Less of a shock

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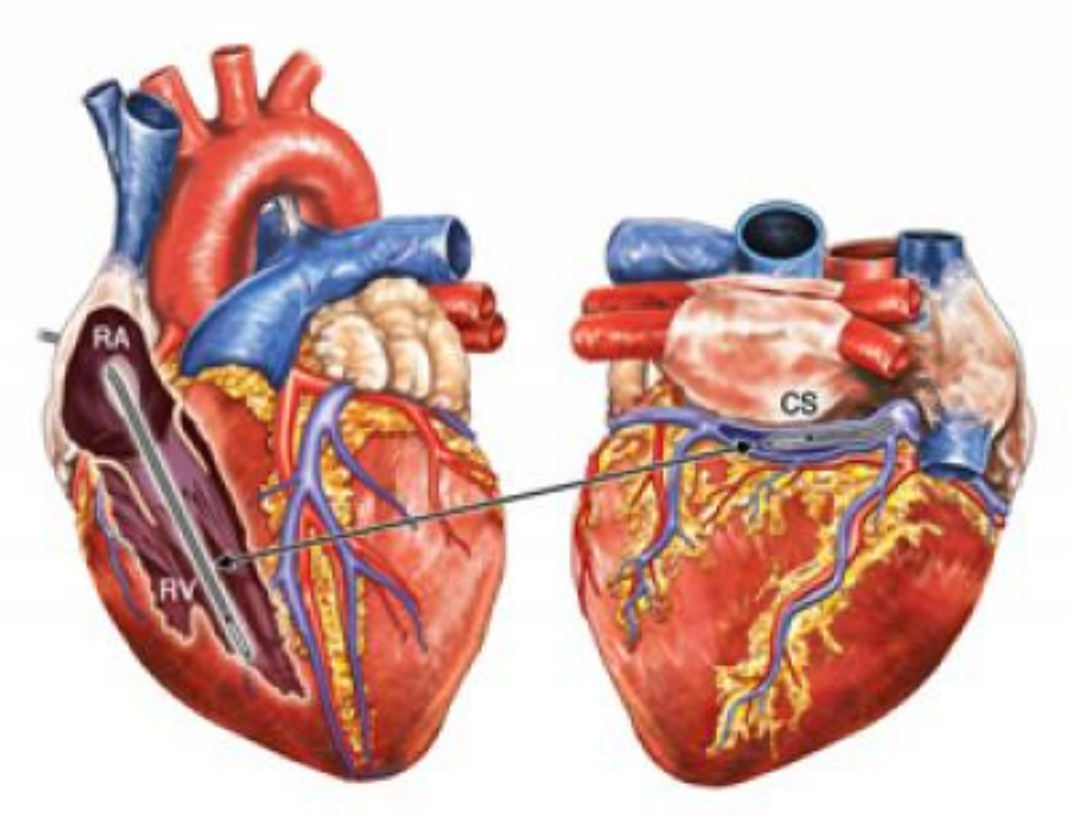

Implantable defibrillators currently on the market apply between 600 and 900 volts to the heart, almost 10 times the voltage from an electric outlet, says Ajit H. Janardhan, MD, PhD, a cardiac electrophysiology fellow at the Washington University's School of Medicine.

After being shocked, he says, some patients get post-traumatic stress disorder. Patients may even go so far as to ask their physicians to remove the defibrillator, even though they understand that the device has saved their lives.

The huge shocks are not only unbearably painful, they damage the heart muscle and have been shown in many studies to be associated with increased mortality. In an advance online edition of the Journal of American College of Cardiology, Janardhan and Igor Efimov, PhD, professor of biomedical engineering in the School of Engineering & Applied Science, report on a low-energy defibrillation scheme that significantly reduces the energy needed to re-establish a normal rhythm in the heart's main chambers.

They hope this electrotherapy will be much less painful than shocks from existing implantable defibrillators, and may even fall beneath the threshold at which patients begin to perceive pain.

The team has just received a National Institutes of Health grant to develop a

prototype low-energy defibrillator for humans and plan to begin clinical trials of the device shortly.

Losing the beat

The lub-dub of the heartbeat begins with an electrical impulse generated by the sinoatrial node, a group of cells on the wall of the right atrium that is the heart's natural pacemaker.

