The Milner Acetylcholine Protocol (MAP) for Management of Cardiac Dysrhythmias

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Background and Innovation

“There are no naturally occurring beta blockers,” Dr. Milner often says in reference to the use of beta-1 adrenergic receptor blockers (beta blockers) in the treatment of arrhythmias and in control of the heart rate. “Nature doesn’t block receptors,” Milner asserts. “It augments antagonist pathways. Acetylcholine is ‘nature’s beta blocker.’”

There are moments in a physician’s career when he sees the biochemistry of a disease process in a whole new way. Beginning in 2008, Dr. Milner revisited his study of the autonomic nervous system (ANS) control of heart rate; he considered the actions of beta blockers which inhibit sympathetic ANS stimulation of the heart. He considered acetylcholine (ACh), the neurotransmitter of the parasympathetic ANS, and its action on heart rate inhibition. He then developed an approach for using oral supplements to augment ACh production and release. This evolved into the Milner Acetylcholine Protocol (MAP) for management of cardiac dysrhythmias.1

Biochemical Plausibility

The biochemical plausibility of the MAP is based on the ACh inhibitory effect on cardiac rate and dysrhythmias through several mechanisms. The sinoatrial (SA) node, the normal pacemaker of the heart, receives input from the 10th cranial nerve (CN X), the vagus nerve, via ACh. That effect on the SA node inhibits automaticity and slows the heart rate. This is achieved by hyperpolarization of the cells through increased potassium permeability, resulting in slower spontaneous depolarization.2

The atrioventricular (AV) node of the heart also receives input from the vagus nerve via ACh. The effect slows or blocks conduction through the node. Again, this is achieved through a prolonged refractory period that results from increased cell permeability to potassium, which increases the phase 4 portion of the cardiac action potential.

The ventricles of the heart are also affected by ACh, though the vagal innervation of the ventricles is sparse. This occurs by virtue of the antagonistic relationship between the sympathetic and parasympathetic halves of the ANS.

Ventricular myocardium is heavily innervated by the sympathetic ANS. Norepinephrine (NE) is the dominant neurotransmitter which agonizes the beta-1 adrenergic receptors of the heart; it increases automaticity and contractility by increasing the permeability of the cells to calcium. Release of ACh in the supraventricular myocardium and from the few parasympathetic fibers in the ventricles inhibits the release of NE from sympathetic fibers. That NE inhibition prevents increased automaticity by the sympathetic ANS.3

ACh in the human body is synthesized from acetyl-coenzyme A (acetyl-CoA) and

Figure 1: Biosynthesis of ACh and Immediate Precursors
Choline in a reaction catalyzed by the enzyme choline acetyl transferase (CAT; Figure 1, p. 73).

Acetyl-CoA comes from any of several sources, including dehydrogenation of pyruvate, end products of fatty acid metabolism, or production of coenzyme A (CoA) from vitamin B5 which is then acetylated before interacting with choline.

Pyruvate is the end product of aerobic glycolysis. It can also be converted from lactate, the end product of anaerobic glycolysis. Pyruvate can then be converted to acetyl-CoA through the action of the pyruvate dehydrogenase complex. Acetyl-CoA then has several potential fates, all of which demand the transfer of acyl groups, including participation in the Krebs cycle, in fatty acid synthesis, in the production of ACh, and others.

Pantothenic acid (PA), vitamin B5, is the initial substrate for de novo CoA synthesis. This five-step process requires both pantothenate (ionized PA) and the sulfur containing amino acid cysteine. Pantethine is a derivative of PA and is the functional component of CoA in the carrying of acyl groups. Pantethine is composed of two pantethine molecules joined by a disulfide bond.

Choline is an essential nutrient. Humans reutilize choline and synthesize it de novo, but in quantities insufficient to meet metabolic demand. Therefore, an adequate intake of choline is necessary for health. The established adequate intake (AI) in adult men is 550 mg/day and in adult women is 425 mg/day unless pregnant (450 mg/d) or breast-feeding (550 mg/d). Eggs are the richest dietary sources of choline, followed by animal protein sources, leaving vegans and several other groups at routinely inadequate daily intakes. A list of choline content in foods can be found in the Micronutrient Information Center article on choline from the Linus Pauling Institute, Oregon State University.

