Nursing from Hawaii Pacific University in 2011. Following graduation she joined QHPP full time and worked with staff electrophysiologists to develop a comprehensive Atrial Fibrillation Management program for The Queens Medical Center. She serves as the chief liaison for our AF patient. She is also responsible for the coordination of patient care for complex EP procedures and left atrial appendage occlusion. She enjoys travelling with her husband and 2 boys, hiking, and gardening.

Michelle Tusa EP Nurse Coordinator
Michelle was raised in rural Illinois. She attended the University of Colorado, Boulder where she conducted Alzheimer's research. She graduated with a BS in Anthropology and obtained her Emergency Medical Technician license in Colorado. Michelle later moved to Hawaii and obtained her bachelor's degree in nursing from Hawaii Pacific University. She is a certified progressive critical care RN and after 6 years experience in cardiac critical care at QMC, Michelle joined Queen's Heart Physician Practice in 2020 as an Electrophysiology RN coordinator. In her free time she enjoys spending time with her 2 young children and family, practicing martial arts, and gardening.