

Clinical Assessment of Hypovolemia

EBEM Commentators

Richard Sinert, DO

Mark Spektor, DO

From the Department of Emergency Medicine, SUNY-Downstate Medical Center, Brooklyn, NY.

0196-0644/\$-see front matter

Copyright © 2005 by the American College of Emergency Physicians.

doi:10.1016/j.annemergmed.2004.09.021

[Ann Emerg Med. 2005;45:327-329.]

SYSTEMATIC REVIEW SOURCE

This is a rational clinical examination abstract, a regular segment of the *Annals'* Evidence-Based Emergency Medicine (EBEM) series. Each segment features an abstract of a rational clinical examination review from the *Journal of the American Medical Association* and a commentary by an emergency physician knowledgeable in the subject area.

The source for this rational clinical examination review abstract is: McGee S, Abernethy WB 3rd, Simel DL. The rational clinical examination. Is this patient hypovolemic? *JAMA*. 1999;281:1022-1029. The *Annals'* Evidence-Based Emergency Medicine editors assisted in the preparation of the abstract of this rational clinical examination review as well as selection of the Evidence-Based Medicine Teaching Points.

OBJECTIVE

This article reviews and summarizes the value of the clinical examination in detecting the presence of hypovolemia in adult patients.

DATA SOURCES

The authors report a MEDLINE search from January 1966 to November 1997 as their single data source. The search strategy was limited to the English language and included only studies involving humans aged older than 16 years. The search strategy consisted of 3 parts, appeared to include all relevant terms, is fully described, and is reproducible. Once articles were retrieved, their bibliographies were searched for additional source material.

STUDY SELECTION

Studies were included that described the operating characteristics of the clinical examination for the diagnosis of hypovolemia in adults. The studies that were included are of 2 types: physiologic studies of healthy volunteers before and after phlebotomy of standardized volumes of blood, and studies of patients presenting to the emergency department (ED) with suspected hypovolemia resulting from vomiting, diarrhea, or decreased oral intake. Patients with blood loss from traumatic injuries were not included.

DATA EXTRACTION AND ANALYSIS

Studies of patients without predetermined blood loss were graded by the authors on the basis of blinding, number of patients, and criteria used to define hypovolemia. The highest grades were given to studies that used specific criteria such as increased blood urea nitrogen–creatinine ratio or elevated serum osmolality to define hypovolemia. Studies were not reviewed that defined hypovolemia by invasive hemodynamic monitoring (central venous pressure or pulmonary capillary wedge pressure), oxygen debt measurement (arterial lactate), or dilution techniques (dye or radioactive tracer). Pooled sensitivities, specificities, and likelihood ratios with confidence intervals (CIs) were calculated using a random-effects model.

MAIN RESULTS

Results are divided into 3 sections: baseline data from normovolemic subjects, data from phlebotomized individuals, and studies of ED patients without blood loss but suspected of hypovolemia resulting from dehydration (vomiting, diarrhea, and decreased intake).

Using postural vital sign data from 3,500 normovolemic patients, mean changes in vital signs with standing included an increase in pulse rate of 10.9 beats/min (95% CI 8.9 to 12.8 beats/min), a decrease in systolic blood pressure of 3.5 mm Hg (95% CI -1.5 to 5.5 mm Hg), and an increase in diastolic blood pressure of 5.2 mm Hg (95% CI 2.8 to 7.6 mm Hg). Pulse rate changes stabilized after 40 to 60 seconds; changes in blood pressure stabilized after 1 to 2 minutes. Postural hypotension (decrease in systolic blood pressure of >20 mm Hg) was found in 10% of normovolemic adults (<65 years) in 1 study and in 11% to 30% of elderly subjects (>65 years) in a number of studies cited by the authors. Capillary refill in normovolemic subjects was 2, 3, and 4 seconds (after 5-second compression of the middle phalanx) for children, adult females, and adult males, respectively.

In phlebotomized healthy subjects, the volume of blood loss was graded as moderate (450 to 630 mL) and large (630 to 1,150 mL) (Table). A "positive postural pulse increment" of 30 beats/min had a sensitivity of 22% (95% CI 6% to 48%) after blood loss of 450 to 630 mL but improved to a sensitivity of 97% (95% CI 91% to 100%) and a specificity of 98% (95% CI 97% to 99%) after blood loss of 630 to 1,150 mL. Postural decrease in systolic blood pressure of more than 20 mm Hg after

moderate blood loss had a sensitivity of 9% (95% CI 6% to 12%) in younger subjects and 27% (95% CI 14% to 40%) in elderly subjects (>65 years). There were insufficient studies to calculate operating characteristics of postural hypotension with higher volumes of blood loss. Mild postural dizziness was a poorly sensitive (22%) indicator of postural hypotension in most studies, although severe dizziness (inability to stand) was highly specific (98%). Postural vital signs in patients with suspected hypovolemia resulting from dehydration showed similar operating characteristics to those with fixed volume blood loss.

