POTASSIUM AND SUDDEN CARDIAC DEATH – UPDATE

INTRODUCTION

In this newsletter I had every intention of continuing my review of the literature on low grade, chronic metabolic acidosis and its relationship to potassium deficiency. While I will continue this series of newsletters in the near future, I now feel compelled to present a review on the research on potassium and sudden cardiac death that has been published since I wrote my initial 6-part series, “Some Thoughts on Sudden Cardiac Death” in 2014 - 2015. Why? Certainly, one reason is the continued reports of people, mainly men in their 50’s and 60’s, who are dying due to sudden and unexpected heart attacks. However, the main reason has to do with just one of those men. He was in his 60’s and died suddenly and unexpectedly due to a heart attack within the last two months. However, for me, the story of that man does not end there. He was a long-time friend and professional colleague – someone who I had known for over 30 years. Furthermore, he was one of the most accomplished healers, clinical nutritionists, and academics that I have ever known. I cannot even begin to count the hundreds of people who have benefited from his work as both a clinician and instructor. Nevertheless, even though his accomplishments were many, and given that there were so many more he could have helped, he died way too soon.

What do I know about his heart condition? Actually, very little. I do know that he had suffered from cardiovascular disease for several years before his death. Nevertheless, like my father who died in 2002 of a heart attack despite being told several weeks before his death that his cardiovascular health had stabilized and he had a very optimistic future, this individual, based on the limited information I received, was assumed to be stable and at no risk for imminent death. Therefore, as I asked myself when my father died, I’m asking about this individual, “What was different about the day of his heart attack from all the days before when his outlook was so optimistic?”

In the case of my father I had enough information about the day he died to create what I consider to be a reasonable hypothesis that he died of ventricular tachycardia and cardiac arrest due to a catastrophic and sudden drop in serum potassium caused by a perfect storm of prior poor health, questionable lifestyle choices (He was a lifelong smoker), plus the “straw that broke the camel’s back” - food choices the day he died that led to insulin spikes and resultant, almost instantaneous, increases in intracellular potassium plus equally instantaneous and catastrophic drops in serum potassium (Refeeding syndrome).

Did a combination of suboptimal cardiovascular health plus some lifestyle choices that led to the same sudden and catastrophic changes in potassium status lead to this individual’s untimely fatal heart attack? Hopefully, I will someday have enough information from the family about all that happened 1-2 days before he died to answer this question. However, because I presently lack this information, I do continue to wonder “Did a sudden, catastrophic, and totally unexpected change in potassium status take another victim?”

While I am frustrated that I cannot answer this question for this individual, I take some satisfaction in knowing that more and more research continues to support my contention I made in 2014 that potassium status is a major cause of cardiovascular morbidity and mortality. Furthermore, I feel it is vastly under-appreciated, misunderstood, and/or ignored by all too many practitioners in both the allopathic and alternative realms.

Therefore, in my continuing crusade to bring the large volume of research on the relationship between potassium status and cardiovascular disease, and, most especially, sudden cardiac death, to the attention of both practitioners and the population at large, I would like to now update you on the
research I have discovered on the subject since the publication of my initial newsletter series.

**SERUM POTASSIUM, ARRHYTHMIAS, AND CARDIOVASCULAR DEATH – AN OVERVIEW**

To begin this update, I would like to present highlights from the literature review paper entitled “Association of abnormal serum potassium levels with arrhythmias and cardiovascular mortality: a systematic review and meta-analysis of observational studies” by Hoppe et al (1). The paper begins with an important fact we sometimes forget in the face of high profile media reports of causes of death ranging from drug opiate addiction to suicide to mass shootings:

“Cardiovascular disease (CVD) is the number one cause of death worldwide. In 2012, CVD was responsible for 31% of all global deaths, representing 17.5 million people.”

