SOME THOUGHTS ON SUDDEN CARDIAC DEATH – PART IV

INTRODUCTION

As you have seen, the central theme in this series has been the under appreciation of sudden depletions of potassium, usually in the serum, and catastrophic illness, specifically heart attack. However, I ended part III with another equally important discussion. If potassium depletion is having such a large impact on acute cardiovascular disorders, why is it so under recognized? My answer, as you saw, was that most in the healthcare community do not fully understand the physiology of potassium metabolism and how readily serum potassium levels can drop as the result of environmental stressors that are so ubiquitous and such an integral part of the everyday lifestyles of so many Americans that they are rarely recognized as stressors that can have a catastrophic impact.

Probably the best example of this phenomenon is what I discussed at the end of part III where extreme dietary habits that either involve extreme food deprivation or, more often, diets that involve rapid ingestion of concentrated carbohydrates after a moderate or prolonged period of minimal or no food ingestion. Often referred to as the “refeeding syndrome,” these patterns of food ingestion create extreme ebbs and flows of insulin production that result in equally extreme and often tragic ebbs and flows of serum potassium levels.

Now I would like to continue with this discussion by pointing out more common dietary patterns that, unbeknownst to many, can create sometimes dangerous alterations in serum potassium metabolism.

CAFFEINE-CONTAINING BEVERAGES: POTENT CONTRIBUTORS TO HEALTH ISSUES RELATED TO LOW SERUM POTASSIUM

In part one of this series I featured the following quote from a paper by Kjeldsen (1):

“In cardiovascular patients, hypokalemia is often caused by nonpotassium-sparing diuretics, insufficient potassium intake and a shift of potassium into stores by increased potassium uptake stimulated by catecholamines, beta-adrenoreceptor agonists and insulin.”

In particular, please note that, just like insulin, “beta-adrenoreceptor agonists” can also cause a clinically significant shift of potassium from extracellular (serum) to intracellular stores. As you will see from several papers that follow, there is another dietary substance that is just as ubiquitous as carbohydrate and just as powerful as carbohydrate in creating illness as a result of intracellular shifts of potassium. What is this substance? It is the beta-adrenoreceptor agonist, caffeine.

What follows are reviews of papers that examine the potassium related health effects of excessive caffeine intake from two major sources – tea and cola.

Tea

The first paper I would like to discuss is a case report about a woman who drank excessive amounts of oolong tea. In “Hypokalemia with syncope caused by habitual drinking of oolong tea” by Aizaki et al (2) the authors first provide a general overview of the patient:

“A 61-year-old woman developed hypokalemia, atrioventricular block and ventricular tachycardia with syncope after habitual drinking of 2 to 3 liters of oolong tea per day. She had been suffering from rheumatoid arthritis and Sjogren’s syndrome and serum albumin was decreased (2.9 g/dl).”

What follows is a brief history and clinical findings upon admission:

“What from 3 years earlier, she began to drink oolong tea. From the beginning of the summer of 1996, she drank 2 to 3 liters of oolong tea daily. In September 1996, she readily felt fatigue in normal life activities. No gastro-intestinal tract symptoms were noted, with no diarrhea and vomiting. On
September 7, 1996, at 3 AM, she suddenly complained of chest discomfort. Syncope occurred, and profuse sweating, rigidity of the extremities and respiratory arrest were recognized by her family. She was immediately brought to the Emergency and Critical Care Center of Kitasato University Hospital. Following admission, her level of consciousness recovered to normal, but she complained of continuous chest pain."

Key laboratory findings were respiratory alkalosis, hypoalbuminaemia, elevated AST, high fasting glucose (225 mg/dl), and, as you might expect, low serum potassium (2.7 mEq/l). Furthermore, as you also might expect, electrocardiogram demonstrated several abnormalities.

Fortunately, treatment, which only involved discontinuation of the oolong tea, resulted in almost complete resolution of symptoms and laboratory findings in about three days. Of course, even though this potentially catastrophic scenario had a very benign ending, it is still important for us to know exactly what caused the patient to end up in such a dire situation. What follows is an excellent description by Aizaki et al (2) of precisely what happened.

