18th ANNUAL CONFERENCE
RIVER of DREAMS
envisioning best practices in trauma care
Learning Objectives

1. Better understand vital signs for what they can tell you (and what they can’t) in the assessment of a trauma patient.

2. Appreciate best practices in obtaining accurate vital signs in trauma patients.

3. Learn what teaching about vital signs is evidence-based and what is not.

4. Explain the importance of vital signs to more accurately triage, diagnose, and confidently disposition our trauma patients.

5. Apply the monitoring (and manipulation of) vital signs to better resuscitate trauma patients.
Disclosure Statement

• Faculty/Presenters/Authors/Content Reviewers/Planners disclose no conflict of interest relative to this educational activity.
Successful Completion

- To successfully complete this course, participants must attend the entire event and complete/submit the evaluation at the end of the session.
- Society of Trauma Nurses is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.
Vital Signs
Vital Signs

Philosophy:

“View vital signs as *compensatory* to the illness/complaint as opposed to *primary*.”

Vital Signs

Truth over Accuracy:

• Document the true status of the patient: sick or not?

• Complete vital signs on every patient, every time, regardless of the chief complaint.

• If vital signs seem misleading or inaccurate, repeat them!

• Beware sending a patient home with abnormal vitals (especially tachycardia)!

• Treat vital signs the same as any other diagnostics—review them carefully prior to disposition.
The Mother’s Vital Sign: Temperature
Case #1 - 76-y/o homeless ♂

CC: 76-y/o homeless ♂ brought to the ED by police for eval. Nighttime temps have been in the 50s-60s. He has been turned away from all shelters due to his chronic etOH use.

VS: T 94°F (oral), HR 96, RR 22, BP 107/97, SaO₂ 92%

PE: Disheveled, malnourished, and intoxicated. No visible injuries. GCS = E1 V4 M5. ⊕ “toxic sock syndrome.”
Case #1 - 76-y/o homeless ♂

T 94°F, HR 96, RR 22, BP 107/97, SaO₂ 92%

Does a GCS of 10 correlate with a core temperature of 94°F (34.4°C)?

No.

Any patient with a temperature ≥ 90°F (32°C) with ongoing AMS needs further workup as to why: intoxication, trauma, infection, etc.

Could a temporal thermometer be used to give a true “core temperature” reading in this patient?  

No. Temporal thermometry “should not be used...in the setting of suspected hypothermia.” Rectal, Esophageal, or Foley catheter thermister is recommended.

Temporal thermometry in adults?

World Journal of Emergency Medicine, 2013

Retrospective chart review of 27,130 adult patients in a high-volume ED over 8 years.

The average difference between the initial temporal artery thermometry (n = 988) and the rectal temp was 1.2° F (0.7°C) (p<0.001).

In almost 1 in 5 patients (18.8%), fever was missed by the initial triage temp.
Temporal thermometry in Trauma?


### TABLE 1

<table>
<thead>
<tr>
<th>Comparison (vs oral)</th>
<th>N</th>
<th>Mean Δ* (°F)</th>
<th>95% CI for Δ*</th>
<th>Percentage with Δ (&gt;0.5°F)</th>
<th>Fever agreement† (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TA, forehead and ear</td>
<td>52</td>
<td>0.27</td>
<td>-2.13 to 2.66</td>
<td>60.6</td>
<td>90.4</td>
</tr>
<tr>
<td>TA, forehead only</td>
<td>56</td>
<td>-0.56</td>
<td>-2.65 to 1.54</td>
<td>60.9</td>
<td>94.6</td>
</tr>
<tr>
<td>TA, ear only</td>
<td>50</td>
<td>-0.26</td>
<td>-2.79 to 2.26</td>
<td>65.6</td>
<td>94.0</td>
</tr>
<tr>
<td>Axillary</td>
<td>57</td>
<td>0.03</td>
<td>-1.97 to 2.03</td>
<td>55.4</td>
<td>89.5</td>
</tr>
</tbody>
</table>

*The average difference in temperature between the 2 methods across all patients. For each comparison, the oral calibrated temperature was subtracted from the temperature determined by the alternative method.

*The range in magnitude of differences that apply to approximately 95% of the patients in the study.

†Fever was defined as an oral temperature ≥101.0°F.
Temporal thermometry in the ICU?

*Tidsskr Nor Laegeforen, 2003*

Comparison study of 164 ICU patients between rectal and TAT measurement.

Fever was a rectal temp $\geq 100.4^\circ F \ (38^\circ C)$; TAT detected fever in 33 of 70 febrile patients. $Sn = 53\%$

Conclusion: TAT’s sensitivity for detecting rectally measured fever is too low to recommend its use in adult ICU patients.

Temperature

Temporal thermometry in adults?

Temporal thermometry in Trauma?

Temporal thermometry in the ICU?
Case #2 - Two ♂s from winter MVC

EMS calls for medical control after responding to a single-car rollover off the side of the road. The driver was found up the road about a mile. The passenger is trapped inside due to significant compartment intrusion, his chest pinned between the dash and the seat. Wind chill is -40°F & C.

The passenger is much warmer than the driver due to being inside the car, but has absent vital signs. The driver has no signs of injury, but as he is dressed in a T-shirt, jeans, and tennis shoes with no socks, and also has no vital signs.