The next quote I would like to feature summarizes the main reason I feel potassium status is an important risk factor for sudden cardiac death and is often ignored or overlooked:

“Sensitivity towards abnormal serum K+ levels seems to be important for the prognosis of CVD and may be different according to the patient’s history of morbidity. In patients with acute myocardial infarction (AMI), for instance, excessively released catecholamines stimulate an intracellular shift of potassium. This results in potassium depletion which in turn increases the risk of ventricular fibrillation.”

As you may recall from my initial series on potassium and sudden cardiac death, a major premise was that potassium status is often overlooked because, for many, if not most patients at risk for sudden cardiac death, baseline, resting, serum potassium levels are almost always in the generally regarded safe range (4.0 – 4.5 mmol/L). However, what I stated in my initial series, which is confirmed in the quote above as well as by papers I will present shortly, is that any lifestyle stressor can potentially increase stress hormone levels to the point where, in individuals already at high risk for significant cardiac dysfunction, life threatening imbalances between serum and intracellular potassium levels can occur. Why don’t more people experience heart attacks with stress hormone induced imbalances in serum and intracellular potassium? Generally, this imbalance does not last long enough to adversely affect heart health in the average, heart-healthy population. For those, who may be “on the edge” so to speak, though, this seconds-long alteration may be enough to put the heart into a lethal tachycardia.

What else can create sometimes short term but, for certain susceptible patients, lethal imbalances between serum and intracellular potassium? As I mentioned, insulin spikes that can often occur in the refeeding syndrome scenario where the first meal after a long period of not eating is composed of a significant volume of refined carbohydrates. This, as you may recall, is what I theorized about my father.

**Hypokalemia and atrial fibrillation**

The next quote I would like to feature from the Hoppe et al paper (1) discusses the relationship between low serum potassium and the increasing common occurrence of atrial fibrillation. As this quote suggests, hypokalemia is not just related to fibrillation of the ventricles:

“…hypokalemia was associated with an increased risk of supraventricular arrhythmias (defined as atrial flutter and atrial fibrillation)…”

However, unlike the relationship between hypokalemia and ventricular fibrillation, the relationship between hypokalemia and atrial fibrillation rarely leads to fatal outcomes:

“…we presume that hypokalemia-induced arrhythmias, especially atrial arrhythmias, rarely have a fatal outcome in the older general population.”

**Hypokalemia and diuretic use**

As I mentioned in my initial series on potassium and sudden cardiac death, many studies have noted a relationship between increased incidence of hypokalemia and diuretic use. Hoppe et al (1) affirm this relationship:

“…diuretic use was more prevalent in hypokalemia subjects and less prevalent in hyperkalemic subjects when compared to subjects with normokalemic levels. This finding supports the assumption that hypertensive subjects who receive diuretic treatment have a particularly high risk of hypokalemia.”
It is my opinion, given the above, that many individuals on diuretic therapy are at particularly high risk for sudden cardiac death when encountering stress-related or diet/insulin-related sudden drops in serum potassium levels.

Some final recommendations from Hoppe et al

In closing, Hoppe et al (1), point out that the generally accepted serum potassium range may be acceptable for the general population, but the ideal range constricts dramatically with increasing levels of heart dysfunction:

“As recommended by experts with affiliation to the American Heart Association, serum K⁺ levels should be maintained between 3.5 and 5.1 mmol/L in the older general population in order to prevent supraventricular and ventricular arrhythmias, as well as cardiovascular mortality. For patients with hypertension or heart failure a higher cutoff of 4.0 mmol/L has been suggested for hypokalemia. This recommendation is supported by the observed increased CV risk for low serum K⁺ levels in these two aforementioned patient groups sometimes treated with non-potassium-sparing diuretics.”

What about patients experiencing a heart attack? The cutoff is even higher:

“For patients with acute myocardial infarction, an even higher threshold for hypokalemia has been suggested, namely 4.5 mmol/L during or shortly after acute myocardial infarction. As we detected a borderline significant 2-fold increased risk of arrhythmias in acute myocardial infarction patients with low serum K⁺ levels, we agree with this recommendation to target serum K⁺ levels in the high-normal range in these patients.”