Supine tachycardia had a high specificity; 96% (95% CI 88% to 99%) of healthy volunteers had a pulse rate of less than 100 beats/min when supine. However, only 12% (95% CI 5% to 24%) of the same volunteers had a pulse rate of greater than 100 beats/min after 630 to 1,150 mL of blood was removed by means of elective phlebotomy. One study showed a significant correlation between bradycardia and volume of blood loss.¹ Supine hypotension (systolic blood pressure <95 mm Hg) was also highly specific (97%) but insensitive for moderate blood loss (13%) and large blood loss (33%).

In studies of patients presenting to EDs with suspected hypovolemia not due to blood loss, no individual physical examination finding appeared to be extremely useful. Most of these studies involved elderly populations. Changes in postural vital signs, whether present or absent, had likelihood ratios near 1, indicating that such changes occur with essentially equal frequency in patients judged to be positive or negative for hypovolemia. However, the absence or presence of multiple findings such as sunken eyes, furrowed tongue, dry mucous membranes, and extremity weakness correlated fairly well with the absence or presence of elevated serum urea nitrogen–creatinine ratios. Delay in capillary refill, when present, had a likelihood ratio of 7 in 1 study on dehydration, although postural changes in vital signs (an extremely poor predictor of dehydration) was used as the criterion standard. In patients with blood loss, capillary refill carried a similarly high specificity (95%) and low sensitivity (34%).

CONCLUSION

When evaluating patients for hypovolemia, postural tachycardia and the inability to stand from postural dizziness are helpful signs, whereas supine hypotension, supine tachycardia, capillary refill, and skin turgor have no proven diagnostic role. Because the clinical examination has such low sensitivity for detecting hypovolemia, the authors conclude that a low threshold for ordering confirmatory laboratory tests (serum urea, creatinine, osmolality and/or sodium) is warranted.

Rational Clinical Examination Author Contact

Steven McGee, MD

University of Washington

Seattle-Puget Sound VA Health Care System

Seattle, WA

E-mail smcgee@u.washington.edu

Table. Operating characteristics of vital signs in detecting hypovolemia.

Physical Examination Finding	Sensitivity/ Specificity, %	Positive LR (95% CI)	Negative LR (95% CI)
Large blood loss			
Postural pulse increment >30 beats/min	97/98	48.5	0.03
Supine tachycardia*	12/96	3.0	0.9
Supine hypotension†	33/97	11.0	0.7
Moderate blood loss			
Postural hypotension‡ (age ≤65 y)	9/94	1.8	1.0
Postural hypotension‡ (age ≥65 y)	27/86	1.9	0.9
Postural pulse increment >30 beats/min	22/98	11.0	0.8
Supine tachycardia*	0/96	NA	NA
Supine hypotension†	13/97	4.3	0.9
Dehydration			
Postural pulse increment >30 beats/min	43/75	1.7 (0.7–4.0)	0.8 (0.5–1.3)
Postural hypotension	29/81	1.5 (0.5–4.6)	0.9 (0.6–1.3)
Mucous membranes dry	85/58	2.0 (1.0–4.0)	0.3 (0.1–0.6)
Tongue dry	59/73	2.1 (0.8–5.8)	0.6 (0.3–1.0)
Furrowed tongue	85/58	2.0 (1.0–4.0)	0.3 (0.1–0.6)
Sunken eyes	62/82	3.4 (1.0–12.2)	0.5 (0.3–0.7)
Confusion	57/73	2.1 (0.8–5.7)	0.6 (0.4–1.0)
Extremity weakness	43/82	2.3 (0.6–8.6)	0.7 (0.5–1.0)
Nonfluent speech	56/82	3.1 (3.2–14.9)	0.7 (0.5–0.9)

LR, Likelihood ratio; NA, not available due to 0% sensitivity.

*Pulse rate >100 beats/min.

†Systolic blood pressure decrease of <95 mm Hg.

‡Systolic blood pressure decrease of >20 mm Hg.

COMMENTARY: CLINICAL IMPLICATION

For the emergency physician, vital signs mark the beginning, middle, and end of each case. Rapid changes in patient conditions and the rapid pace of ED care mandate that emergency physicians be well versed in the indicators of evolving emergency conditions. Although the criterion standard markers used by studies examining non–blood loss hypovolemia (blood urea nitrogen–creatinine ratio, osmolality, serum sodium) are themselves unproven, the authors argue that abnormal vital signs were helpful for ruling in hypovolemia, and unreliable for ruling it out.

If we consider healthy phlebotomized individuals as models for young, previously healthy patients sustaining acute traumatic injuries, we may infer some useful information from these studies. Most significantly, the poor sensitivity of vital signs or postural changes for blood loss is notable and is consistent with studies demonstrating that both young and elderly trauma patients frequently maintain normal vital signs in the face of significant oxygen debt after large blood loss.^{2,3} Postural vital signs may be of little use except as a potentially sensitive marker of major blood loss in otherwise healthy patients with isolated hemorrhage (generally trauma patients, who are rarely asked to stand for postural vital sign examination). A more direct and

more valuable indicator than vital signs in hemorrhagic shock appears to be anaerobic respiration, with initial elevations of lactate and base deficit demonstrating high predictive values for both blood loss and mortality.^{4,5} The high specificity of vital signs for hypovolemia in the controlled setting of standardized phlebotomy cannot be reliably translated to an ED population given the various alternative explanations for tachycardia in the ED (eg, pain, anxiety, drug use).