First, consider the following quote about the properties of oolong tea and the caffeine it contains:

“Oolong tea usually contains approximately 20 mg/dl caffeine. Thus, if 2 to 3 liters of oolong tea is ingested daily, 400 to 600 mg of caffeine is ingested daily as well. Caffeine is trimethylxanthine (1-, 3-, 7-trimethylxanthine) and one of its metabolites is theophylline. Theophylline stimulates the central nervous system for respiratory regulation, and can thus induce hyperventilation and respiratory alkalosis. Accordingly, the hypokalemia in this case appeared to be due to respiratory alkalosis. Theophylline does not increase urine secretion of potassium, but causes potassium shifting from the circulation into the intracellular space. Hall et al found that 12 to 15 patients with theophylline intoxication developed both respiratory alkalosis and hypokalemia.”

As you can see from this quote, theophylline, which is a caffeine metabolite, can have exactly the same impact on potassium status as excessive carbohydrate intake on an empty stomach, even though the impact of caffeine is not insulin mediated. The next two quotes from Aizaki et al (2) provide data on the amount of caffeine required to adversely impact potassium metabolism and, to provide perspective, how much caffeine the typical American ingests daily:

“Passmore et al emphasized that oral intake of 180 to 360 mg caffeine can provoke serious hypokalemia.”

In addition:

“Twenty to thirty percent of adult Americans intake more than 500 to 600 milligrams per day of caffeine and surprisingly 10% take over 1 g.”

Next the authors discuss the metabolism of caffeine:

“The normal elimination half-life of caffeine in adults is reported to be 2.5-8 hours. Therefore, our patient’s serum potassium level was normalized within 2 days after abstinence from oolong tea. The serum protein binding caffeine is albumin. Accordingly, in patients with hypoalbuminemia, caffeine is apt to induce hypokalemia.”

What patients are most likely to demonstrate low albumin? As noted by Lee et al (3) in their recently published paper “Serum albumin and prealbumin in calorically-restricted, non-diseased individuals: a systematic review,” those who are malnourished and those who demonstrate increased inflammation (Elevated CRP, etc.). Therefore, similar to refeeding syndrome, patients most susceptible to caffeine-induced hypokalemia are the elderly and those ingesting unusual, fad diets.

The next quote I would like to feature from the Aizaki et al (2) paper discusses the direct cardiac impact of caffeine:

“In principle, caffeine itself is arrhythmogenic. Caffeine is able to lower the threshold for ventricular fibrillation by 30-40%. Caffeine shortens the refractory period and increases the duration and amplitude of the myocardial action potential. Accordingly, caffeine can provoke atrial tachycardia, ventricular tachycardia, and bigeminy and atrioventricular block.”

Therefore, it appears that caffeine can affect heart function in two ways:

• Through direct impact on heart function
• Through alteration of potassium metabolism

Aizaki et al (2) conclude their paper by pointing out that, especially in patients who may exhibit low albumin levels (i.e., elderly, malnourished patients), ingestion of significant levels of caffeine-containing...
beverages can have a major adverse impact on both potassium and metabolism and cardiac function:

“In summary, several so-called ‘healthy’ drinks contain caffeine. They may be dangerous for individuals whose serum albumin is decreased and who drink great quantities of such drinks. These individuals may develop hypokalemia, atrioventricular block and/or ventricular tachycardia with syncope.”

Colas

The next paper I am going to review is a case report that particularly interested me because of the increased reports of sudden, unexplained paralysis that I have been seeing on the nightly news the last few months. In “Hypokalaemic paralysis induced by large amounts of cola consumption” by Lee et al (4), the authors discuss a 52-year-old man admitted to the emergency room:

“A 52-year-old man was admitted to our emergency room with progressive paralysis in both extremities. He had started medication for alcoholism and major depression 30 months previously. He was intermittently treated in an adjacent psychiatric hospital for resolution of his psychiatric problems. Whenever he was admitted, he drank large amounts of cola without the permission of the hospital staff.”

The authors continue:

“As you might expect, his serum potassium was quite low (2.3 mmol/l).

With the above in mind, Lee et al (4) assumed the following:

“There were no explanations for the hypokalemia, other than excessive consumption of cola.”

With this assumption in mind, the authors instituted the following treatment with the following results:

“After cola was withdrawn and only 170 mmol of potassium chloride was replaced via intravenous site, his serum potassium level was 4.2 mmol/l and paralysis was completely improved 48 h later.”