Unfortunately, since labs do not report different acceptable serum potassium ranges based on different patient presentations, it has been my experience that many in both the allopathic and alternative medicine community deem the lab range for serum potassium as acceptable for all patients. Since there is a risk that this assumption may lead to increases in the risk for fatal outcomes, it is my hope that, as soon as possible, more practitioners will become aware of what I am reporting.

ANOTHER REASON WHY THE RELATIONSHIP BETWEEN POTASSIUM AND SUDDEN CARDIAC DEATH IS SO UNDER-APPRECIATED

It is my guess that, when questioned, many if not most health care practitioners will state that virtually all heart attacks are a function of ischemia related to poor coronary circulation. Of course, this assumption is not new. As you may recall, the first major theory of heart attack causation several decades ago was the “clogged pipe” theory of poor cardiac circulation where dietary “gunk” such as cholesterol from eggs would build up in the coronary arteries like a clogged drain pipe, causing poor circulation, ischemia, and a heart attack. Even though there may be some practitioners who still subscribe to the idea that this theory of heart attack is the norm, it has been generally concluded by the research community that this virtually never occurs in the average heart attack patient. What does occur, as has been demonstrated by many studies, to create heart attack-inducing ischemia is formation of inflammation induced coronary artery plaques that can rupture. These ruptured plaques then lead to clot formation which can, in turn, precipitate ischemic heart myocardial infarction.

Of course, what both of these theories of heart attack share in common is that heart attack is almost always a function of ischemia caused by poor coronary artery circulation, where the status of electrolytes such as potassium is not a central concern. Does this belief that virtually all heart attacks are a function of poor circulation reflect reality? Urso et al in their paper “Analysis of electrolyte abnormalities and the mechanisms leading to arrhythmias in heart failure. A literature review” (2) suggest that the answer is no. In fact, as you will see in the quote below, about 50% of heart failure cases intimately involve electrolytes such as potassium:

“About 50% of deaths from heart failure (HF) are sudden, presumably referable to arrhythmias. Electrolyte and acid-base abnormalities are a frequent and potentially dangerous complication in HF patients. Their incidence is almost always correlated with the severity of cardiac dysfunction; furthermore leading to arrhythmias. These imbalances are associated with a poor prognosis. The frequency of ventricular ectopic beats and sudden cardiac death
correlate with both plasma and whole body levels of potassium... The early recognition of these alterations and the knowledge of the pathophysiological mechanisms are useful for the management of these HF patients.”

It is my opinion that one reason the connection between potassium and sudden cardiac death has not gained more widespread acceptance in the healthcare community is the false assumption that the vast majority of fatal heart attacks are due to poor coronary circulation. In contrast, as noted in the above quote by Urso et al (2), almost half of all fatal heart attacks are related to arrhythmias that are often a function of suboptimal fluid and electrolyte metabolism.

CURRENT INVESTIGATIONS CONTINUE TO AFFIRM THE RELATIONSHIP BETWEEN POTASSIUM STATUS AND SUDDEN CARDIAC DEATH

In a very recently published study entitled “Long-term potassium monitoring and dynamics in heart failure and risk of mortality” by Nunez et al (3), the following patients with heart dysfunction were evaluated:

“The study sample included 2164 patients (50.4% men) with a total of 16,116 potassium observations. The mean age was 73 ± 11 years. Left ventricular systolic dysfunction was present in 31.2% of the patients...”

The findings of the study are as follows:

“In a large nonselect cohort of patients discharged from an episode of acutely decompensated heart failure, low and high serum potassium levels, as well, measured in a time-varying setting, were associated with higher risk of mortality through a U-shaped trajectory. Likewise when modeling potassium as clinical categories, we found that patients with hypokalemia or hyperkalemia also had a higher risk of mortality.”