EMS has a transport time of 40 minutes and as they are a two-person crew, can only resuscitate one person.
Case #2 - Two ♂s from winter MVC

Which patient should EMS transfer and attempt to resuscitate: the driver with apparent hypothermia, the passenger still trapped inside the relatively warm car, both, or neither? The driver.

Successful resuscitation following blunt trauma is approximately 1-2%. Primary hypothermia (even in the face of cardiopulmonary arrest) has a resuscitation chance of approximately 50%

Case #2 - 21-y/o ♂ found outside

EMS: The driver is placed on a cardiac monitor.

PE: Bradycardia rate of 24 bpm is seen with a widened QRS, but no pulse can be palpated. The patient is apneic. They have no thermometer on their rig. SaO2 cannot capture a waveform.
Case #2 - 21-y/o ♂ found outside

Temp ??, HR 24, RR Ø, BP Ø, SaO₂ Ø

EMS asks how should the patient’s airway be managed as they don’t want to do something that might change the pulseless bradycardia to V-fib.

Intubate the patient. Hypothermic patients are at ↑d risk for aspiration. Intubation facilitates warmed humidified air administration and has NOT been shown to cause dysrhythmias in hypothermia.

Case #2 - 21-y/o ♂ found outside
Temp ??, HR 24, RR Ø, BP Ø, SaO₂ Ø

EMS asks, “Should they start CPR on this patient en route to your facility?”

YES!

He has no palpable pulse! CPR will perfuse the brain until definitive resuscitation is available.

What treatment should be initiated to treat the patient’s bradycardia: atropine, external pacing, dopamine, epinephrine, or something else?

Treat the hypothermia. Bradycardia is due to ↓d spontaneous depolarization of pacemaker cells. Atropine will be ineffective. Cold myocardium is irritable, and external pacing may convert an organized rhythm into V-fib.
Case #2 - 21-y/o ♂ found outside

Temp ??, HR 24, RR Ø, BP Ø, SaO₂ Ø

EMS asks how should they treat the hypothermia while en route?

Prevent further heat loss.
Dry and cover the patient.
Turn up the heat and administer warm IVF and warmed/humidified O₂ if possible.

ED: The patient arrives and is placed on a cardiac monitor.

PE: Asystole is seen. The patient is apneic. Pupils are fixed and dilated. Nursing staff reports to you a rectal temperature of 82° F. No blood pressure (obviously) and no signs of trauma on your secondary survey.
Case #2 - 21-y/o ♂ found outside

T 82° F (28°C), HR Ø, RR Ø, BP Ø, SaO₂ Ø

What should be done now for your asystolic, apneic, Ø blood pressure, fixed/dilated pupil patient with a core temperature of 82° F (28°C)?

Initiate ECMO / CPB. Hypothermia patients with cardiac arrest who are treated at an ECMO/CPB Center have a 50% survival rate. Hypothermia patients with cardiac arrest who are treated without ECMO / CPB have a 10% survival rate.

How do you know if your patient is truly dead?
“They’re not dead until they’re warm and dead.”

Warm and Dead?

Warm adults to 32°C (90° F) before pronouncing.
Warm children to 35°C (95° F) before pronouncing.
A potassium (K+) level > 12 mEq/L and ammonia (NH₃) > 250 mmol/L are both reasons to stop resuscitation regardless of core temperature.

Nose or mouth occluded with ice (preventing ventilation) or a core temp < 15°C (59°F).

Obvious lethal injuries are a reason to cease resuscitative efforts.

A rigid, non-compressible thorax is also a reason to stop hypothermic resuscitation.

Frozen or clotted blood is a sign that the resuscitation should be stopped.

Signs of Irreversible Death
Dead or not?
Dead or not?
Hypothermia: Children vs. Adults

• Infant BSA:mass is 3x adult
• Child BSA:mass is 2x adult
• The large surface-area to body-mass ratio results in quicker heat loss for infants and children
• Faster cooling $\rightarrow$ cerebral protection from hypoxia-- even in submersion injury
Dead or Not?

Avalanche Burial

< 35 min. burial → NOT hypothermia

> 35 min. burial → possibly hypothermia
In hypothermia, these signs ≠ death!
Dead or not?
Key Facts: Temperature

A hypothermic patient warmed to 90° F (32°C) with ongoing AMS needs further workup as to why.

Temporal thermometry has been shown to be unreliable in the setting of suspected hypothermia, the ICU, in adults, and in trauma.

At temperatures < 86° F (30°C) anticipate cardiac arrest and initiate ECMO/CPB in appropriate patients (got cold...then died). Duration of CPR is not a predictor of survival in hypothermia.

Drowning (especially children), lightning strike, and hypothermia are all special circumstances that suggest a resuscitation attempt even in the face of apparent death.

SIRS Criteria is $T \geq 38^\circ C (100.4^\circ F)$ or $\leq 36^\circ C (96.8^\circ F)$, a HR > 90 bpm, a RR > 20 bpm, and a WBC > 12 or $< 4$ or $> 10\%$ bands.