Lee et al (4) conclude their paper with sobering data about the impact of caffeine on potassium metabolism and a warning:

“It is known that an oral intake of only 180-360 mg caffeine can provoke serious hypokalaemia. Cola includes 130 mg caffeine per litre: this patient thus consumed approximately 1000 mg caffeine. There are several potential mechanisms by which caffeine may induce hypokalemia:

(i) Redistribution of potassium into cells
(ii) Caffeine induces catecholamine release, probably by means of adenosine antagonism.
(iii) Caffeine may increase renal excretion of potassium.
(iv) Caffeine-induced hyperventilation with respiratory alkalosis.

Caffeine-induced hypokalaemia may well be an overlooked cause. When faced with unexplained hypokalemia, patients should be asked to provide a thorough history of caffeine intake, such as cola, coffee, cocoa and oriental tea.”

A similar case to the one described above was reported by Appel and Myles (5) in “Caffeine-induced hypokalemic paralysis in pregnancy.” The patient experience was briefly described in the abstract:

“A 24-year-old women...at 33 weeks’ gestation presented with muscular paralysis and hypokalemia secondary to drinking 6 to 7 L of cola per day with little other oral intake. After potassium replacement and stopping caffeine ingestion, the symptoms resolved quickly.”

Based on the above the authors concluded:

“The physiologic changes of pregnancy might potentiate the effect of caffeine on serum potassium concentration.”

Recalling the statements above about the potentiating effect of low serum albumin on the creation of hypokalemia by significant caffeine intake, it should be noted that, according to Maher...
et al (6), serum albumin levels decrease during pregnancy.

The next case report I would like to review is similar to the two discussed above in that it also involves disturbances in muscle function. Fortunately, though, this case only involves muscle myopathy, not paralysis. In “Hypokalemic myopathy due to excessive consumption of cola” Yaguchi and Yaguchi (7) present the following case report:

“A 30-year-old man was admitted for hypokalemic myopathy due to excessive consumption of cola and oolong tea. He had a 4-year history of manic-depressive illness and polydipsia, and had consumed 1.5-2.5 L of cola (0.12 mg/mL caffeine content) and 1.5-3.0 L of oolong tea (0.2 mg/mL caffeine content) daily for the previous 3 months.”

Pertinent laboratory findings were the following:

“Laboratory findings included serum potassium 2.3 mmol/dL and creatine kinase 12.285 U/L. Two months before admission, his potassium level had been 4.3 mmol/dL.”

Concerning outcome, the authors pointed out:

“Steady clinical recovery was noted following treatment that included cessation of soft drink consumption and potassium supplementation.”

The case report was concluded with the following hypothesis:

“Caffeine intoxication is thought to have played a major role in cola-induced hypokalemia.”

**Is hypokalemia induced by excessive cola ingestion more than just an issue of too much caffeine?**

The last paper I would like to discuss concerning the relationship between colas and hypokalemia is a review paper that suggests, unlike the other papers reviewed above, the central issue is more than just a function of caffeine intake. In “Cola-induced hypokalemia: pathophysiological mechanisms and clinical implications” by Tsimihodimos et al (8) the first quote I would like to feature reiterates what was stated above that excessive intake of colas poses a major threat to optimal potassium metabolism and health issues that relate to potassium status:

“…several lines of evidence suggest that the chronic consumption of large amounts of cola-based soft drinks may result in severe symptomatic hypokalemia.”

Interestingly, the authors suggest several reasons for this phenomenon that include caffeine and much more. One reason other than caffeine content is the significant content of sweetener that can often cause diarrhea:

“Glucose-induced hypokalemia: cola soft drinks may contain large amounts of glucose (up to 11 g of sugar per dl in regular colas). Thus the excessive consumption of these preparations may lead to osmotic diuresis and inappropriate urinary potassium loses.”

Another reason suggested by Tsimihodimos et al (8) relates to the “refeeding syndrome” scenario I discuss previously in this series:

“In addition, the large glycaemic load may result in hyperinsulinaemia which, in turn, may lead to potassium redistribution into cells.”

The next aspect of colas discussed by the authors that may adversely affect potassium status is one with which we are all familiar, high fructose corn syrup:

“…when fructose in ingested alone or in excess (as in the case of high fructose corn syrup consumption), it is absorbed in limited quantities by a mechanism of facilitated transport. Therefore, large amounts of unabsorbed fructose pass into the colon where they may result in the development of osmotic diarrhoea. Indeed, previously published reports have underscored the role of high fructose corn syrup in the development of chronic osmotic diarrhoea and potassium depletion.”