Choline is an essential substrate in the synthesis of the neurotransmitter ACh, but is also vital in several other bodily processes. Choline is necessary: as a component of cell membranes, in cell signaling molecules, in lipid metabolism and transportation, and as a major source of methyl groups. Methylation reactions turn body processes on and off and activate and inactivate metabolic intermediates.

Choline deficient states may play a part in diseases and conditions including liver disease and fatty liver infiltration, neural tube defects, methylation defects, hypertension, atherosclerosis, neurological disorders, inflammatory disorders, and cancer. Population research has demonstrated that many groups do not achieve their AI of choline. For instance, there is evidence that only 2% of postmenopausal women reach their choline AI on a daily basis.

Research has established that genetic polymorphisms which alter choline or folate metabolism may increase an individual’s dietary requirement for choline. For instance, when 5-methyltetrahydrofolate (5-MTHF) is deficient due to a polymorphism (or other reasons), the body depends on betaine from choline for the conversion of homocysteine to methionine. Methionine is then converted to S-adenosyl-methionine (SAMe), the major methyl donor for one-carbon transfers in the body. When choline is required as a methyl donor it is shunted away from other biochemical processes. Therefore, greater demand for dietary choline may be prevalent in the population based on genetic polymorphisms that are prevalent.

Therapeutic Plausibility

The therapeutic plausibility of utilizing the ACh pathway in the treatment of cardiac dysrhythmias through supplementing oral precursors requires both that (1) oral supplementation be able to raise ACh levels in the neurons; and (2) those increases demonstrate clinical effects.

It is well established that the amount of ACh in the central nervous system is directly affected by the oral intake of choline. Studies and reviews which date back to the late 1970s establish that the synthesis of ACh in the brain (CNS) responds to the availability of choline in the blood and that the ability of neurons to make and release neurotransmitters, including ACh, depends directly on the concentration of amino acids and choline in the blood.

Excellent research has been produced by Marty Hinz, MD, and his groups over the recent decades substantiating that oral supplementation of amino acid precursors can raise endogenous neurotransmitter levels. His work pertains primarily to serotonin and catecholamines, but is applicable to ACh via analogous mechanisms. More importantly, the work of Hinz et al. has established that oral precursor dosing leads to demonstrable clinical effects and improved patient outcomes.

The half-life of ACh is very short at 2 minutes. To ensure fidelity in neuromuscular transmission, the body further enhances this breakdown of ACh enzymatically through acetylcholine esterase enzymes. While ACh is reclaimed by reuptake at the synaptic cleft, augmenting production and release to the extent necessary to inhibit cardiac automaticity will require the persistent availability of substrates for ACh production, a problem that we will need to address.

While we can find no evidence in the medical literature for the use of choline to address cardiac dysrhythmias, we can establish that the biochemical action of ACh exerts control over the cardiac rate and depolarization. The fact that supplementing oral choline raises levels of ACh in the CNS is long established in the medical literature, as cited. Therefore, the biochemical and therapeutic plausibility for the choline aspect of MAP is present.

Pantethine has been reported to have antiarrhythmic action on experimental models of cardiac arrhythmias. In a study of a dog model of hypoxic cardiac perfusion, administration of pantethine was shown to significantly prolong the cardiac action potential and refractory period. While the mechanism of that effect was not described, it is noteworthy that this is the same action which ACh has on the supraventricular myocardial cells, as described above.

Research on rate models of cardiac function has demonstrated that the intravenous (IV) administration of CoA results in a transient decrease in the heart rate. These studies make it appear that it is the adenosine portion of the CoA molecule which may be responsible for the decrease in heart rate rather than the pantetheine, pantothenic acid, or cysteine constituents of the molecule.

The accumulation of long chain acyl fatty acid intermediates promotes cardiac arrhythmias. This has been...
demonstrated in several respects. Cardiac arrhythmia may be the first clinical presentation in children with fatty acid oxidation disorders.25 Experimental models have demonstrated that buildup of acylcarnitines and other intermediates promote dysregulation of calcium resulting in dysrhythmias.26 Ischemic injury in myocardial infarction has also been associated with the buildup of these intermediates promoting arrhythmogenesis. Meanwhile, shorter chain metabolites such as acetyl-CoA and palmitoyl-CoA do not promote arrhythmogenesis.26,27

The potential for statin drugs (HMG-CoA reductase inhibitors) to exert an antiarrhythmic effect on the heart has been an area of research interest. Statins reduce recurrences of supraventricular and ventricular arrhythmias in patients with and without coronary artery disease. Several mechanisms have been proposed for this antiarrhythmic effect, yet none have been proved. Hypotheses include: lowering of LDL cholesterol, improved endothelial function, stabilization of atherosclerosis, anti-inflammatory effects, modulation of membrane ion flux, and improvement of autonomic function.28,29 Experiments using other antilipidemic agents have not shown similar antiarrhythmic effects; therefore something unique about statins must give them their antiarrhythmic properties, rather than their LDL lowering or atherosclerosis stabilizing effects.