Sepsis - 2 out of 4 SIRS criteria + Infection

Septic Shock - 2/4 SIRS criteria + Infection + hypoTN after IVF

Severe Sepsis - 2/4 SIRS criteria + Infection + Lactate > 4
The Paramedic’s Vital Sign: Pulse
CC: 28-y/o ♀ with bike vs. auto. Car passed her on left and clipped her handlebars with the side mirror causing her to crash. ☀ Helmet. Estimated speed was ~28mph.

Meds: MVI, OCPs, and Ibuprofen.

VS: HR 54, RR 18, BP 105/89, SaO₂ 100%

PE: Appears pale but in no acute distress. Pelvis is grossly unstable.
Case #3 - 28-y/o ♀ bike vs. auto
Afebrile, HR 54, RR 18, BP 105/89, SaO₂ 100%

Does this patient’s relative bradycardia effectively rule-out significant blood loss? No.

Paradoxical bradycardia has been reported in the literature in cases of massive blood loss.

Case #3 - 28-y/o ♀ bike vs. auto
Afebrile, HR 54, RR 18, BP 105/89, SaO₂ 100%

What is this patient’s pulse pressure?

What is a normal value?

What does an abnormal value imply?

16mm Hg

30 to 40mm Hg

Narrow pulse pressure in the setting trauma is suggestive of ↑↑↑↑ blood loss (↓ preload → ↓ CO).

Case #3 - 28-y/o ♀ bike vs. auto

Afebrile, HR 54, RR 18, BP 105/89, SaO₂ 100%

What does this patient’s SaO₂ of 100% suggest to you about her hemoglobin level?

It’s probably LOW. A low hemoglobin will → a falsely increased SaO₂. The degree of oxygen saturation is inversely related to the amount of hemoglobin present.

Pulse-oximetry & Hb

• Hemoglobin levels can affect pulse oximetry.

• ↓ Hb (anemia) → the easier it is to saturate Hb. The SaO2 may be high, but total oxygen content will be low.

• ↑ Hb (polycythemia) → difficult to saturate the extra molecules of Hb with oxygen → pseudo-hypoxemia (when the patient’s oxygen content may well be normal).

On arrival in the ED, the patient has normal femoral & carotid pulses bilaterally with diminished radial pulses. What does this tell you about her systolic BP?

Nothing.

The presence or absence of pulses does not consistently correlate with any given SBP.


## ATLS Classes of Hemorrhagic Shock

<table>
<thead>
<tr>
<th>Class of haemorrhagic shock</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss (mL)</td>
<td>Up to 750</td>
<td>750–1500</td>
<td>1500–2000</td>
<td>&gt; 2000</td>
</tr>
<tr>
<td>Blood loss (% blood volume)</td>
<td>Up to 15</td>
<td>15–30</td>
<td>30–40</td>
<td>&gt; 40</td>
</tr>
<tr>
<td>Pulse rate (per minute)</td>
<td>&lt; 100</td>
<td>100–120</td>
<td>120–140</td>
<td>&gt; 140</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>Normal or increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Respiratory rate (per minute)</td>
<td>14–20</td>
<td>20–30</td>
<td>30–40</td>
<td>&gt; 35</td>
</tr>
<tr>
<td>Urine output (mL/hour)</td>
<td>&gt; 30</td>
<td>20–30</td>
<td>5–15</td>
<td>Negligible</td>
</tr>
<tr>
<td>Central nervous system/mental status</td>
<td>Slightly anxious</td>
<td>Mildly anxious</td>
<td>Anxious, confused</td>
<td>Confused, lethargic</td>
</tr>
</tbody>
</table>


ATLS: Palpable pulses correlate with SBP

Radial pulse: SBP > 80 mmHg

Carotid & Femoral pulse only: SBP 70 - 80 mmHg

Carotid pulse only: SBP 60 - 70 mmHg

Vital signs and estimated blood loss in patients with major trauma: Testing the validity of the ATLS classification of hypovolemic shock

Resuscitation, May, 2011


In trauma patients there is an inter-relationship between derangements of HR, SBP, RR and GCS but not to the same degree as that suggested by the ATLS classification of shock.

<table>
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<th>Blood loss (% blood volume)</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
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<tbody>
<tr>
<td>Blood loss (mm Hg)</td>
<td>Normal or Increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>Normal or Increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>Normal or Increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Central venous pressure</td>
<td>Normal or Increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Mental status</td>
<td>Normal or Increased</td>
<td>Confused</td>
<td>Confused</td>
<td>Confused</td>
</tr>
</tbody>
</table>


Accurate of ATLS guidelines in predicting SBP using carotid, femoral, and radial pulses.

*BMJ*, September, 2000

Observer blinded assessment of 20 patients with hypovolemic shock and arterial lines.

The disappearance of pulse always occurred in the following order radial → femoral → carotid pulse.

Mean difference of actual and estimated BP using ATLS was 34 mmHg

Conclusion: ATLS guidelines that correlate pulse presence/absence with SBP in hypovolemic shock tend to overestimate the patient’s SBP.


Paradoxical bradycardia has been reported in the literature in cases of massive GI bleed as well as hypovolemic trauma.

A low hemoglobin will → a falsely increased SaO2. The degree of oxygen saturation is inversely related to the amount of hemoglobin present.

**Maximal heart rate** = (220 - patient’s age).

HR < 150 are usually due to disease / injury.

HR > 150 imply a primary cardiac dysrhythmia.
A heart range of 50-90 bpm has been proposed as the new normal for a healthy population.