More research on hypertension, potassium, and mortality risk

As was mentioned above, the presence of hypertension necessitates a much more constricted optimum serum potassium range to reduce the risk of sudden cardiac death. The recently published study entitled “Short-term mortality risk of serum potassium levels in hypertension: a retrospective analysis of nationwide registry data” by Krogager et al (4) affirms this. In a study of Danish National Registries, 44,799 hypertensive patients aged 30 years or older who had a serum potassium measurement within 90 days from diagnosis between 1995 and 2012, were considered. In the introduction to this paper the authors point out the disturbing relationship between the use of hypertensive medications, potassium disturbances, and survival:

“Many hypertensive agents including diuretics, β-blockers, angiotensin-converting enzyme inhibitors (ACEIs), and angiotensin receptor blockers (ARBs) can cause potassium disturbances and influence survival.”

With the above in mind, data analysis revealed hypertensive patients on medication were at increased risk of mortality when they demonstrated various serum potassium ranges generally regarded as having little or no concern:

“This study analysed the short-time mortality risk in relation to different potassium intervals in hypertensive patients administered various classes of blood-pressure-lowering drugs. The major finding was that even mild deviations with the normal potassium range (3.5 – 5.0 mmol/L) were associated with increased mortality. Although it was expected that hypo- and hyperkalemia would be associated with increased risk of death, it was unexpected that three normal potassium levels (K: 3.5-3.7, 3.8-4.0, and 4.8-5.0 mmol/L) were also associated with a significantly increased mortality in hypertensive patients.”

With the above in mind, Krogager et al (4) concluded the following concerning optimal serum potassium levels in hypertensive patients:

“All these analyses indicated that serum potassium >4.0 and <4.7 is optimal in hypertensive patients.”

Stroke, mortality, and potassium status

With all that I have stated thus far, would it be correct to assume that potassium status represents a significant risk factor for stroke and stroke-related
death? The recently published study “Prognostic impact of mild hypokalemia in terms of death and stroke in the general population – A prospective population study” by Mattsson et al (5) answers this question in the affirmative. In a study of 5916 participants aged 48-76 years in Denmark, the following was noted:

“Potassium <3.7 mmol/L was associated with increased risk of stroke but not mortality.”

“Potassium <3.4 mmol/L was associated with increased risk of mortality and stroke.”

With the above in mind, the authors concluded:

“In a general population mild hypokalemia is associated with increased stroke risk and, to a lesser degree, increased mortality risk.”

**DOES A PATIENT WITH OPTIMAL FASTING SERUM POTASSIUM HAVE NO RISK FOR HYPOKALEMIC SUDDEN CARDIAC DEATH?**

It is my guess that many who have read not only the above, but my previous series on potassium and sudden cardiac death, may have checked blood chemistries on themselves, loved ones, and/or patients. It is also my guess that, upon consideration, many of these blood chemistries showed serum potassium levels were within optimal ranges (Within the 4.1 – 4.6 mmol/L range). Is it safe to conclude, based on these fasting potassium levels, that there is no risk of hypokalemic sudden cardiac death? As I stated above and will now demonstrate, the answer is most decidedly no. Why? Most patients will have blood drawn for fasting blood chemistries early in the morning before the typical stresses of the day are encountered. Why does this matter? As will be demonstrated in the following review of two papers, significant stressors can rapidly and drastically reduce serum potassium levels. Is this dangerous from a cardiac standpoint for the average healthy individual? Probably not, since, in a generally healthy individual, stress-induced hypokalemia will not last long enough to have a significant impact on a healthy heart. However, in an individual with pre-existing suboptimal heart function, even a period as short as a few seconds of significant hypokalemia could precipitate a state of ventricular fibrillation and a possible life-threatening outcome.

