Acute Compartment Syndrome: The Quagmire between the Devil and a Hard Place – Some New Promises

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By definition, the diagnosis of acute compartment syndrome (ACS) implies tissue necrosis due to ischemia in a specific tissue compartment. A recent review of the topic provides a reasonable and basic overview of the types of compartment syndrome and the diagnostic dilemmas they present.[1] The reader is encouraged to read this publication. The controversy regarding the use of regional anesthesia in patients with trauma has also been widely discussed,[1–6] and it seems that prevailing opinion tends toward the idea that ischemic pain cannot be blocked, and that while a regional block most probably does not mask the development of ACS, it may be therapeutic by increasing tissue blood flow.[7] However, this notion remains controversial at such an early stage of our knowledge.

The quagmire of the diagnostic and therapeutic dilemma surrounding ACS is that once the diagnosis of ACS is made, tissue necrosis has already begun and all treatment becomes aimed at limiting that tissue damage, making time a critical commodity. The tissues at risk are nerve and muscle tissue. In an ideal world, one would like to decompress the tissue before necrosis develops, usually, through prophylactic compartment release by fasciotomy. This early intervention becomes a judgment call, but this call is based on very soft and non-specific clinical symptoms and signs (pain out of proportion to what is expected and a positive stretch test), and even softer and less specific special examination (compartmental pressure and delta pressure measurement). Thus, one has to treat a condition that does not (yet) exist. This would not be such a difficult judgment call to make if the treatment were not so disfiguring, dramatic, and riddled with such severe possible unwanted side effects and complications. In other words, one needs to treat a condition that does not exist (but what the patient may be at high risk for and even have some soft symptoms, signs, and positive special tests for) with a devastatingly disfiguring surgery riddled with possible complications.

To illustrate this quagmire better, one can use a different example. In this example, you are a woman, and your mother, sister, and grandmother all had breast cancer. You are therefore at high risk of developing breast cancer, but you do not yet have it. The only way to prevent your body from developing breast cancer is to do prophylactic bilateral mastectomies. This is harsh and disfiguring (and preventative). However, in the case of breast cancer, one has the luxury of time and extensive psychological, genetic, surgical, and other counseling. In the case of ACS, however, this luxury does not exist and one has only minutes to make the decision to proceed with the disfiguring surgery.

The bad news is that clinical evaluation and compartmental pressure measurements are close to useless, and in the humble opinion of this author, pressure measurement is merely a waste of valuable time. The good news is that there may be some superior and more specific tests on the horizon that are undergoing evaluation at present in our institution and others. Johnstone and coworkers have recently shown that to achieve a sensitivity of 95%, an absolute compartment pressure of >30 mmHg was 30% specific for ACS, whereas a compartmental delta pressure of < 33 mmHg was 27% specific for ACS.[8] This same group, who based their study on the work of KGB Elliott from the University of Aberdeen in the United Kingdom,[9] also demonstrated that for the same sensitivity of 95%, an intramuscular pH of 6.38 was 80% specific for the early diagnosis of ACS.[8] They concluded that intramuscular pH monitoring radically outperformed both the highest intracompartmental pressure and the lowest intracompartmental delta pressure. If pH change is the cause of the sensation of ischemic pain, then it seems reasonable to assume that pH measurement could replace the near-impossible measurement and interpretation of "pain out of proportion to what is expected."

This development holds great promise, and this author foresees for the near future that a continuous intramuscular pH measuring microprobe will be developed that can be placed in the threatened compartments of all patients at high risk of developing ACS. Other monitoring techniques such as near-infrared spectroscopy, which measures deep tissue oxygenation, and pulsed phase-locked ultrasound, which can analyze fascial displacement, also hold some limited promise. It seems, however, that intramuscular pH monitoring may prove to be the least cumbersome, most cost-effective, and most accurate to use routinely for the early diagnosis of ACS.

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