Sinus tachycardia demonstrates variability. A constant tachycardia that does not vary suggests a cardiac dysrhythmia.

In trauma patients there is an inter-relationship between derangements of HR, SBP, RR and GCS but not to the same degree as that suggested by the ATLS classification of shock.
The Nurse’s Vital Sign: Blood Pressure
Case #4 – 32-y/o ♀ c/o “headache”

CC: 32-year-old ♀ presents to ED c/o “migraine headache.” She gets these 3 to 4 times / year. Trauma service is consulted b/c of a car accident three weeks ago. Denies any change in pattern, location, or intensity of HA.

PMHx: HAs. BTL.

VS: Afebrile, HR 86, RR 18, BP 182/104, SaO₂ 99%

PE: Normal neuro exam.
Case #4 – 32-y/o ♀ c/o “headache”

Afebrile, HR 86, RR 18, BP 182/104, SaO2 99%

How will lowering this patient’s blood pressure help her headache?

It won’t. Hypertension is likely not the cause of a headache in a patient with an otherwise normal neurologic exam.
Blood pressure

Hypertension & Headache

• Two large epidemiologic studies, the Nord Trøndelag Health Survey 1984-86 (HUNT-1) and 1995-97 (HUNT-2), to evaluate the association between BP (SBP, DP, MAP, PP) and migraine & nonmigrainous headache.

• ↑SBP and ↑PP were associated with ↓prevalence of both types of headache for both sexes.

Case #5 – 56-y/o ♀ c/o “dizzy”

CC: 56-year-old ♀ c/o syncope. She was at home when she fainted and fell down 5 stairs. As she meets criteria for “dangerous mechanism of injury,” the trauma service is consulted. Feels worse with standing and better when seated or supine.

PMHx: HTN on HCTZ.

VS: Afebrile, HR 94, RR 19, BP 168/97, SaO2 97%.

PE: Normal, ambulatory.
Case #5 – 56-y/o ♀ c/o “dizzy”
Afebrile, HR 94, RR 19, BP 168/97, SaO2 97%

What would orthostatic vital signs add to the work-up and management of this patient?
• Orthostatic vital signs are a non-invasive way to look for occult hypovolemia in a patient in whom you wouldn’t otherwise expect to find it.

• For example, a patient presenting with syncope.

• Positive orthostats = hypovolemia is a likely cause.

• Negative orthostats = something else → syncope.

• And now that you understand how they are supposed to work...
Orthostatic vital signs are neither sensitive nor specific for hypovolemia, and their presence or absence should not be used to explain this patient’s syncope.

- Orthostats may be seen in 23% of those younger than 60 years.
- Orthostats are present in up to 40% of asymptomatic patients older than 70 years.
- In frail elderly individuals living in nursing homes, the prevalence of orthostatic hypotension is 50% or higher.

Case #5 – 56-y/o ♀ c/o “dizzy”
98.6° F (37°C), HR 94, RR 19, BP 168/97, SaO2 97%

Bottom line?
Treat the patient to their symptoms.
Oral rehydration is substantially under-utilized in most EDs in the United States.
Cases #6 & 7 - 21- & 23-y/o ♂s MVC trauma

CC: 21-y/o ♂ s/p MVC. Unrestrained driver. + LOC. + Windshield spidering. Multiple facial lacerations. EMS fully immobilized.


Excluding blood products, the best resuscitation fluid (0.9NS or LR) is… Irrelevant. Initial fluid choice has not been shown to affect outcomes (morbidity or mortality) in trauma resuscitation.


An intubated patient with TBI should have an FIO2 of 100% maintained for how long post-injury?

As short as possible!

Once intubated, a TBI patient should have the FIO2 ↓ as quickly as possible, titrating to a PaO2 of 200 to 300mmHg.

Even normal oxygenation in the body can have hypoxia in the brain in TBI.

- Optimal care for the serious TBI patient is to keep SaO2 around 99 to 100% (PaO2 of 200 - 300 mmHg).
- An FIO2 of 50% will have a maximum PaO2 of around 350 mmHg.
- Hyperventilation appears to be even more harmful than hyperoxia--use the ETCO2 monitor to titrate between 35 and 40 mmHg.

What is the minimum target SBP for the motorcyclist (the patient with no evidence of TBI)?

For uncontrolled hemorrhage (in the absence of TBI), target resuscitation to a SBP between 70 and 90 mm Hg or normal mentation and palpable peripheral pulses.

Blood pressure

DCR: Damage Control Resuscitation

Hemorrhage → Fluid replacement → Inflammation → Tissue leakage → Hypotension

T/F – In patients with TBI, it has been shown that even a single episode of hypotension causes a doubling of mortality in this patient population. **TRUE.** Any treatment strategies that permit hypotension in patients with TBI are absolutely contraindicated.

Permissive hypotension... Damage control resuscitation... Delayed resuscitation... are all only for trauma patients without TBI!

None of these principles are applicable to hypotensive medical patients!
CC: 77-y/o ♀ c/o fatigue and malaise. Symptoms present “for months.” Denies pain. 
No travel history.

PMHx: Denies. Ø tobacco. Ø alcohol. Ø drugs.

