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Trend Alert: The trending and interpretation of vital signs

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Edward T. Dickinson, MD, NREMT-P, FACEP
Kirkland N. Lozada, BA, NREMT-B

This clinical review feature article is presented in conjunction with the Department of Emergency Medicine Education at the University of Texas Southwestern Medical Center, Dallas.

Learning Objectives

>> Identify national prehospital training standards for obtaining vital signs during the initial assessment and reassessment of unstable patients, and for patients undergoing ALS procedures.

>> List normal parameters for adult vital signs to include respiratory and heart rates, as well as systolic and diastolic blood pressures.

>> Describe normal and abnormal vital sign physiology and pathophysiology to include: cardiac output, stroke volume and vascular resistance.

>> List conditions that increase and decrease blood pressure, heart rate and respiratory rate.

>> Explain the terms trending and linked reassessment as they relate to vital signs and patient assessment.

Key Terms

Peripheral vascular resistance (PVR): The resistance that occurs in blood vessel walls as blood moves through them. Muscles lining blood vessels allow them to dilate (lowering resistance and blood pressure) and constrict (increasing resistance and blood pressure) to maintain perfusion and balance.

Trending vital signs: The use of more than one set of vital signs to detect changes or "trends" in patient conditions.

Linked reassessment: Reassessment of the patient based on vital sign changes, or reassessment of vital signs because of changes or new complaints from the patient.

Vital signs are the fundamental objective data collected on all patients by medical personnel. At a glance, review of the initial heart rate, blood pressure and respiratory rate gives you an immediate sense of whether your patient is "sick" or "stable." Patients with marked abnormalities in their vital signs, such as rapid heart rate or very low blood pressure, provide obvious, immediate patient information. However, the practice of taking several sets of vital signs and looking for **trends** within them is often overlooked by EMS providers. The subtle changes in vital signs that can be observed over time are a critical part of the ongoing assessment.

Case Presentation

You work as an EMT for a rural BLS rescue squad and respond to a motor-vehicle crash. Upon arrival, you and your partner find a 53-year-old male who has driven his car into a telephone pole. You're able to extricate the patient via the passenger-side door. The patient is visibly intoxicated with slurred speech. Initial vital signs are BP 120/64, pulse 100, respirations 18 and ambient SpO 99%.

The physical exam reveals no obvious external signs of trauma, his lung sounds are clear, and there's no evidence of abdominal tenderness or distension. The patient repeatedly states, "I'm fine," and denies any specific pain. His neurological exam is normal, and he has a Glasgow Coma Scale (GCS) score of 15. You board and collar the patient and load him into the ambulance to begin the 30-minute transport to the nearest hospital.

About 15 minutes into the transport, the patient reports increasing abdominal pain. His repeat vital signs are: BP 110/84, pulse 130, respirations 26, SpO 99%. Your re-exam of his abdomen finds diffuse tenderness and mild rigidity that you didn't appreciate when you initially examined him. You now consider the patient unstable based on his significantly increased heartbeat and newly presenting abdominal findings.

You place the patient on high-flow oxygen, request an ALS intercept and begin taking vital signs at five-minute intervals. In addition, you notify the receiving hospital that you now have a potentially unstable trauma patient.

The ALS unit meets your ambulance seven minutes later, just as you complete your third set of vitals. His heart rate continues to elevate—now at 144. His blood pressure is now 82/palpation, his respirations are 30, and his skin has become cold and moist to the touch. He's now difficult to communicate with and losing consciousness. The medic places the patient on a cardiac monitor and barely has time to establish a large-bore IV before arrival at the hospital, where a general surgeon has already responded to the emergency department (ED), and an operating room has been placed on standby. Within 12 minutes of arrival, the patient is taken to the operating room, where he's found to have more than two liters of blood in his abdominal cavity. His shattered spleen is surgically removed, and then he survives.

Discussion

National EMT prehospital training standards require providers to obtain a baseline set of vital signs as part of the initial assessment, and subsequent sets of vital signs as part of patient reassessment—every 15 minutes in stable patients and every five minutes in unstable patients. In addition, ALS providers are traditionally trained to obtain repeat vital signs after each ALS intervention, such as a drug administration.

Because most of our patients' initial (aka, "baseline") vital signs are within normal limits, we tend to become complacent about obtaining and **trending vital signs.** Sometimes emergency medical providers even fabricate vital signs (especially respiratory rates) rather than actually taking the time to properly obtain additional sets of accurate vital signs.

Physiology

What constitutes "normal" adult vital signs is a matter of some debate and must always be viewed in the context of each patient. A systolic blood pressure of 92 mmHg in a 100-pound female athlete may be a normal value for her, whereas the same blood pressure in a 300-pound male with a history of hypertension would clearly be an abnormal value.

Generally accepted "normal" vital sign parameters in adults are as follows:

Normal Respiratory Rate 12 to 20 breaths/minute

Normal Heart Rate 60 to 100 beats/minute

Normal Systolic

Blood Pressure 90 to 150 mmHg

Normal Diastolic

Blood Pressure 60 to 90 mmHg

Understanding the basic physiology and pathophysiology that determine vital signs makes interpretation of vital sign data easier. The two most intertwined of the vital signs are heart rate and blood pressure. This relationship is the result of two key physiologic equations:

1.**Cardiac Output**= Heart Rate X Stroke Volume (the amount of blood the heart pumps out with each beat)

2.**Blood Pressure**= Cardiac Output X Vascular Resistance of the body's blood vessels

In both these equations, the body strives to maintain a constant cardiac output and a constant blood pressure. If the stroke volume falls in such situations as blood loss from trauma, the body will mobilize the endocrine and sympathetic nervous systems to elevate the heart rate and maintain cardiac output.