Statin drugs inhibit the effect of the HMG-CoA reductase enzyme, leading to an upstream increase in metabolic substrates, especially CoA. It may be that buildup of CoA is responsible, in whole or in part, for the antiarrhythmic effects of statins or for an improvement in autonomic function.

Therapeutic plausibility for the effect of CoA/pantethine/PA on cardiac dysrhythmias is present in the medical literature. Since biochemical and therapeutic plausibility exists for use of either PA/pantethine or choline singly, the potential exists for their combined effect to be greater than the sum of its parts. This is the principle of the MAP.

Protocol, Materials, and Dose Escalation

The principle of the MAP treatment is to give the two essential nutrient precursors for the endogenous production of ACh. These two nutrients are choline and pantothentic acid or pantetheine. At various times we have preferred various forms of these two essential ingredients.

Our current preference is to implement the protocol using choline bitartrate and pantetheine.

Our preference for choline bitartrate developed due to its high solubility in water, its potency, and the challenges in sourcing a lecithin product that is not genetically modified. Furthermore, a significant number of patients are soy or egg sensitive and most lecithin products in the marketplace are soy or egg derived. Our preference for pantetheine over pantothenic acid (PA) comes from our observation that some patients do not respond to PA, though they do respond to pantetheine.

We used to begin the protocol with PA due to the fact that it is considerably less expensive than pantetheine; if a patient did not respond to PA, we would then switch to pantetheine. We now tend to work in the reverse. We know that if we escalate through the dosing stages of MAP using pantetheine and the arrhythmia does not come under control, MAP will not likely work for that patient. Conversely, if we can gain control of the arrhythmia using pantetheine and choline, we can then try a switch to PA if the patient desires a lower cost option.

Achieving a delivery system which keeps the substrates at constant blood levels is a challenge. As discussed, if we want to ensure that adequate levels of precursors are available to be therapeutic, they must persist at reasonable blood concentrations at all times. We have found only one way to provide this type of steady state and that is to dose the nutrients in a beverage which the patient can drink continuously throughout the day.

We instruct patients to mix their nutrients in a 32 fluid ounce (fl. oz.), drinking bottle with water at the beginning of the day. Assuming that the patient is awake for 16 hours per day, we divide the day into 4-hour segments. During the first 4 hours of the day the patient drinks 4 fl. oz. of the beverage, between hours 5 to 8 they drink 8 fl. oz., during hours 8 to 12 they drink another 8 fl. oz., and during their final 4 hours awake they drink 12 fl. oz., ensuring adequate levels overnight (Table 1).

The MAP dosing works in a three-stage dose escalation (Table 1). At stage 1 we deliver 500 mg of pantetheine and 2000 mg of choline per day. Due to the very short half-life of ACh we are able to tell within one week whether this dose is adequate. If the patient achieves complete symptomatic or objective improvement over the course of the first week then the MAP is maintained at stage 1; if not, we escalate to stage 2. Stage 2 dosing is 1000 mg of pantetheine and 4000 mg of choline; after one week we reassess. If resolution of symptoms or objective findings is complete or near complete we stop at stage 2, otherwise we escalate to stage 3. Stage 3 dosing is 1500 mg of pantetheine and 6000 mg of choline. Reassessment is made after one week in the same manner as previous stages. It is rare that more than 1500 mg of pantetheine will demonstrate further benefit, though we have had patients who needed to escalate the initial dose of choline up to 10 grams before coming under rhythm control. After several weeks of a persistent symptom-free/arrhythmia-free period, a down titration can be attempted to find the lowest effective dose that maintains the arrhythmia resolution. When PA is substituted for pantetheine, it is used at