VS: Temp 96.8° F (36°C), HR 52, RR 8, BP 86/51, SaO2 93%.
Case #8 - 77-y/o ♀ c/o fatigue & malaise

Temp 96.8° F (36°C), HR 52, RR 8, BP 86/51, SaO₂ 93%

Assuming a normal cardiac exam (EKG), peripheral pulses, circulating volume (CBC), and no underlying infection (UA & CXR), could these vitals be considered otherwise normal for this patient?

No. This patient has classic vital signs and physical appearance for hypothyroidism with likely progression to myxedema coma if left untreated.
Blood pressure

**Definition**

Arterial BP reflects the dynamic balance between cardiac output and peripheral vascular resistance.
What number of physiologic components determine a patient’s blood pressure?
Blood pressure

Five Components

△ Cardiac output (rate & contractility)
△ Circulating volume (hemorrhage, dehydration)
△ Vascular tone (neurogenic or inflammatory)
△ Vascular permeability (anaphylaxis, sepsis)
△ Endocrine system (hypothyroidism, adrenal tumor)
Blood pressure

**Hypotension: Definition**

**Adults**

SBP < 90 mm Hg

SBP ↓ by ≥ 40mm Hg from baseline

**Pediatrics**

SBP < 70mm Hg + [2 x age] from 0 - 10 yrs
Pediatric Vital Signs

Do we really need a blood pressure in kids less than 3 years of age?

• Blood pressure is a vital sign no matter the age of the child.

• Most pediatric cases “under review” are missing blood pressure on their medical record.


The day you’ve bantered about for years finally happens.
A busload of hemophiliacs crashes on their way to their annual meeting of the Organisation de Hemophlia et Société Hémoglobine de International Transfusion.

How can vital signs guide you as to who will need blood?

Case #9 – A bus crash...
Early recognition and management of hypovolemic shock remains one of the most challenging tasks in the initial assessment of trauma patients.

Isolated vital signs (SBP & HR) are limited in their ability to identify life-threatening hypovolemic shock.

The Shock Index (SI) correlates with the extent of hypovolemia in severely injured patients, as reflected by increased transfusion requirement, higher rates of massive transfusion, morbidity & mortality.

Mutschler et al.: The Shock Index revisited – a fast guide to transfusion requirement? A retrospective analysis on 21,853 patients derived from the TraumaRegister DGUW. Critical Care 2013 17:R172.
Blood pressure

The Shock Index (SI)

\[ \text{SI} = \frac{\text{Heart Rate}}{\text{SBP}} \]

- SI of 80 \( \div \) 120 = 0.6
- SI of 100 \( \div \) 120 = 0.83
- SI of 120 \( \div \) 100 = 1.2
- SI of 140 \( \div \) 80 = 1.75

Mutschler et al.: The Shock Index revisited – a fast guide to transfusion requirement? A retrospective analysis on 21,853 patients derived from the Trauma Register DGUW. *Critical Care* 2013 17:R172.
Blood pressure

The Shock Index (SI)

- Between 2002 and 2011, 21,853 adult trauma patients from the Trauma-Register database were divided into 4 groups.

- Units of transfused blood increased from 1.0 (± 4.8) in Group I to 21.4 (± 26.2) in Group IV.

- The Shock Index at ED presentation can be used as a clinical indicator of hypovolemic shock.

- The four SI groups also parallel the recently published Base Deficit-based classification.

- In daily clinical practice, SI may be used to assess for hypovolemic shock if point-of-care testing / technology is not readily available.

Mutschler et al.: The Shock Index revisited – a fast guide to transfusion requirement? A retrospective analysis on 21,853 patients derived from the Trauma Register DGUW. Critical Care 2013 17:R172.
Blood pressure

\[ SI = \frac{\text{Heart Rate}}{\text{SBP}} \]

**Shock:**
- Class I: \( \emptyset - \text{min.} \)
- Class II: mild
- Class III: moderate
- Class IV: severe

**SI:**
- < 0.6
- \( \geq 0.6 \) to < 1
- \( \geq 1 \) to < 1.4
- \( \geq 1.4 \)

**Need blood?**
- watch
- consider
- act
- MT!

Mutschler et al.: The Shock Index revisited – a fast guide to transfusion requirement? A retrospective analysis on 21,853 patients derived from the Trauma Register DGUW. *Critical Care* 2013 17:R172.
### Base Deficit-based Classification of Hypovolemic Shock

<table>
<thead>
<tr>
<th>Shock:</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>SI:</td>
<td>Ø - min.</td>
<td>mild</td>
<td>moderate</td>
<td>severe</td>
</tr>
<tr>
<td>Admit BD (mmol/L):</td>
<td>≤ 2</td>
<td>&gt; 2 to 6</td>
<td>&gt; 6 to 10</td>
<td>&gt; 10</td>
</tr>
<tr>
<td>Blood?</td>
<td>watch</td>
<td>consider</td>
<td>act</td>
<td>MT!</td>
</tr>
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Mutschler et al.: The Shock Index revisited – a fast guide to transfusion requirement? A retrospective analysis on 21,853 patients derived from the Trauma Register DGUW. *Critical Care* 2013 17:R172.
Hypertension is not the cause of headache in a patient with an otherwise normal neurologic exam.

Orthostatic vital signs are neither sensitive nor specific for hypovolemia, and their presence or absence should not be used to diagnose or disposition patients.