Ultimately, the body can only raise the heart rate so far to compensate (to perhaps 140 or 150 bpm in the adult), and cardiac output will begin to fall as blood loss continues. As cardiac output falls, the body will strive to maintain blood pressure by increasing the **peripheral vascular resistance (PVR).** PVR is also increased by stimulation of the endocrine and sympathetic nervous systems and the release of vasoactive chemicals, such as epinephrine and norepinephrine.

Increased PVR manifests in several ways. Because the diastolic blood pressure is dependent on PVR, an initial elevation in the diastolic blood pressure may be seen, as it was in our case example. However, as hypoperfusion (shock) progresses, the increased PVR results in such classic shock findings as decreased blood flow to skin, which results in pale, cool and clammy skin seen on examination. Ultimately, as all these compensations fail in the face of continued blood loss, the blood pressure will eventually fall.

Respiratory rate is controlled by the brain stem, which varies the rate of breathing in response to multiple inputs, including levels of carbon dioxide and the acid base balance (measured in pH) of the body. Increased respirations are a normal part of exercise physiology and provide needed oxygen delivery (and carbon dioxide removal) for the increased metabolic demands of the heart and muscles. Although simple hyperventilation syndrome can also cause an increased respiratory rate, it should be considered only when other, more serious causes have been excluded.

In emergency situations, tachypnea (an elevated respiratory rate) is a very important indicator of potential critical illness, especially shock. Because increased respirations serve the dual role of delivering more oxygen and removing more carbon dioxide, increasing the respiratory rate is the body's primary mechanism for trying to correct metabolic acidosis seen in shock. Metabolic acidosis occurs during shock because of reduced oxygen delivery to cells. This occurs when the body's cells, which are starved of oxygen, undergo anaerobic (rather than normal aerobic) metabolism and produce lactic acid as a byproduct. Lactic acid in the blood lowers the patient's pH and causes the respiratory rate to increase by triggering pH sensors in the brain stem.

An increased rate of breathing causes the body to develop respiratory alkalosis (high pH) to buffer metabolic acidosis (low pH) by "blowing off" carbon dioxide. In addition, increased respirations can serve to increase oxygen delivery to distressed cells (an effect enhanced when supplemental oxygen is administered).

Be aware that heart rate, respiratory rate and blood pressure can be affected by a wide range of conditions commonly encountered in emergency patients (See Table 1, April *JEMS* 2010,p. 68.)

Critical Concepts

Two critical concepts are key in the interpretation of all vital signs: trending and **linked reassessment.** First, as already mentioned, trending of vital signs allows you to see if the patient changes (or doesn't change) over time. The second critical concept is to always link a change in vital signs or patient complaint to a physical reassessment of the patient. For example, a standard 15-minute reassessment of vital signs reveals an abnormally slow heart rate that wasn't present in the baseline vitals. This prompts you to re-examine the patient, and she reports a new complaint of lightheadedness. Conversely, during transport, a patient tells you she's suddenly feeling lightheaded. This prompts you to obtain a new full set of vital signs, and you discover her heart rate has dropped from 55 to 45.

Return to the Case

The first scenario demonstrates a classic deterioration of vital signs that's seen in the progression of hypovolemic shock. At the outset, it was clear that the patient had a mechanism of injury that placed him at risk for serious injury. His initial vital signs (BP 120/64, pulse 100, respirations 18) were "stable." However, his first repeat set of vital signs (BP 110/84, pulse 130, respirations 26), which were obtained as part of a linked reassessment when he began to complain of abdominal pain, were a clear indicator of his body attempting to compensate for ongoing blood loss. His subtle change in blood pressure from 120/64 to 110/84 revealed an increased diastolic pressure as his blood vessels constricted to increase his PVR and counter a falling cardiac output as his blood volume began to fall because of internal bleeding.

Even more obvious in his second set of vital signs was the fact that his heart rate had markedly increased in an attempt to compensate and maintain cardiac output in the face of blood volume loss.

The significant increase in the patient's respiratory rate on reassessment was also very concerning and consistent with his body's attempt to compensate for developing shock from uncontrolled intra-abdominal blood loss. Because of the developing hemorrhagic shock, his body was building up high levels of lactic acid, which stimulated his brain stem to increase respirations to counter the developing metabolic acidosis.

His final set of vital signs represents fully decompensated hypovolemic shock. His heart rate was maximized in the 140s, his stroke volume continued to fall, and his ability to clamp down peripherally (as evidenced by his cold and clammy skin and decreased mental status) was maximized. With these vital signs, he was hypotensive with a systolic blood pressure of 82 mmHg as he teetered on the edge of succumbing to his injuries.

This case study illustrates the importance of accurately recording and then taking a moment to interpret and reassess the patients' vital signs. This is important for rendering quality care and can't be overstated. If only one set of vital signs had been obtained in the trauma patient presented, the receiving hospital wouldn't have been as well prepared as they were to rapidly evaluate and definitively treat the patient, and save his life. **JEMS**

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