The caffeine connection is next discussed in detail with an elaborate description of the biochemistry and physiology:

“Caffeine-induced hypokalemia: cola soft drinks contain sufficient amounts of caffeine ranging from 95 to 160 mg/l. It is well known that the consumption of moderate quantities of caffeine (180-360 mg) may result in severe hypokalemia because of potassium redistribution into cells, increased renal excretion of potassium or a combination of these mechanisms. The inhibition of phosphodiesterase and the resulting elevation in the levels of intracellular cyclic adenosine monophosphate, along with the
caffeine-induced respiratory alkalosis and β-adrenergic stimulation possibly represent the main mechanisms that underlie the shift of potassium into the cells. On the other hand, the caffeine-mediated increase in diuresis may underlie the renal wasting of potassium, whereas the caffeine-induced increase in renin release may also play a contributory role.”

Of course, where this series began was with a genuine concern about the link between potassium status and catastrophic illness such as sudden cardiac death. Do Tsimihodimos et al (8) feel that excessive cola consumption should be included in a discussion of this magnitude? Absolutely!!

“Although cola discontinuation and potassium supplementation usually lead to an uneventful recovery in most of the cases, the cola-induced chronic hypokalemia clearly predisposes to the development of potentially fatal complications such as cardiac arrhythmias. In addition, chronic hypokalaemia may be a cause of increased morbidity because of fatigue, loss of productivity and muscular symptoms that vary from mild weakness to profound paralysis.”

**LICORICE AND HYPOKALEMIA**

Up to now, in this installment of this series, dietary substances that can adversely affect potassium status all had one attribute in common – they tended to stimulate catecholamine-mediated stress receptors. Could foods that affect cortisol metabolism also have an adverse effect on potassium metabolism? As you will see in the discussion that follows, the answer is yes. In “Licorice-induced severe hypokalemia with recurrent torsade de pointes” by Panduranga and Al-Rawahi (9) the authors discuss a cause of severe hypokalemia that was caused by excessive licorice ingestion and its adverse impact on cortisol metabolism.

The initial case presentation is the following:

“A 38-year-old obese woman, mother of nine children, non-diabetic, hypertensive, presented with a regional hospital with recurrent syncope. She was postpartum 6 months, on methyldopa for hypertension. She was not on any other medication and denied any vomiting or diarrhea. On presentation, she was hypotensive with monitor showing polymorphic ventricular tachycardia (PVT). She was defibrillated promptly. However she developed recurrent PVT with hypotension and syncope necessitating 40 DC shocks over 2 hour’s period. She was electively intubated and transferred to our institute on inotropic support. Her echocardiogram showed global hypokinesia with an ejection fraction of 25%.”

As you might expect, her serum potassium was very low – 2.4 mmol/L.

Her pertinent history is as follows:

“…for the past 2 months, she was ingesting licorice root tea thrice daily to reduce weight. She was hiding this history so that her husband and family do not know about her licorice use. This history clinched the diagnosis to licorice-induced hypokalemia with secondary PVT…”

Treatment and the results of that treatment are as follows:

“She was treated with magnesium sulphate and potassium infusions followed by oral replacement bringing the potassium levels to normal within 12-24 hours resulting in disappearance of PVT. During this period of replacement she needed another 8 DC shocks for her PVT.”

One month later the authors reported the following:

“At 1 month follow-up and off licorice use, she was well within normal potassium level (4.2 mmol/L), a normal QTc interval…and 24-hour Holter study. Her blood pressure was well controlled with Atenolol.”

In the next quote Panduranga and Al-Rawahi (9) discuss the specifics of how excessive licorice use caused the situation described above:

“Licorice contains glycyrrhetinic acid which inhibits renal 11 beta-hydroxysteroid dehydrogenase type 2, which normally inactivates cortisol. Inhibition of this enzyme by licorice leads to excess cortisol which stimulates mineralocorticoid receptors resulting in a state of ‘pseudohyperaldosteronism’ causing hypertension, renal potassium loss, high bicarbonate and metabolic alkalosis. Thus, cortisol effects mimic aldosterone excess (apparent mineralocorticoid excess syndrome), although aldosterone remains low or normal during licorice overdose. In addition, licorice is known to reduce body fat by inhibiting 11 beta-hydroxysteroid dehydrogenase type 1 at the level of fat cells, thus increasing fat oxidation. Studies
have indicated that licorice consumption should be no more than 10-30 g licorice per day or no more than half a cup of licorice tea a day.”