Table 1: MAP Dosage Forms, Dose Escalation, and Delivery Method

<table>
<thead>
<tr>
<th>Stage</th>
<th>Pantothentic acid/Pantetheine</th>
<th>Lecithin/Choline Bitartrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>1000 mg/500 mg</td>
<td>2 Tablespoons/2000 mg</td>
</tr>
<tr>
<td>Stage 2</td>
<td>2000 mg/1000 mg</td>
<td>4 Tablespoons/4000 mg</td>
</tr>
<tr>
<td>Stage 3</td>
<td>3000 mg/1500 mg</td>
<td>6 Tablespoons/6000 mg</td>
</tr>
</tbody>
</table>

*** Mixed with a sufficient quantity of water to make 32 fluid ounces (fl. oz.).

This beverage is drunk throughout the day according to the following schedule:

<table>
<thead>
<tr>
<th>Awake Hours</th>
<th>Drink this Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hours 1–4</td>
<td>4 fl. oz.</td>
</tr>
<tr>
<td>Hours 5–8</td>
<td>8 fl. oz.</td>
</tr>
<tr>
<td>Hours 9–12</td>
<td>8 fl. oz.</td>
</tr>
<tr>
<td>Hours 13–16</td>
<td>12 fl. oz.</td>
</tr>
</tbody>
</table>

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double the dose of pantethine used for that stage: 1000 mg at stage 1, 2000 mg at stage 2, 3000 mg at stage 3. When lecithin is substituted for choline bitartrate we use a non-GMO soy lecithin which contains 1000 mg of choline per tablespoon (Table 1).

**Indications**
MAP is indicated for any tachyarrhythmia, including sinus tachycardia, atrial tachycardia, supraventricular tachycardia, atrial fibrillation, atrial flutter, and accelerated ventricular rhythms, provided there are no contraindications with other aspects of treatment. MAP is also indicated for ectopic arrhythmias such as premature atrial contractions (PACs) and premature ventricular contractions (PVCs). MAP is appropriate as adjuvant therapy in the vast majority of circumstances; however, potential interactions with other treatments and medications should always be scrutinized. Patients should be closely monitored for adverse events during initiation and dose escalation. Several noncardiac indications for MAP also exist, including anxiety, hyperactivity, reactive attachment disorder, PTSD, and other deficits of neurotransmitter and ANS balance.

**Side Benefits**
There are several potential side benefits to supplementation with choline and pantethine or PA. Patients who take methotrexate or who have defects in folate or choline metabolism are known to have a higher dietary requirement for choline.7 Those who take oral contraception, estrogen, or progesterin may have an increased requirement for pantethanic acid.5

Choline is important in several aspects of cardiovascular health. It is vital in the endothelial functions that regulate blood pressure; choline insufficiency has been implicated in atherosclerosis, and it has been shown to lower levels of homocysteine.5,30–32 It may help to control severity and need for medication in asthma.33,34 Choline may lower the incidence of neural tube defects.14,35 It may have a positive effect on several neurological conditions and processes, including psychiatric disorders, dementia, memory, and cognition.36–39 Diets deficient in choline induce fatty liver disease, and oral consumption of choline has been shown to help reverse fatty liver disease in several studies.40–42 Therefore, it is an important therapy in patients with this condition or at risk for it due to other conditions such as metabolic syndrome. Several studies have linked the importance of dietary choline to the prevention of colon, breast, and other cancers.43–45 Pantethine may improve responses to stress.46 Pantethine has been shown to improve LDL cholesterol and triglyceride levels in repeated research at doses of at least 600 to 900 mg/day.47–51

**Contraindications and Side Effects**
It is theoretically contraindicated to use MAP in cases of bradycardia and bradydysrhythmias or in conditions in which increasing the refractory period of the SA or AV node would be contraindicated such as first, second, or third degree AV heart blocks, SA node exit blocks, sinus pauses, and sinus arrest. There are no known interactions between choline and other supplements, drugs, diseases, conditions, or lab tests.32 The most concerning possibility for interactions between pantethine or PA and other drugs, supplements, and conditions is in anticoagulation/antiplatelet therapy and bleeding disorders.33,34 Extreme caution should be used in these circumstances and best practices would dictate counseling patients to discontinue use of pantethine two weeks before surgery.33