Initial fluid choice (excluding blood products!) has not been shown to affect outcomes (morbidity or mortality) in trauma resuscitation.

For uncontrolled hemorrhage (in the absence of TBI), target resuscitation to a SBP between 70 and 90 mm Hg or normal mentation and palpable peripheral pulses.

Hypotension in adults is a SBP < 90. Hypotension in pediatrics is a SBP < 70 + [2 x age in years]
Key Facts: Blood Pressure

BP is a vital sign no matter the age of the child; most pediatric cases “under review” are missing BP on their medical record.

The Shock Index is HR ÷ SBP and (at ED presentation) can be used as a clinical indicator of hypovolemic shock.

Arterial BP reflects the dynamic balance between cardiac output and peripheral vascular resistance and is determined by five components:

1. Cardiac output (rate & contractility)
2. Circulating volume (hemorrhage, dehydration)
3. Vascular tone (neurogenic or inflammatory)
4. Vascular permeability (anaphylaxis, sepsis)
5. Endocrine regulation (hypothyroid, adrenal tumor)
The Physician’s Vital Sign: Respiratory Rate
CC: 17-y/o ♀ brought to ED following a minor MVC for “not acting right.” Similar symptoms intermittently over the last few weeks. Patient without c/o.

PMHx: Denied. OCPs. ✥ THC “once”.

VS: Temp 97.8° F (36.6°C) HR 74, RR 8, BP 114/73, \( \text{SaO}_2 \) 97%

PE: Benign exam. Poor eye contact. Hypoactive bowel sounds noted.
Given a patient who is otherwise healthy and appears unremarkable (and is awake), what is the most likely explanation for the bradypnea?

A respiratory rate $\leq 12$ in a patient who is not asleep strongly suggests opioid intoxication.

Key Points: Respiratory Rate

Among elderly patients, the respiratory rate is the most sensitive in detecting early systemic infection, sepsis, or a progressive metabolic acidosis.

Respiratory rate is a highly sensitive indicator of acute illness in elderly patients.

Abnormal respiratory patterns may be a primary respiratory insult or metabolic or CNS disease.

Subtle tachypnea can be the only sign of serious illness.

A respiratory rate ≤ 12 in a patient who is not asleep strongly suggests opioid intoxication.
The Student’s Vital Sign: Pulse Oximetry
Cases #11-12, 22-y/o ♀ & 44-y/o ♂ c/o SOB

CC: A 22-y/o ♀ c/o SOB. Onset < 1 hour PTA. Recent hx includes UTI tx and dental procedure. Denies previous SOB.

PE: Temp 98.6° F (37°C), HR 112, RR 28, BP 110/56, SaO2 85%. Lungs CTA B/L. No rhonchi, rales, or wheezing heard.

CC: 44-y/o ♂ firefighter c/o SOB immediately after recent fire. Tank ran out during blaze, but he continued to rescue / work without it.

PE: Temp 99.5° F (37.5°C), HR 118, RR 26, BP 147/96, SaO2 99%. Lungs CTA B/L. No rhonchi, rales, or wheeze.
Case #11 - 22-y/o ♀ c/o SOB
98.6°F (37°C), HR 112, RR 28, BP 110/56, SaO2 85%

Additional information:
Recent medications include: TMP-SMX, phenazopyridine, benzocaine, and bupivicaine. Supplemental O2 has no effect on her pulse oximetry.

Given this clinical history and the SaO2 of 85%, what do you suspect is the reason for this patient’s dyspnea?
The oxidation of iron Fe^{++} from Fe^{++} (ferrous) → Fe^{+++} (ferric)

- Methemoglobin does not bind & carry oxygen the way normal Hb does.
- Pulse oximetry will be inaccurate in a dose-dependent fashion, typically 85% to 90%.
- Co-oximetry will show ↑↑ meth-Hb; a value > 20% generally threshold for treatment.
Case #12 - 44-y/o ♂ firefighter c/o SOB
99.5° F (37.5°C), HR 118, RR 26, BP 147/96, SaO2 99%

Given a clinical history of smoke inhalation and the seemingly normal SaO2 of 99%, what do you suspect is the reason for this patient’s dyspnea?
Carboxyhemoglobinemia

CO binds to Hb with an affinity 250 x that of O₂, preventing loading & unloading of O₂ to lungs & cells.

- Pulse oximetry will be normal b/c at the two wavelengths of light the pulse oximeter reads, oxyHb absorbs identical to carboxyHb.

- Treatment is 100% oxygen by NRBR (or hyperbaric O₂), ↓ the half-life of carboxyHb from 300 min. → 75 min.
Case #13 - 80 y/o ♂ c/o weak & AMS

CC: 80-y/o ♂ c/o several day hx of progressive weakness and confusion.

PMHx: A-fib, cardiomyopathy (EF ~30%), HTN, DM

VS: 97.6° F (36.4°C), HR 63, RR 20, BP 122/57, SaO₂ 100%

PE: Confused. No focal neurologic findings. Unable to ambulate.
Case #14 - 80 y/o ♂ c/o weak & AMS

97.6° F (36.4°C), HR 63, RR 20, BP 122/57, SaO₂ 100%

Given this patient’s PMHx of HTN, DM, A-fib, and cardiomyopathy, which of his vital signs is most abnormal for him?