Before beginning my review of these papers, I would like to briefly discuss two key points that these papers emphasize. First, the impact of significant stressors on potassium status and resultant cardiac dysfunction is intricately interlinked with magnesium status. More specifically, hypokalemia induced by significant stressors cannot be optimally addressed without concurrent correction of suboptimal magnesium status. Second, the most obvious disturbance of cardiac function that occurs with stress-induced hypokalemia is a finding on electrocardiogram called “prolonged QTc interval.” As you will see, this prolonged QTc interval that can occur due to hypokalemia is directly linked with both atrial and ventricular fibrillation and, therefore, is an important and easy to identify risk factor for sudden cardiac death in certain high-risk patients.

The first paper I would like to review is “Cation interdependency in acute stressor states” by Khan et al (6). The first quote I would like to feature discusses the cellular pumps that regulate the amount of potassium that is in the serum and how much is in the intracellular environment. In turn, these pumps play a critical role in determining intracellular/extracellular potassium ratios that are so important in controlling the presence of optimal or suboptimal heart contractions:

“Na/K ATPase, a membrane-bound, energy-dependent pump whose activity contributes to the regulation of intracellular K⁺, has an obligatory dependence on Mg²⁺. A large number of these pumps are present in skeletal muscle, where they are regulated by catecholamines.”

What is the impact of increased catecholamine production that occurs significant stressor states? The authors continue:

“Increments in plasma epinephrine and norepinephrine that accompany acute stressor states activate these pumps leading to marked K⁺ uptake by muscle and the rapid appearance of hypokalemia. Reductions in myocardial K⁺ are accompanied by delayed repolarization and prolongation of the QTc interval of the electrocardiogram – a pathophysiologic scenario that favors an increased propensity for supra- and ventricular arrhythmias.”

What is the magnitude of the impact of catecholamine induced hypokalemia in terms of
actual serum potassium measurements? Khan et al (6) discuss the results of an experiment on normal human volunteers:

“Catecholamines are responsible for acute hypokalemia that appears with acute stressor states. This response was reported in normal human volunteers given intravenous epinephrine, in which a prompt and marked fall in serum K⁺ from 4.0 to 3.2 mEq/L…occurred…”

Therefore, even in healthy individuals, an acute stressor can rapidly take serum potassium levels from the optimal to the unacceptable. Of course, as mentioned above, this decrease, which will usually be short-term in healthy individuals is probably of little or no clinical consequence. However, what about the at-risk cardiac patient who may be taking one or more medications? Could stressors have an additive effect with potassium depletion effect of certain drugs, thus increasing the risk of adverse cardiac outcomes? Khan et al (6) note:

“The underlying K⁺ balance that exists before bodily injury is a determinant of the severity of the ensuing hypokalemia that occurs during an acute stressor state. This was again demonstrated in normal volunteers who were given a thiazide diuretic before the epinephrine infusion. The diuretic-induced loss of K⁺ predisposed to marked hypokalemia in response to the catecholamine.”

Is this significant for the patient with pre-existing CVD? The authors note:

“Patients with arterial hypertension or congestive heart failure, who are receiving long-term thiazide or loop diuretic treatment, respectively, may develop marginal K⁺ and Mg²⁺ reserves. These limited reserves are further compromised by the hyperadrenergic state (eg, motor vehicle accident) that may quickly lead to marked hypokalemia and hypomagnesemia with QTc prolongation and a propensity for arrhythmias.”

The next quote points out that many other medications can demonstrate a similar additive effect:

“Albuterol, a short-acting β₂ agonist and catecholamine with bronchodilator properties, also predisposes to hypokalemia and hypomagnesemia in normal volunteers, and even more so in those receiving a diuretic. Chronic excessive use of such a β₂-receptor agonist may lead to marked hypokalemia and a greater propensity for arrhythmias. Clenbuterol, a longer acting β₂ receptor agonist, has anabolic properties that contribute to an increase in skeletal muscle mass. Its inappropriate use by body builders can lead to rapid-onset hypokalemia, hypomagnesemia and QTc prolongation associated with cardiac arrhythmias. Drug-induced prolongation of myocardial repolarization and QTc interval occurs in association with certain antibiotics, antidepressants, and antipsychotics.”

