The Excitable Heart

Part II: Mechanisms of Cardiac Arrhythmias

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Like the hum of a well-tuned car, the beating of a healthy heart goes largely unnoticed. Both depend on a highly sophisticated electrical distribution system. In the case of our heart, that distribution system is comprised of special tissues and cells designed to conduct electrical impulses, unlike the vast majority of heart cells which are designed to contract. As discussed in Part I of this series, it is the flow of ions such as potassium, sodium and calcium across the cardiac cell membrane that produces the electrical impulse that triggers the contraction of individual muscle cells, thus making possible the pumping action of the heart.

The electrical signal that sets the heart in motion originates in the right upper chamber (right atrium) in a structure known as the sinus node. Traveling through specialized pathways, the impulse spreads throughout the right and left atria and then squeezes through an isthmus that connects the upper and lower chambers (ventricles) of the heart. This junction known as the atrioventricular (AV) node serves as a relay station where the impulse is delayed, providing enough time for the blood in the atria to empty into the ventricles. Because it serves as the gatekeeper for the ventricles, the AV node is also referred to as a "safety valve", regulating the rate at which impulses can propagate from the atria to the ventricles. From there the electrical impulse journeys down a divided cellular highway (bundle branches), simultaneously activating both ventricles. The conduction system terminates in a webbed network of specialized (Purkinje) fibers that permit nearly synchronous activation of the cells of the ventricles from the bottom up so as to effectively squeeze out the blood. This well organized sequence of activation, the result of millions of years of evolution, allows for quiet and efficient pumping of blood by the heart for the purpose of providing oxygen and nutrients to the rest of the body.

The healthy heart follows this same pattern of electrical activation about 70 times each minute; this is the basis for the normal heart rate. The efficiency of the heart as a pump can become seriously compromised when the electrical distribution system breaks down. Just as a faulty distributor can cause a car to stutter and sputter, a faulty distribution system in the heart can cause rhythm disturbances, or arrhythmias, that can alter its normal function. An arrhythmia occurs when the heart beats too slowly (bradycardia), too rapidly (tachycardia) or erratically.

A very slow heart rate results when either the sinus node, the primary pacemaker of the heart, is defective, or the AV node, the structure through which the sinus impulse must pass to activate the ventricles, is defective. When impulse conduction between the atria and ventricles is blocked (heart block), the heart does not stop completely because subsidiary (backup) pacemakers located in the ventricles take over control of the heart. However, their rate, usually less than 40 beats per minute, is too slow to maintain normal function and in some cases is too slow to maintain consciousness. Fortunately, the problem is easily remedied by the implantation of an artificial pacemaker, a device that

provides electrical pulses to stimulate the heart and thus restore normal rate and rhythm. Although very slow heart rates can be life-threatening, most arrhythmic deaths result from abnormally rapid heart rates.

A tachycardia results when either the atria or the ventricles beat too rapidly. There are several mechanisms by which this can occur. Abnormal impulse formation due to altered movement of ions across the membrane of *pacemaking* cells is one mechanism. Another, more common mechanism, involves the recirculation (reentry) of the electrical impulse around a real or functional obstacle in a never ending loop characteristic of a dog chasing its tail. The normal pattern of electrical distribution is therefore disrupted, resulting a short-circuiting of a part of the heart. Tachycardia can degenerate into fibrillation, also known as sudden cardiac death when it occurs in the ventricles, if the electrical activity becomes further disorganized.

Because the ventricles do most of the pumping, a rapid rate in the ventricles is generally more serious than in the atria. Inasmuch as the AV node limits the number of atrial beats that reach the ventricles, an abnormally fast heart rhythm can exist in the atria while the ventricles beat at a near normal rate. Medical treatment of fast heart rhythms ideally should be tailored to the underlying mechanism responsible for the arrhythmia. Artificial pacemakers, automatic defibrillators, antiarrhythmic drugs and ablation techniques are today available to treat and prevent the development of life-threatening cardiac arrhythmias.

These therapeutic modalities are available today due in part to the vital research conducted by the internationally renowned Experimental Cardiology team at the Masonic Medical Research Laboratory. The research findings of the MMRL's scientists together with those of researchers at other institutes worldwide have translated into therapies and preventative measures that have saved countless lives. In the next segment of this series, I hope to expand on the causes of tachycardia and fibrillation and to further discuss how research can provide a new lease on life for many patients.

The Masonic Medical Research Laboratory (MMRL) is a 501(c)3 not-for-profit corporation founded and sponsored by Freemasonry. Recognized as a one of the finest biomedical research centers in the world, the MMRL has contributed importantly to the modern day practice of cardiology. Over the past five decades MMRL investigators have been credited with either discovering or unraveling the mechanisms of a majority of known cardiac arrhythmias and is currently one of a handful of medical research institutes worldwide capable of studying the genetic causes of the lethal cardiac arrhythmias responsible for sudden death in young adults, children and infants. The MMRL is leading the way in the development of innovative safe and effective pharmacological treatment for atrial fibrillation, one of the greatest unmet medical needs facing our society.

The Masonic Medical Research Laboratory is the Flagship Charity of the Grand Lodge of Florida, Free and Accepted Masons. The institute is also supported by Grand Chapter of Florida, Royal Arch Masons and the general public as well as through corporate,

foundation, National Institutes of Health and American Heart Association grants. On average, 84 cents of every dollar donated goes to cardiac research.

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