A pulse oximetry of 100% is clearly abnormal in this elderly patient with multiple medical problems.
Only patients with fully functional CV/Pulm systems should have an SaO2 of 100% on RA.

- Elderly with chronic medical problems have fluid retention, alveolar interstitial edema, scarring, protein deposition → ↓ gas exchange in the lungs.
- Severe dehydration removes that alveolar fluid → falsely reassuring SaO2 of 100%
- 80-y/o ♂’s labs ultimately showed renal failure (BUN 160, Cr 6.4) due to severe intravascular hypovolemia.

Pulse oximetry

SaO2 of 100% = Abnormal
Pulse oximetry

Confounders

In methemoglobinemia, the pulse oximetry will be 85% (usually) because MetHb → large pulsatile absorbance signal at the red & IR wavelengths → absorbance ratio to unity (read as 85%).

In carboxyhemoglobinemia, the pulse oximetry will be falsely normal (b/c at two wavelengths of light that pulse oximeter reads, oxyHb absorbs identical to COHb).
The Procedural Vital Sign: Capnography (ETCO₂)
Capnography (ETCO\textsubscript{2})

“Breath by breath” measure of respiratory rate and CO\textsubscript{2} exchange

- ETCO\textsubscript{2} closely approximates arterial CO\textsubscript{2} levels
- Capnography gives an early warning (~1 min.) device to identify Subclinical Respiratory Depression (respiratory depression without hypoxia) before hypoxia occurs.
- This physician’s opinion is that it should be standard care in procedural sedation & analgesia.

Cases #14-16 – Three ♂s with mult. c/ os

CC: 5-y/o ♂ c/o unresponsive. “Little sick” yesterday, now unable to be awakened by Mom.

VS: 97.3° F, RR 36, HR 134, BP 83/62, SaO₂ 98%

PE: Moans to pain. No visible trauma.

CC: 21-y/o ♂ c/o seizure & confused. EMS transport from cross-country race.

VS: 100.1° F, RR 24, HR 119, BP 113/56, SaO₂ 99%

PE: Sweaty and ill appearing.

CC: 75-y/o ♂ c/o stroke. Left-sided hemiplegia 1° PTA. Speech is garbled.

VS: 97.4°F, RR 18, HR 98, BP 173/107, SaO₂ 96%

PMHx: HTN, DM.
“I’ve just been informed that your CT scanner is down indefinitely. Per unwritten hospital policy, no one will tell you when it is back up and running, and you will be criticized mercilessly for calling three times in the next 12 hours to ask for an update.”
In these clinical scenarios, what vital sign abnormality is common to all three patients?
The Forgotten Vital Sign: Blood Glucose
• The only fuel the brain can use is glucose.
• If you don’t have enough glucose to feed your brain, you (and your friends) will notice the difference.
• Glucose—*there simply is no substitute.*
Cases #14-16 – Three ♂s with mult. c/os

CC: 5-y/o ♂ c/o unresponsive.
VS: 97.3° F, RR 36, HR 134, BP 83/62, SaO₂ 98%

CC: 21-y/o ♂ c/o seizure & confused.
VS: 100.1° F, RR 24, HR 119, BP 113/56, SaO₂ 99%

CC: 75-y/o ♂ c/o Left-sided stroke.
VS: 97.4°F, RR 18, HR 98, BP 173/107, SaO₂ 96%

New-onset Type I Diabetes Mellitus

Hypoglycemia (didn’t eat before his race)

Hypoglycemia (took insulin and forgot to eat lunch)
Hypoglycemia is defined according to the following serum glucose levels:

- < 50 mg/dL in men
- < 45 mg/dL in women
- < 40 mg/dL in infants and children

Or any decrease in the blood glucose level (or its utilization) that → demonstrable signs or symptoms

The Unspoken Vital Sign: Gestalt
Fever in the elderly (>65 y/o) is frequently associated with serious illness.

Of 470 elderly patients with “serious illness”, 76% had associated clinical features: Temp > 103°F, RR > 30, or pulse > 120 bpm.


The Rule of 60s:

In an infant...

...a heart rate ~ 60 bpm

...a respiratory rate ~ 60 bpm

...or a systolic BP ~ 60 mmHg

Are all signs that the baby in front of you is dying--get busy.
The Vital Sign of Pregnancy: Fetal Heart Tones
Fetal Heart Tones

• Fetal heart tones and maternal perception of fetal movement are the single best indicators of fetal well being.

• First able to auscultate FHTs around 10 weeks gestational age.

• Normal FHT range is 110 – 160 bpm.

• Trauma in pregnancy is the most common cause of non-obstetrical maternal death.

• Focus all resuscitative efforts on mom—if she dies, the baby dies.

Supine Hypotensive Syndrome. Place the patient in a left lateral decubitus position. Hypotension due to position is a diagnosis of exclusion--think of bleeding first!

- Increased heart rate.
- Decreased blood pressure starting in the 2nd trimester.
- Pregnant patients have increased plasma volume which can mask hemorrhagic shock until collapse.
- Uterine blood flow is 20% of cardiac output; in hemorrhage, blood is shunted away from the fetus.
- Increased minute ventilation and tidal volume.