Choline can cause adverse reactions including sweating, gastrointestinal distress, nausea, vomiting, diarrhea, and a fishy body odor.7 Guidelines indicate that daily choline doses should not exceed 3500 mg per day in adults; however, doses of 12,000 to 16,000 mg/day have been used in a study on seizure disorders and doses of 7500 mg/day in a study on high blood pressure, both without significant adverse events.7,55 A single prospective investigation found an association between high choline diets and colorectal adenomas in women, though this observation may have been attributable to any of several other factors.56 People

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**Table 2: MAP – Summary of Cases 1–3**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Presentation</th>
<th>Objective Findings</th>
<th>Final MAP Dose</th>
<th>Other Treatments</th>
<th>Final Outcome on Stable MAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>44 yo female</td>
<td>Ten years of palpitations, two week exacerbation palpitations, occurring every 30 seconds.</td>
<td>ECG: accelerated junctional rhythm with nonspecific ST changes.</td>
<td>Stage 1: PA 1000 mg/ Lecithin 2000 mg</td>
<td>None</td>
<td>After one week: normal ECG. After one month: only a single episode of palpitations over the previous two-week period.</td>
</tr>
<tr>
<td>29 yo male</td>
<td>Ten month history of palpitations, 30 seconds in duration, up to three times daily, worse post-exercise, worse anxiety, especially test anxiety.</td>
<td>Exercise stress test: atrial bigeminy and quadrigeminy, up to 13 PACs/min, ectopic atrial pacemaker, inappropriate blood pressure response to exercise.</td>
<td>Stage 2: Pantethine 1000 mg / Choline bitartrate 4000 mg</td>
<td>B complex vitamin 2/d, magnesium glycinate 480 mg/d, botanical formula 2 mL, 3×/d; all of these unchanged since MAP stage 1.</td>
<td>After one month: no palpitations or arrhythmic beats, no complaints or symptoms postexercise, before or during stressful events or exams.</td>
</tr>
<tr>
<td>59 yo female</td>
<td>3 months of palpitations, “flutters” lasting up to a week or more.</td>
<td>ECG: persistent ectopic rhythm. Holter monitor: atrial tachycardia, premature atrial and ventricular beats.</td>
<td>Stage 2: Pantethine 1000 mg/Choline bitartrate 4000 mg</td>
<td>Quetiapine 12.5 mg/d, tyrosine 1000 mg/d 5-HTP 300 mg/d, methylfolate 3.75 mg/d; all since before MAP initiation.</td>
<td>After two weeks: self report of “significant improvements” in palpitations and symptomatic heartbeats.</td>
</tr>
</tbody>
</table>
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with liver or kidney disease, Parkinson’s disease, depression, or trimethylaminuria may be at an increased risk of adverse events when consuming choline at a level up to or near the upper limit, though otherwise there is no known toxicity of choline to humans.\textsuperscript{7,57} It is regarded as likely to be safe in pregnancy and lactation when the upper limit is not exceeded.\textsuperscript{52}

Pantethine is exceptionally well tolerated at doses up to 1200 mg.\textsuperscript{5,47–51,53,54} The most common side effects are gastrointestinal in nature and include nausea, diarrhea, and epigastric discomfort. When using doses in excess of 1200 mg, monitor closely and down titrate or discontinue when necessary for side effect control. There is insufficient evidence to support the safety of its use in pregnancy and lactation and should therefore be avoided.\textsuperscript{51} There is no known toxicity to humans even at very high levels of intake.\textsuperscript{5,53}

**Patient Data**

We have used the MAP protocol to address tachyarrhythmias, including atrial fibrillation, ectopic rhythms, and palpitations. In the upcoming months, we will publish a peer-reviewed case series on the success of the use of MAP in patients with these conditions. A small sampling of successful case data will be discussed here and is summarized in Table 2.

**Case 1**

C. F., a 44-year-old female, presented to clinic with complaints of palpitations, pounding heart, and rapid heart rate. Occasional “flip-flop” sensations in her heart had occurred for more than 10 years, but had increased in frequency to every 30 seconds over the previous two weeks. Her physical exam was normal across seven systems. Her electrocardiogram (ECG) revealed an accelerated junctional rhythm, diffuse nonspecific ST segment changes, and no arrhythmic beats (Figure 2A). Blood chemistries and thyroid lab tests were within normal limits. MAP was initiated at 1000 mg of pantothenic acid and two tablespoons soy lecithin mixed in water and used daily.