Spreading through conductive pathways in the heart, the electrical signal first causes the two upper chambers of the heart (the atria) to contract, and then, a split second later, the two lower chambers (the ventricles), coordinated motions that efficiently pump blood to the rest of the body.

The synchronized squeezing of a normal heartbeat is called sinus rhythm, after the node that triggers it.

The rhythm can go wrong in many different ways, but the real killer is ventricular tachycardia. Ventricular tachycardia is an abnormal heart rhythm that starts in the ventricles rather than from the sinoatrial node, and that causes the heart to beat at a rate too fast (tachy is Greek for rapid or fast) to efficiently pump blood to the rest of the body.

Moreover, the rapid heartbeat can degenerate precipitously into ventricular fibrillation, or the loss of all rhythm, says Efimov. During ventricular fibrillation the uncoordinated contraction of heart muscle prevents the heart from pumping blood at all, and without immediate intervention, death quickly follows.

Most people who develop ventricular tachycardia and ventricular fibrillation outside the hospital die, says Janardhan, but studies show that if we implant a defibrillator in patients with a weak heart that does not pump as strongly as it should, we can significantly reduce mortality.

Restarting the rhythm

There are really only three therapies for ventricular tachycardia, Efimov says. One is drugs that reduce the likelihood of tachycardia, but drugs are often ineffective.

The second is ablation, or the deliberate creation of nonconductive scar tissue within the heart that blocks abnormal conductive patterns and redirects electrical activity to more normal pathways.

The major problem with ablation, says Efimov, is recurrence. It's a temporary measure, not a cure. Patients typically need additional treatment within five years.

The third therapy is an implantable cardioverter defibrillator, or ICD. These devices are placed beneath the skin in the chest and monitor the rate and rhythm of the heart. If they detect ventricular tachycardia, they try to break the rhythm by pacing the heart at a rate faster than its intrinsic rate, a strategy anti-tachycardia pacing.

Anti-tachycardia pacing is very low energy, so low that patients may not even sense it. But it is relatively ineffective when the heart is beating 200 time per minute or faster. At these higher rates, the ICS zaps the heart with a strong electrical shock that resets it and, with luck, allows the pacemaker node to restart it with a normal rhythm.

A novel electrotherapy

The scientists knew from earlier experiments that the voltage needed to shut down ventricular tachycardia depended on the timing of the shock. This led them to ask whether a sequence of multiple, closely timed low-voltage shocks might be more effective than a single high-voltage shock, and be less sensitive to timing.

Indeed it turned out that if they shocked the heart multiple times they could reduce the peak shock amplitude from well over 200 volts to 20 volts, timing no longer mattered, and the therapy worked even if the ventricular tachycardia was very rapid.

Although this electrotherapy involves multiple shocks, the total energy it delivers is still lower than that of a single large shock, roughly 80 times lower.

Why do multiple shocks work better? Arrhythmias generate electrical wave vortices — little electrical tornadoes in the heart — and it is these vortices, or re-entrant circuits, that make the heart beat too fast and prevent it from pumping properly.

But immediately after it contracts, heart muscle goes through a refractory, or unresponsive, period during which it does not respond to electrical stimulation. The multiple shocks may do a better job of extinguishing the re-entrant circuits by creating an area of unresponsive muscle into which the re-entrant wavefront –the electrical tornado – crashes, the scientists suggest.

Relocating the Electrodes

Defibrillators now on the market apply shocks between the right ventricle (RV) and an "active can" located above the chest wall, below the collarbone. The shocks are painful in part because they pass through the chest wall muscle and sensory nerves.

The investigators found they could reduce peak shock voltages by an additional 50 percent if they applied shocks between the RV and coronary sinus (CS), a vessel that collects deoxygenated blood from the heart muscle, rather than through the chest wall. Less energy was required because the shocks were confined to the heart itself, and for the same reason they were also less painful.

In an earlier paper, Efimov's student Wenwen Li, PhD, now at St. Jude Medical, had reported on a similar strategy for restoring the rhythm of the atria, the two upper chambers of the heart, for a less serious but more common rhythm abnormality.

The team has already developed the first low-energy atrial defibrillator, which will soon be entering clinical trials. They hope for similarly rapid progress with the ventricular defibrillator.

"We think this technology can and will be implemented soon," says Janardhan. "There's a lot of cardiac research that may pan out 20 or 30 years from now," he says, "but as a physician I want something that can help my patients now."

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