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 ➤

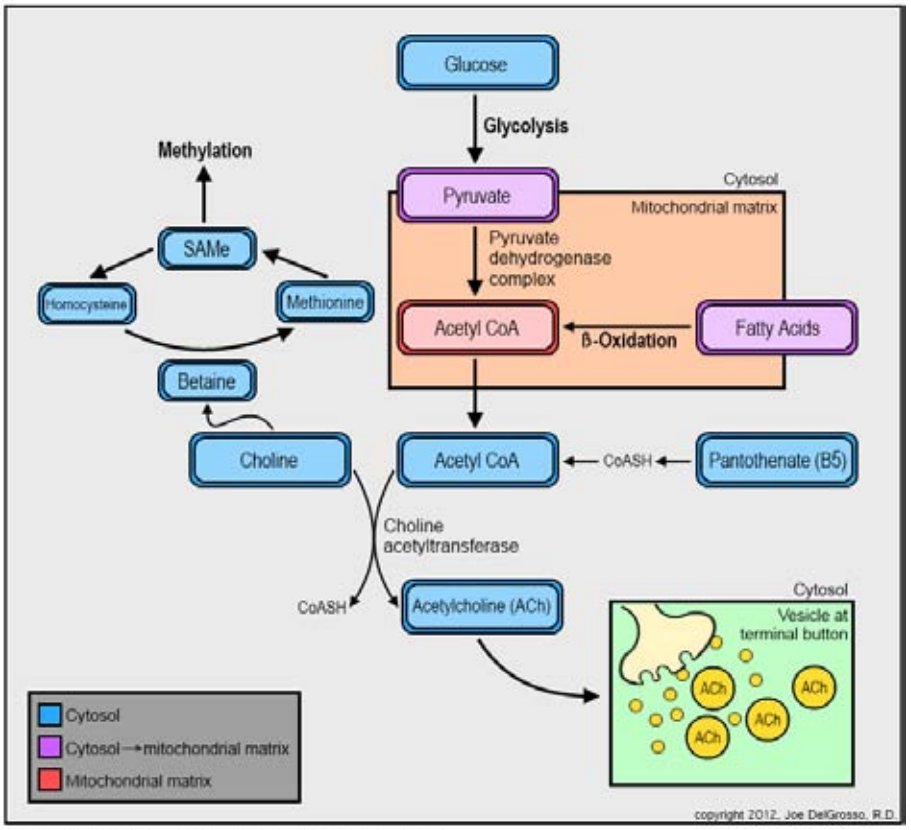

Figure 1: Biosynthesis of ACh and Immediate Precursors