One week later, the patient reported noticing a reduction in heart rate within three minutes of her initial use of the MAP. She was still having occasional palpitations but much less frequently and lasting only 10 seconds; she had not been using the MAP daily. Her ECG on that date was normal, demonstrating a sinus rhythm at a rate of 60 bpm and no ST changes (Figure 2B). The patient was reminded to use the MAP on a daily basis and was maintained at stage 1.

One month after the initial visit, the patient reported only one episode of palpitations over the previous two week period which lasted less than one minute in duration. Overall, she was pleased and thought that the treatment plan had been effective.

**Case 2**

X. C., a 29-year-old male, presented with complaints of palpitations lasting approximately 30 seconds and occurring every other day up to 2 to 3 times daily for the previous 10 months. They felt like early, strong, or pounding beats in his chest and neck that gave him a “dropping” feeling in his chest. An exercise stress test revealed premature atrial contractions (PACs) in patterns of bigeminy and quadrigeminy, 13 PACs per minute, an ectopic atrial pacemaker, and inappropriate blood pressure response to exercise.

The patient reported daily use of a B vitamin complex and 1 gram of essential fatty acids. The MAP stage 1 was initiated using pantethine 500 mg and choline bitartrate 2000 mg along with B complex twice daily, a botanical formula (two parts each Passiflora incarnata and Crataegus oxycantha, one part each of Valeriana officinalis and Leonurus cardiaca), 2 mL three times/day, and magnesium glycinate 480 mg/day.

One week later, he reported noticing improvements after the first three days of treatment. He was feeling fewer palpitations and less anxiety in stressful situations, but was still having 1 or 2 symptomatic palpitations after daily exercise. The MAP was increased to stage 2 and no other changes were made to the treatment plan.

At two-week follow up, the patient reported that his palpitations had improved further, but were not yet completely gone. He still noticed palpitations if he did not get enough rest. Their quality and severity had decreased; he now reported them as “very subtle.” Stage 2 MAP was maintained.

At follow-up two weeks later, the patient reported that he had not been having palpitations. Cardiac monitoring for heart rate variability revealed no arrhythmic beats.

**Case 3**

G. S., a 59-year-old female, presented with “flutters” after receiving “a lot of epinephrine” during a dental procedure 3 months prior. Immediately after the procedure she began experiencing “strong and weak flutters” that could last a week or more. The severity of her palpitations had decreased somewhat over the three

![Figure 2: 12 lead electrocardiograms for Case 1 – 44-year-old female before (A) and after (B) MAP treatment.](image-url)
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months, but they persisted and were worse at night. She had an irregularly irregular pulse in a recent past visit with her PCP. She presented to us for a resting ECG which demonstrated persistent nonsinus rhythm, nonspecific ST changes, no arrhythmic beats. Follow-up 24-hour Holter monitoring revealed PACs, PVCs, and short periods of atrial tachycardia. Her history was significant for PTSD treated with quetiapine 12.5 mg, L-tyrosine 1000 mg, 5-HTP 300 mg, and methylfolate 3.75 mg, daily. MAP was initiated at stage 2 using 1000 mg pantethine and 4000 mg choline bitartrate.

Two weeks later, at the first follow-up visit, the patient reported “significant improvements” in her palpitations and her experience of discernible heartbeats.

Conclusion
MAP therapy has been successful in many patients with varying types of tachycardia and dysrhythmias over its years of use. We have successfully combined it with other natural, medical, and surgical interventions to equal success. There are patients who are nonresponders to MAP, but they can be identified and redirected to other therapies within a few weeks of MAP trial. MAP is our first line natural treatment for tachydysrhythmias, especially when comorbidities such as dyslipidemia or metabolic syndrome exist.

We have presented only a small sample of straightforward case data here. We will continue to publish on the successful use of MAP in more complex cases beginning with an upcoming peer-reviewed case series. Following that, we intend to begin comparative effectiveness trials of MAP plus usual therapy (conventional pharmaceutical rate and rhythm control agents) versus usual therapy plus placebo. The MAP is a promising treatment option that warrants further investigation.

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These authors have no financial conflicts of interest to declare.

Notes
16. da Costa KA, Kozyreva OG, Song J, Galanko JA, Fischer LM, Zeisel SH. Common genetic polymorphisms affect...