As you can see from the above quote, there existed a legitimate rationale for ingesting licorice tea to promote weight loss. Unfortunately, as happens all too often when individuals not thoroughly familiar with the therapeutic use of herbs employ herbal remedies to address a single issue, the fact that herbs can have sometimes several ways of impacting human health other than the one being addressed, can lead to unintended but still tragic outcomes. In this case, the tragic outcome was a potentially fatal cardiac dysfunction related to massive decreases in serum potassium.

**SOME FINAL THOUGHTS ON DIETARY SUBSTANCES THAT CAN AFFECT POTASSIUM STATUS AND CARDIAC FUNCTION**

As you may recall, this series began with my desire to understand why my father had a fatal ventricular arrhythmia on a particular night in 2002 even though he had been deemed in “good health” by his doctors based on a lack of symptoms and an “ideal” lipid panel. Knowing that his last meal which occurred just 3-4 hours before he died may have very likely included coffee, a pasta dish, a sugar-laden dessert, and a soft drink plus, to top it off, was probably ingested after a 5-6 hour period of no food ingestion, is it really a totally unpredictable, coincidental anomaly, as so many have suggested, that he died that night? While that meal, certainly in and of itself, would not be considered lethal even by the most idealistic of nutritionists, was it “the straw that broke the camel’s back” so to speak? Furthermore, given it is very likely that a meal such as this is ingested thousands of times a day by millions of people in this country, for how many people will this meal, for the reasons I have given, be their last meal? I wonder.

**DISEASE STATES AND HYPOKALEMIA**

Now I would like to take this discussion on potassium and catastrophic illness in a different direction and consider the clinical conditions that are most commonly associated with hypokalemia. The first that I would like to discuss is first for a very good reason. It has become truly an epidemic in today’s American society – diabetes mellitus.

**Diabetes mellitus and hypokalemia**

The first paper I would like to discuss in this category considers potassium from a big picture perspective – the fact that potassium is just one of many key electrolytes that can become imbalanced in diabetics. In “Diabetes mellitus and electrolyte disorders” by Liamis et al (10) the authors first discuss how common electrolyte disorders are in clinical practice:

“Electrolyte disorders are common in clinical practice. They are mainly encountered in hospital populations occurring in a broad spectrum of patients (from asymptomatic to critically ill) and being associated with increased morbidity and mortality. The disturbances of electrolyte homeostasis are also frequently observed in community subjects. Community-acquired electrolyte disorders, even chronic and mild, are related to poor prognosis. Electrolyte disorders are usually multifactorial in nature. Various pathophysiological factors, such as nutritional status, gastrointestinal absorption capacity, co-existent acid-base abnormalities, pharmacological agents, other comorbid diseases (mainly renal disease) or acute illness, alone or in combination, play a key role.

Diabetes mellitus (DM) is included among the diseases with increased frequency of electrolyte abnormalities given that the aforementioned factors (especially impaired renal function, malabsorption syndromes acid-base disorders and multidrug regimens) are often present in diabetics.”

Next I would like to focus on the statements made by Liamis et al (10) that are specific to hypokalemia. The first quote discusses redistribution related to insulin that has been a major focus of this series because it can be caused by insulin administration per se or dietary patterns that cause sudden increased insulin production:

“The causes of hypokalemia in diabetics include: (1) redistribution of potassium $[K^+]$ from the extracellular to the intracellular fluid compartment (shift hypokalemia due to insulin administration);…”

The next two causes relate to gut and renal function:

“(2) gastrointestinal loss of $K^+$ due to malabsorption syndromes (diabetic-induced motility disorders, bacterial overgrowth, chronic diarrheal states); and
renal loss of K+ (due to osmotic diuresis and/or coexistent hypomagnesemia).”

Please note again the reference to magnesium. As I have suggested previously, there is an intimate relationship between potassium and magnesium metabolism that I will discuss in more detail later in this series. For now, consider the following quote by Liamis et al (10) that points out the fact that low magnesium can cause hypokalemia:

“Hypomagnesemia can cause hypokalemia possibly because a low intracellular magnesium [Mg2+] concentration activates the renal outer medullary K+ channel to secrete more K+.”

The next quote points out the specific impact of exogenous insulin administration:

“Exogenous insulin can induce mild hypokalemia because it promotes the entry of K+ into skeletal muscles and hepatic cells by increasing the activity of the Na+-K+-ATPase pump. The increased secretion of epinephrine due to insulin-induced hypoglycemia may also play a contributory role. The major setting in which insulin administration leads to hypokalemia is during the treatment of severe hyperglycemia. The majority of patients with diabetic ketoacidosis (DKA) and hyperglycemia hyperosmolar syndrome (HHS) are markedly K+-depleted.”