Still another medication that can have a similar impact is digoxin:

“Digoxin, a Na/K ATPase inhibitor, accentuates the dyshomeostasis of intracellular K⁺ and Mg²⁺ predisposing to QTc prolongation and arrhythmias.”

The final two quotes I would like to feature from the Khan et al (6) paper bring two extremely important points. First:

“The correction of impaired K⁺ balance and hypokalemia will prove difficult unless Mg²⁺ is first replaced.”

This quote makes a point that I cannot emphasize enough in terms of potassium and magnesium supplementation where, all too often, clinicians will supplement one without the other. Both must be supplemented concurrently to attain optimal clinical results.

The last quote I would to feature is the following:

“The QTc interval and its abnormal prolongation (>440 milliseconds) identify a deficiency of myocardial K⁺ and Mg²⁺.”

Many of your patients with CVD will often have routine electrocardiograms as part of their regular physical examinations. If you are concerned about a need for potassium and magnesium supplementation in these patients or whether the supplementation you are providing is effective, ask the patient to provide you a copy of these electrocardiograms. As noted in the above quote, the presence of a prolonged QTc interval will provide some answers for these concerns.
The second paper I would like to feature is “K+ and Mg2+ dyshomeostasis in acute hyperadrenergic stressor states” by Nayyar et al (7). The first quote from this paper I would like to present provides more information about the relationship between stressors and potassium and magnesium status:

“K+ and Mg2+ equilibrium is threatened by acute stressor states and is promoted by their inextricable association with neurohormonal activation, where effector hormones of the hypothalamic-pituitary-adrenal axis and the adrenergic nervous system in particular are elaborated in concert. The resultant hormonal onslaught, a homeostatic stressor response, provokes iterations in K+ and Mg2+ concentrations. This includes translocation of these cations from the vascular space into the intracellular compartment of soft tissues (e.g., muscle) to culminate in the concordant appearance of hypokalemia and hypomagnesemia.”

The next quote makes it clear that not only acute diseases create this scenario. In fact, illnesses similar to what we see in our chronically ill patients can lead to clinically significant hypokalemic and hypomagnesemic states:

“Illnesses that are akin to bodily injury and invoke neurohormonal activation include sepsis, pneumonia, pancreatitis and diabetic ketoacidosis. These stressor-induced iterations in K+ and Mg2+ can be contemporaneous in time and concordant in direction.”

The last quote I would like to feature from the Nayyar et al (7) paper gives some goals for serum levels when supplementing with potassium and magnesium:

“Maintenance of serum K+ and Mg2+ at physiologically relevant concentrations of > 4.0 mg/DL and 2.0 mg/dL (the 4 and 2 rule) is suggested and must not rely on the hospital laboratory’s reference range. Daily monitoring of QTc interval and its normalization provides an indirect measure of intracellular K+ and Mg2+ and valuable adjunct to the surveillance of serum levels.”

**DIET, SERUM POTASSIUM AND SUDDEN CARDIAC DEATH**

As noted above, stressful situations are not the only concern when attempting to prevent sudden, possibly life-threatening drops in serum potassium in certain high risk individuals. As I discussed extensively in my initial potassium and sudden cardiac death series, diet can have a major impact in creating sudden drops in serum potassium. The principal way is through the refeeding syndrome scenario where ingesting refined carbohydrates after a significant period of eating little or no food can lead to insulin spikes. These insulin spikes, in turn, can lead to rapid increases in intracellular potassium at the expense of serum potassium. This extracellular/intracellular imbalance can then, as has been well documented in certain high risk patients with a history of cardiac dysfunction, precipitate atrial and/or ventricular arrhythmias and increased risk of sudden death.

