News
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******Researchers probing effects of circulatory shock on the heart.

DALLAS--Circulatory shock. It's the signal that death is now a very real possibility.

Labeled as one of the world's leading killers, circulatory shock claims a mortality rate of from 50 to 80 percent. Once shock reaches the advanced stages, irreversible processes are set in motion.

The high mortality rate of circulatory shock indicates to many medical professionals both the seriousness of the problem and the relative lack of understanding of how to treat it. Medical seminars dealing with the subject often end in debate on which things are of primary importance as to how circulatory shock functions and which things are peripheral.

Drs. Janet Parker and H. Richard Adams of the pharmacology faculty at Southwestern Medical School are researchers who suspect that the heart is a primary factor in shock. Aided by a grant from the American Heart Association, the two are performing animal studies to isolate heart muscle, evaluate the function of the heart in shock, and check the heart's responsiveness to various drugs.

Shock may occur in cases of severe burns, injury or infection, or it may follow a heart attack. Circulatory shock is generally characterized by a collapse of the circulatory system--cardiac output decreases, blood pressure drops and blood flow is limited. The reduction of blood flow and the subsequent lack of blood-carried oxygen to the body's vital organs often damages those organs beyond repair.

Externally, shock victims often have pale, clammy skin, a weak pulse and labored breathing. Internally, shock can make profound changes on even the cellular level. Body cells swell, pointing to possible damage to the cell membrane, and as shock develops in severity, the cell membrane will eventually break up and the cell will die.

Currently, doctors treat shock victims as symptoms occur, but treatments have little control over the mechanisms causing the problem.

Parker and Adams are studying the mechanisms of the heart in shock by taking heart muscle from animals in shock and then placing the shocked heart muscle in a special bath which allows it to keep contracting for several hours. The two are examining differences in strength of contractility between the shocked heart and a normal heart, and they are able to measure changes in the heart muscle when drugs are put into the surrounding bath.

"Some researchers and clinicians believe that the heart functions relatively normally in the shock patient, and other have suggested that cardiac failure may contribute to the shock state. However, our shock model has the advantage of enabling us to evaluate the heart by itself, independent of the nerves, hormones and other factors circulating around it in the body and influencing it," explains Dr. Parker.

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She and Adams have noticed that the heart of an animal in shock doesn't beat as hard as it should. They have taken measurements and found that the shocked heart beats about half as strong as normal. Parker says that this fact is significant since in the human patient this inherent weakness in the heart might be masked for a period of time by nerves and hormonal influences which support the heart. When the support diminishes the already depressed heart may seem to fail suddenly.

Calcium, which plays a central role in the normally contracting heart, may not be getting into the cell in sufficient quantities, says Parker. Since the heart can't beat without it, she and Adams suspect that the shocked heart muscle cell does not metabolize calcium normally. They have found that if they give calcium by adding it to the bath, the contractility of the shocked heart returns to normal. "So we can induce normality in our isolated heart muscle," says Parker. "Contractility of the heart in shock can be normal if supplied with enough calcium."

Calcium has been given to patients in severe shock, but this treatment has not been successful since cardiac output can't be maintained.

"We're using calcium to gain insight into possible mechanisms of the shock-induced dysfunction," says Parker. "There is evidence that something is keeping sufficient amounts of calcium from getting into the cell. Now we're using different drugs that affect calcium movement across the cell membrane to see what they do in the shocked heart. These are drugs that either block calcium activity or influence calcium movement. The heart in shock does not respond normally to these drugs, which also indicates a calcium-related problem of the heart cell."

She says that their ultimate goal is to find a drug or treatment which will prevent these cellular changes and reduce the depressed reaction of the heart to circulatory shock.

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