The Eye’s Vital Sign: Visual Acuity
Visual acuity should be assessed for all eye-related complaints.
The Bane of Vital Signs: Pain
“A Pain-Drug Champion Has Second Thoughts”
Thomas Catan and Evan Perez
Wall Street Journal
December 17, 2012
In 1986 at age 31, Dr. Russell Portenoy, co-wrote a landmark paper arguing that opioids could be used in non-cancer patients with chronic pain. His paper was based on 38 cases.

Previous to the 1990s, opioids were characterized as highly addictive, potentially dangerous, and were largely reserved only for cancer-related pain.

Charming and articulate, Portenoy became a sought-after public speaker and rose to Chairman of Pain Medicine & Palliative Care at Beth Israel Medical Center in New York.
Dr. Portenoy sought to “de-stigmatize these drugs.”

Steven Passik, a psychologist and colleague of Dr. Portenoy admits their message wasn't based on scientific evidence so much as a zeal to improve patients' lives. "It had all the makings of a religious movement at the time," he said.

Drug companies noticed. In 1996, Purdue Pharma LP released OxyContin (a form of oxycodone) in a patented, time-release form, and other drug manufacturers began to compete. Today, sales of opioid painkillers total more than $9 billion a year.
Dr. Portenoy's ideas caught momentum.

In a 1998 talk in Houston, Alan Spanos, a South Carolina pain specialist, said patients with chronic non-cancer pain could be trusted to decide themselves how many pills to take without risk of overdose.

Dr. Spanos said his understanding was that a patient would simply "go to sleep" before s/he stopped breathing. While asleep, the patient "can't take a dangerous dose. It sounds scary, but as far as I know, nobody anywhere is getting burned...doing it this way."

Dr. Spanos declined to say whether he still agreed with this previous statement.
In the late 1990s, groups such as the American Pain Foundation, of which Dr. Portenoy was a director, urged tackling what they called an epidemic of untreated pain.

The American Pain Society, of which he was president, campaigned to make pain referred to as the "fifth vital sign."

Dr. Portenoy helped compose a landmark 1996 consensus statement by two professional pain societies that said there was little risk of addiction or overdose among pain patients. In lectures he quoted a statistic that < 1% of opioid users became addicted.
Today, opioid supporters say that figure was incorrect. "It's obviously crazy to think that only 1% of the population is at risk for opioid addiction," said Lynn Webster, president-elect of the American Academy of Pain Medicine (one of the publishers of the 1996 statement). "It's just not true."

The 1% figure comes from a single-paragraph in a 1980 *NEJM* article describing hospitalized patients briefly given opioids.

Dr. Portenoy now admits that information was irrelevant for patients with chronic non-cancer pain as there is little evidence that opioids are safe & effective for long-term use in those patients.
In 1998, the Federation of State Medical Boards released a recommended policy reassuring doctors that they wouldn't face regulatory action for prescribing even large amounts of narcotics, as long as it was in the course of medical treatment.

In 2004 the group called on state medical boards to make undertreatment of pain punishable for the first time. That policy was drawn up with the help of several people with links to opioid makers, including David Haddox, a senior Purdue Pharma executive then and now.

The FSMB said it has received nearly $2 million from opioid makers since 1997.
In 2001, the Joint Commission, which accredits U.S. hospitals, issued new standards telling hospitals to regularly ask patients about pain and to make treating it a priority. The now-familiar pain scale was introduced in many hospitals, with patients being asked to rate their pain from 1 to 10 and circle a smiling or frowning face.

The Joint Commission published a guide sponsored by Purdue Pharma. "Some clinicians have inaccurate and exaggerated concerns" about addiction, tolerance and risk of death, the guide said. "This attitude prevails despite the fact there is no evidence that addiction is a significant issue when persons are given opioids for pain control."
Over his career, Dr. Portenoy has disclosed relationships with more than a dozen companies, most of which produce opioid painkillers. "My viewpoint is that I can have those relationships, they would benefit my educational mission, they benefit in my research mission, and to some extent, they can benefit my own pocketbook, without producing in me any tendency to engage in undue influence or misinformation" he said.

Dr. Portenoy and Beth Israel declined to give details of their funding by drug companies. A 2007 fundraising prospectus shows that Dr. Portenoy's program received millions of dollars over the preceding decade in funding from opioid makers including Endo, Abbott Laboratories, Cephalon, Purdue Pharma, and Johnson & Johnson.
“A Pain-Drug Champion Has Second Thoughts,”


Doctor who championed use of opioids for chronic pain now says “it was the wrong thing to do,” *The Rehab Center, Inc.*, 2012.

The "King of Pain" Recants - Pharmaceutical Paid Key Opinion Leader Admits it was all “Misinformation” *Health Care Renewal*, hcrenewal.blogspot.com, 12/2012
Summary – Adult Vital Signs in Trauma

The Mother’s Vital Sign: Temperature

The Paramedic’s Vital Sign: Pulse

The Nurse’s Vital Sign: Blood Pressure

The Physician’s Vital Sign: Respiratory Rate

The Student’s Vital Sign: Pulse Oximetry

The Procedure Vital Sign: Capnography (ETCO₂)

The Forgotten Vital Sign: Blood Glucose

The Unspoken Vital Sign: Gestalt

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References