Another dietary issue that can have potentially profound implications on potassium status, as I discussed, in the initial potassium and sudden cardiac death series, is caffeine intake. Since I wrote that series, though, I found another paper that discusses the relationship between caffeine and serum potassium levels, which I would like to share with you now. What makes this paper truly unique and interesting is that it does not discuss the situation in generalities and population trends. Rather it provides actual data on specific amounts of caffeine and their specific impact on serum potassium.

In “Effect of caffeine on serum and urinary electrolytes” by Geethavani et al (8) 30 healthy male volunteers aged 19-22 years were evaluated. Caffeine was provided in the following manner:

“All the participants were given 100 mg of caffeine in gelantine capsules. This was repeated at 30 minute intervals for a total period of 120 minutes which accounts to a total amount of 400 mg caffeine.”

To give perspective, a typical cup of coffee contains about 100 mg of caffeine. As we all know, it is not unusual for patients to consume four cups of coffee in a two hour period, which would yield the same amount and rate of caffeine intake as in the Geethavani et al (8) study. What were the results in terms of serum potassium?

“Serum potassium was found to decrease significantly from 4.4 ± 0.2 in the control period to 3.9 ± 0.2 at 400 mg of caffeine intake.”
As has been documented in the many studies I have discussed both in this newsletter and the previous potassium and sudden cardiac death newsletter series, for patients already suffering from suboptimal cardiovascular function, a drop from a generally regarded safe level of 4.4 mmol/L to 3.9 mmol/L in a period of two hours due to coffee intake could have major, potentially life-threatening implications. 

SOME FINAL THOUGHTS

Since my father died of sudden ventricular tachycardia in 2002 despite the claims of his cardiologist that his heart disease was stabilized with little to no risk of imminent death, I have tried to do everything I can to learn about the biochemistry and physiology that lead to his death and share the information I have found, hopefully, prevent others from sharing the same fate. What I have discovered and continue to discover is that, while, many factors came together to form the perfect storm of his death, it seems clear to me that a crisis in potassium and magnesium metabolism was not only part of the picture but, if it had been addressed even minimally before the fatal heart attack, he may have had significantly more time to spend with me, the rest of our family, and all who cared for him.

Could this information have prevented the unexpected and untimely fate of my good friend and professional colleague discussed at the beginning of this newsletter? Unfortunately, I may never know. For, while potassium and magnesium imbalances are major unrecognized and under-appreciated causes of sudden cardiac death, there are certainly many other causes that may have come into play. Nevertheless, as I hope I have demonstrated, the research community, as evidenced by published papers such as those reviewed in this update, is certainly of the opinion that potassium and magnesium imbalances deserve more attention when issues revolving sudden cardiac death are considered.

Is progress occurring in terms of getting the word out? Based on your response to my initial potassium and sudden cardiac death series, I feel confident in saying that inroads are being made. However, I feel I must, sadly, end this commentary with some sobering perspective. I just received a paper published on June 27, 2018 by Al-Khatib and Stevenson entitled “Management of ventricular arrhythmias and sudden cardiac death risk associated with cardiac channelopathies” (9). It is part of the JAMA Cardiology Clinical Guidelines Synopsis. While it is certainly an outstanding paper highlighting important pharmaceutical and crises care interventions for the ventricular arrhythmia patient at risk for sudden cardiac death, not one word, literally, was mentioned about the need to address electrolyte status of any form, including evaluation of potassium and magnesium levels.

Needless to say, while progress is being made, we still have a long way to go. However, as long as papers are being published on the subject and I continue to hear about the tragedy of sudden and unexpected cardiac death due to ventricular fibrillation and cardiac arrest, I will continue to write and talk about the subject no matter how few are actually paying attention.

REFERENCES

9. Al-Khatib SM & Stevenson WG. Management of ventricular arrhythmias and sudden cardiac death risk associated with cardiac channelopathies. JAMA Cardiology. 2018;Published online ahead of print June 27, 2018.