Therefore, when dealing with uncontrolled diabetics, particularly those who are demonstrating ketone bodies in the urine, assume that hypokalemia exists and consider potassium supplementation.

The following quote shows how exogenous insulin administration can induce a refeeding syndrome type scenario:

“Insulin therapy lowers K+ concentration driving K+ into cells (both directly and indirectly by reversing hyperglycemia). Therefore, insulin therapy may cause severe hypokalemia, particularly in patients with normal or low serum K+ concentrations at presentation. Insulin administration in patients with massive K+ deficits who are hypokalemic prior to therapy should be delayed until the serum K+ is above 3.3 mEq/L to avoid possible arrhythmias, cardiac arrest and respiratory muscle weakness.”

The final quote from Liamis et al (10) that I would like to feature on the subject of hypokalemia and diabetes mellitus points out the sobering phenomenon where hypokalemia and diabetes mellitus can both contribute to the other, creating a vicious circle scenario:

“Hypokalemia is associated with impaired insulin secretion and decreased peripheral glucose utilization resulting in carbohydrate intolerance and hyperglycemia. This is particularly problematic in diabetic patients causing a vicious circle where low serum K+ levels lead to poorly controlled DM and vice versa.”

As you might expect, the suboptimal potassium status seen with diabetics, as noted by Sotirakopoulos (11) et al in “Acid-base and electrolyte disorders in patients with diabetes mellitus,” can lead to acid/alkaline imbalances:

“The patients suffering from diabetes mellitus have disturbances in the electrolytes and in the acid-base balance. These disturbances are caused by the diabetes (glucose balance), renal diseases and medications (diuretics and calcium channel blockers).”

Therefore, it may be wise in diabetic patients to routinely provide them with pH paper so that they can report to you first morning urine pH.

The authors go on to point out that, in both insulin-dependent and non-insulin-dependent diabetics, not only can the serum be low in potassium as noted by Liamis et al (10), but total body potassium can also be reduced:

“The total potassium of the body in insulin-dependent patients and in non-insulin-dependent patients is reduced during periods of poor control of diabetes mellitus and increases when the blood glucose levels are normal.”

The final paper I would like to review on the subject of diabetes mellitus and potassium provides more detail on whole body potassium status in diabetics, particularly those who are uncontrolled. In “Studies on whole-body potassium in non-ketoacidotic diabetics before and after treatment” by Walsh et al (12), the first quote I would like to present makes it clear that potassium depletion is a major issue for uncontrolled diabetics who require insulin:

“…whole-body potassium is reduced in most uncontrolled diabetics who require insulin.”

The next quote points out the physiologic factors that lead to this potassium depletion:
“Tissue breakdown and thereby loss of lean body mass is an important factor in the potassium depletion of ketoacidotic diabetic patients, but cellular depletion of potassium may be more important.”

Before continuing, please note again the statement about “lean body mass” in the above quote. As you know, I feel that sarcopenia (loss of muscle mass) is an extremely important clinical issue with all chronically ill patients. In particular, with uncontrolled diabetics who demonstrate evidence of sarcopenia, as suggested above, it may be wise to make optimization of potassium status a major priority.

The next quote specifically discusses the relationship between diabetic ketoacidosis and potassium status:

“Diabetic ketoacidosis is associated with substantial potassium depletion.”

The final quote from the Walsh et al (12) paper presents an intriguing hypothesis that optimizing potassium status in diabetics may have a positive impact on the health of pancreatic islet cells:

“A further interesting speculation is related to the possible effect of correction of potassium depletion on recovery of islet-cell function. It is not unusual to find that once good control has been achieved in a new diabetic needing insulin the insulin requirements may fall considerably, and occasionally insulin may not be necessary for some months. As potassium depletion impairs insulin secretion and repletion of potassium stores may reverse this abnormality it is possible that the increase in whole-body potassium temporarily improves endogenous insulin production.”

As I hope you can see, it would certainly appear from the research presented above that, when making efforts to prevent catastrophic cardiac events in diabetic patients, evaluation of and, if needed, optimization of potassium status should be an integral part of these efforts.

In the next installment of this series I will review papers that discuss still more clinical conditions associated with suboptimal potassium status.

REFERENCES