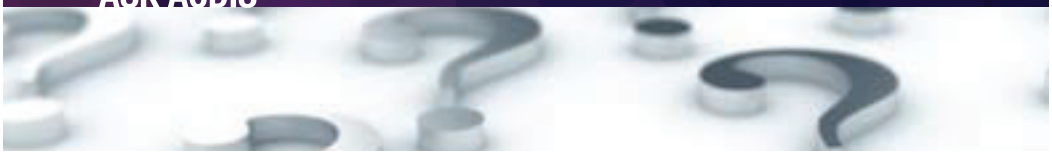

Sepsis FAQs

Q: I've heard that, due to the inflammatory response related to septic tissue, perfusion will decrease, and hyperlactatemia and mottling are likely if left untreated. Decreased perfusion means that organs/tissues would get less blood. Is that how mottling (discoloration in irregular areas) happens?

A: Mottling is the observable manifestation of decreased tissue health in the visible skin. This is also considered a possible indicator of shock. The decrease in oxygen utilization is not limited to the organs; the skin will be affected as well.

Q: I know that hyperlactatemia means increased levels of lactic acid in the blood, but how is it related to the decreased perfusion?

A: As the tissues become hypoxic, there is a switch to anaerobic cycle and function without oxygen in order to burn glucose and stay alive. This process is nearly identical to what happens with strenuous physical activity in skeletal muscle.

The process is much worse in septic patients, as it is not limited to overworked muscles but happens in the vital organs as well. If you remember the Krebs cycle in school, this process is referred to as anaerobic glycolysis. Well, that's the HISTORICAL viewpoint.

Increased lactate accumulation can happen even with adequate oxygen to the tissue. For this reason, the concept of "blood flow to the tissues" is incorrect. It isn't about blood perfusion; it's about oxygen utilization and cellular functioning. To demonstrate the point, even the lungs are producing lactate during sepsis, and they have full access to oxygen.

So, what's happening then? Essentially, the inflammatory response produces endogenous epinephrine and stimulates the beta-2 adrenergic receptors. This, in turn, increases glycolysis and generates a byproduct known as pyruvate used by the mitochondria in excess. The excess pyruvate is then converted directly into lactate, and voilà—excess lactate production. The entire process occurs in an entirely aerobic state.

Increased lactate may even be a positive compensatory mechanism that helps fuel cardiac and neurological processes, but the presence of significant elevations in some cases is a sign that the patient is severely ill. Remember, however, that lactate is not a waste product at normal levels; it is an important metabolic cofactor necessary for cellular function.

This is why occult shock is absolutely a thing and can occur in the presence of both adequate oxygenation and adequate perfusion—as opposed to the Sepsis-3 definition, which only defines shock as hemodynamic instability. These patients are having a catecholamine-dependent shock state, not dissimilar to a shock state supported with norepinephrine or vasopressors.

Patients with sepsis-induced hypotension actually have a better prognosis in many cases than patients in septic shock with lactic acidosis; therefore, the elevated lactate levels signal the increased catecholamine release and serve as a useful marker of both sepsis and septic shock.

There is even some criticism that the Sepsis-3 cutoff for a lactate level of 4 being an indicator of shock is somewhat arbitrary, considering that elevations of lactate have continuous curvilinear association with mortality.

Keep in mind that there are many other known physiological mechanisms that will artificially elevate lactate but have nothing to do with sepsis or shock (normal production but poor lactate clearance, organ failure preexisting or unrelated to any shock state, for example, abnormal metabolic process from neoplastic tissue, sampling and collection

errors, ARDS, ischemic or infarcted tissue, bowel compromise, muscle fatigue from increased work of breathing, etc.)

The concept of "elevated lactate = give more volume and oxygen" may at best not be very helpful and at worst actually cause harm, assuming a reasonable amount of fluid is already on board. The elevated lactate may instead be signaling that we do not have adequately functioning liver and kidneys, or that there is tissue damage occurring due to concomitant processes that are not necessarily related to volume and perfusion.

If you'd like to take a deeper dive into lactate in sepsis, I'd recommend *this article from PulmCrit.*

Q: How should I present this type of information to providers? I get intimidated by providers very easily (because there is so much clinical stuff that I do not know). What would be the best way to present this type of information?

A: There is no need to be intimidated. Providers are already very well aware of the standard deviations for procalcitonin, the significance and values of elevated lactate levels, etc.

All you need to do is present the clinically indicated and relevant abnormal values in a coherent way with a cogent and compliant query. The physician, upon seeing that you chose relevant data points and asked a reasonable question, should (usually) respond in kind with an appropriate clinical judgment.

Editor's note: Allen Frady, RN, BSN, CCDS, CCS, CRC, CDI education specialist for HCPro in Middleton, Massachusetts, answered these questions. Contact him at *AFrady@hcpro.com*. For information regarding CDI Boot Camps, *click here*.