clinical commentary with particular reference to the singular and understated sentence: “Recent studies suggest that cardiac troponin, a myocardial regulatory protein, is a more specific marker than CK-MB for cardiac injury,” citing one reference.1

Review of the recent reports, the sporadic medical literature, and the current basic research indicates that the cardiac myotropins are not only highly specific for the diagnosis of myocardial injury, regardless of causation, but in the case of cardiac troponin I (CTI), it may now be considered to be the supporting “gold” standard for the diagnosis of myocardial injury. Cardiac troponin T (CTT) can also be effectively utilized for a more specific diagnosis of myocardial injury than creatine phosphokinase-MB fraction (CPK-MB) if one realizes that its detection has been reported to be increased in patients with renal failure, polymyositis, and possibly in other regenerating skeletal muscle conditions.2 In fact, the re-expression of the cardiac troponin T-2 isoform has been noted in severe heart failure of varying etiologies.3 The specificity of CTT in all probability has not as yet been fully defined; however, one report in which CTT was determined by a more recent enzyme immunoassay technique claims cardiосpecificity for the monoclonal antibodies utilized in the procedure.4

Both CTI and CTT are long-lived markers of cardiac injury, and abnormal levels can persist for 4 to 7 days or longer following various types of myocardial injury such as severe ischemia, infarction, and blunt cardiac trauma, thus obviating the use of CPK and LDH isoenzymes in most clinical situations where confusion is believed to exist. Other than their obvious benefit in the delayed diagnosis of myocardial infarction and in the diagnosis of myocardial contusion, they would, for instance, be of great value in the diagnosis of ischemic myocardial injury in a distance runner, where ordinarily there is much confusion caused by using other enzyme assays.5 For all practical purposes, troponin I is not significantly expressed in any tissue except heart muscle, making it highly specific as a marker for cardiac injury, and if not slightly more sensitive than CPK-MB, it is at least as sensitive. The common use of myoglobin levels in such situations is not without its problems either, since it too, like creatine kinase, is derived largely from skeletal muscle. In response to myocardial injury, however, myoglobin is much more rapidly released into the circulation for appropriate measurement.

As biochemical markers for cardiac injury, it is my understanding that it is the type of immunoassay presently used by the manufacturers concerned that determines the specificity of their product, and in part, its sensitivity. The producers of both the troponin T assay and the troponin I assay claim high degrees of specificity, exceeding other markers for cardiac muscle damage.

Presently, it is my recommendation that all patients in whom a myocardial contusion is suspected or must be ruled out, should have the following studies: electrocardiogram (serially), chest x-ray, echocardiogram, and cardiac troponin analysis. I have well-advised general and specific reasons for each, regardless of the opinions and recommendations which others may provide. I will not attempt to discuss it further other than stating that it is my opinion that this approach is absolutely necessary to avoid the medical-legal problems that have become quite common following blunt chest trauma.

In conclusion, it is justifiable to recommend that the measurement of cardiac troponins replace the assay of CPK-MB as a criterion in the laboratory diagnosis of myocardial contusion. The work of Adams et al,1,2 Anderson,3 and others,4,6 is supportive of this opinion.

Basil M. RuDusky, MD, FCCP, Wilkes-Barre, Pennsylvania

References


Goodbye to ABCD Resuscitation?

To the Editor:

Dr. Idiro’s editorial (CHEST 1995; 108:1490-91) reminds us that expired air resuscitation may not, after all, be the first step for best results from CPR. Even a quarter of a century ago,1 it was clear that there were a number of good reasons for abandoning the now hallowed ABCD sequence. They include the following considerations:

• Except in anoxic cardiac arrest, the lungs normally contain enough oxygen for about 30 s of apnea and for some minutes after breathing pure oxygen.
• A normal person can hold his or her breath for several minutes without ill effects, although, of course, at the price of enhanced oxygen extraction.
• The brain is more tolerant of hypoxia than ischemia, probably because it has a capacity, albeit limited, for anaerobic metabolism. “Better black blood than no blood.”
• Artificial respiration does not circulate blood to the brain.
• The arrested heart continues metabolic activity, and it seems more likely to restart while it is still oxygenated.
• Expired air resuscitation is a more skilled technique than chest compression, and it is likely to waste precious time with inexperienced hands.
• Successful cardiac resuscitation has been reported using only diffusion respiration during apnea.
• With swift chest compression, normal breathing may continue for some moments, probably because the midbrain continues to function normally while its oxygenation remains satisfactory.
• Currently, for esthetic and health reasons, bystanders more than ever are reluctant to initiate expired-air resuscitation, and it is very possible that the loss of potential survivors is increasing.
• The somewhat formerly popular technique of thumping the chest may have owed its success to its immediacy, with the heart being still oxygenated.
• Finally, there is no clinical evidence to support ABCD in cardiac arrest.

Alan Gilston, MB
London, England

Reference