When considering studies evaluating the determination of dehydration (as judged by elevated blood urea nitrogen–creatinine ratio), the predictive value of the clinical examination is unhelpful when considering only individual findings, but helpful when findings are considered cumulatively. The presence or absence of multiple findings such as dry mucous membranes, confusion, extremity weakness, sunken eyes, furrowed tongue, dry tongue, and nonfluent speech are all helpful. In moderate to severely elevated blood urea nitrogen–creatinine ratios, on average 4 to 6 of these findings were present, whereas in those without elevation only 1 was typically present. Therefore, significantly dehydrated patients and those without significant dehydration are often recognizable as such by performing a thorough examination. Vital signs were remarkably insensitive for various surrogate markers of dehydration, and the various alternative explanations for vital sign changes among elderly ED patients (eg, infection and sepsis, chronic and acute comorbidities, anxiety, injury, pain) render the specificities unreliable in our setting.

TAKE HOME MESSAGE

Traditional vital signs and their postural variants should be considered insensitive measures of hypovolemia. Although abnormal findings such as severe postural dizziness should increase the index of suspicion for hypovolemia, postural and static vital signs are normal in most hypovolemic and dehydrated study subjects. Vital signs such as supine hypotension are generally insensitive to all but large amounts of blood loss exceeding 1,000 mL (likelihood ratio=11). Physical examination markers traditionally thought to be associated with dehydration (eg, dry membranes, sunken eyes) appear to be helpful when multiple positive findings are present. Overall, clinical judgment may remain the best marker we have for determining volume status.

EBEM Commentator Contact

Richard Sinert, DO
 Department of Emergency Medicine
 SUNY-Downstate Medical Center
 Brooklyn, NY
 E-mail nephron1@bellatlantic.net

EVIDENCE-BASED EMERGENCY MEDICINE TEACHING POINT

Interpretation of likelihood ratios. The likelihood ratio is a statistical combination and representation of the sensitivity and specificity of a diagnostic test. Specifically, a positive likelihood ratio is the ratio of the percentage of true positives

(ie, sensitivity) found using a given test to the percentage of false positives (ie, 1–specificity). If 90% of positive test results were true positives and 10% were false positives, the positive likelihood ratio would be 90:10 (9.0). This tells us that when a positive test result is found, it is 9 times more likely that the disease is present than that the disease is absent. A negative likelihood ratio is the ratio of the percentage of false negatives (ie, 1–sensitivity) to the percentage of true negatives (ie, specificity). Because the false value is in the numerator and the true value is in the denominator, the smaller the negative likelihood ratio the better the test. If 9% of the negative test results were false negatives and 91% were true negatives, the negative likelihood ratio would be 9.91, or approximately 0.1. This would tell us that when a negative test result is found, it is only one tenth as likely that the disease is present than that it is absent (or, said another way, that it is 10 times as likely that the disease is absent as that the disease is present). Generally, a positive likelihood ratio greater than 10 is almost conclusive of the presence of disease and a negative likelihood ratio less than 0.1 usually excludes the disease. A likelihood ratio of 1.0 is useless, signifying the test result failed to make any change from pretest to posttest probability of disease.

In a letter to the editor of *The New England Journal of Medicine* in 1975, Dr. Fagan⁶ developed a nomogram to quickly calculate posttest probability by connecting with a straight-edge pretest probability and likelihood ratio.

The likelihood ratio is therefore a complete statement about how a given diagnostic test may be used in clinical practice and offers a statistical probability of disease from a test result on which to base decisions. By convention, it is generally thought that a positive likelihood ratio of 10 and a negative likelihood ratio of 0.1 demonstrate excellent performance in a diagnostic test and allow its integration into decisionmaking as a helpful single marker for the presence or absence of disease. However, significantly less extreme likelihood ratios may be considered to be helpful when a test is used as one of multiple adjuncts to clinical decisionmaking.

Publication dates: Available online January 11, 2005.

REFERENCES

1. Sander-Jensen K, Secher NH, Bie P, et al. Vagal slowing of the heart during haemorrhage: observations from 20 consecutive hypotensive patients. *Br Med J (Clin Res Ed)*. 1986;292:364-366.
2. Abou-Khalil B, Scalea TM, Trooskin SZ, et al. Hemodynamic responses to shock in young trauma patients: need for invasive monitoring. *Crit Care Med*. 1994;22:633-639.
3. Scalea TM, Simon HM, Duncan AO, et al. Geriatric blunt multiple trauma: improved survival with early invasive monitoring. *J Trauma*. 1990;30:129-136.
4. Davis JW, Parks SN, Kaups KL, et al. Admission base deficit predicts transfusion requirements and risk of complications. *J Trauma*. 1996;41:769-774.
5. Husain FA, Martin MJ, Mullenix PS, et al. Serum lactate and base deficit as predictors of mortality and morbidity. *Am J Surg*. 2003; 185:485-491.
6. Fagan TJ. Nomogram for Bayes theorem [letter]. *N Engl J Med*. 1975;293:257.