Interpretation of changes in hematocrit in the integral system of condition assessment of the body
Andrey Belousov


ABSTRACT

The focus of the article is rather situated on current faults and recommendations for transfusion of red blood assessment, clinical evaluation of changes in hematocrit. The main task of therapy for acute massive blood loss is not urgent thoughtless transfusion of red blood cells for the fast recovery of the hemoglobin and hematocrit levels. The oxygen-carrying capacity of blood does not directly reflect the delivery of oxygen to tissues. The severity of the patient’s condition depends on individual ability of the organism to resist hypoxia, mechanisms resulting in physiological compensation for the anemia caused by blood loss. The main tasks of therapy are timely maintaining appropriate and effective compensatory-adaptive reactions of an organism, providing of the sanogenetic processes. Quickly and comfortable algorithm assessment changes in hematocrit was presented for used in practice. Objective analysis hematocrit and hemoglobin levels should be carried out only in combination with data on blood pressure, pulse rate, respiratory rate, urine output and shock index.

Key Words: Hematocrit; Hemoglobin; Assessment system; Blood loss; Transfusion of red blood

In modern health care today, blood transfusion plays a vital role. Blood transfusion can alleviate health and save life if used appropriately. According to WHO (1), appropriate use of blood products is defined as "the transfusion of safe blood products only to treat a condition leading to significant morbidity or mortality that cannot be prevented or managed effectively by other means". RBC transfusions are used to treat hemorrhage and to improve oxygen delivery to tissues. Transfusion of RBCs should be based on the patient’s clinical condition. Indications for RBC transfusion include acute sickle cell crisis (for stroke prevention), or acute blood loss of greater than 1,500 mL or 30 percent of blood volume. Patients with symptomatic anemia should be transfused if they cannot function without treating the anemia [2]. Symptoms of anemia may include fatigue, weakness, dizziness, reduced exercise tolerance, shortness of breath, changes in mental status, muscle cramps, and anemia or severe congestive heart failure. The 10/30 rule - transfusion when a patient has a hemoglobin level less than or equal to 10 g per dL (100 g per L) and a hematocrit level less than or equal to 30 percent - was used until the 1980s as the trigger to transfuse, regardless of the patient’s clinical presentation (2,3).

CASE STUDY

In 1999, a randomized, multicenter, controlled clinical trial evaluated a restrictive transfusion trigger (hemoglobin level of 7 to 9 g per dL [70 to 90 g per L]) versus a liberal transfusion trigger (hemoglobin level of 10 to 12 g per dL [100 to 120 g per L]) in patients who were critically ill. Restrictive transfusion practices resulted in a 54 percent relative decrease in the number of units transfused and a reduction in the 30-day mortality rate. The authors recommended transfusion when hemoglobin is less than 7 g per dL, and maintenance of a hemoglobin level between 7 to 9 g per dL (4). A recently updated Cochrane review supports the use of restrictive transfusion triggers in patients who do not have cardiac disease (5).

A similar study was carried out in critically ill children. The restrictive transfusion trigger was a hemoglobin level of 7 g per dL, with a target level of 8.5 to 9.5 g per dL (85 to 95 g per L). The liberal transfusion trigger was a hemoglobin level of 9.5 g per dL, with a target level of 11 to 12 g per dL (110 to 120 g per L). Patients in the restrictive group received 44 percent fewer blood transfusions, with no difference in rates of multiple organ dysfunction syndrome or death. The restrictive transfusion strategy is useful for children who are stable patients in intensive care. It should not be used in preterm neonates or in children with severe hypoxemia, active blood loss, hemodynamic instability, or cyanotic heart disease (6). Key recommendations for transfusion of red blood cells is presented in Table 1.

The main purpose is on the contrary show that hematocrit and hemoglobin levels not only does not reflects the severity of the patient with blood loss, but also a priori are not targeted indicators for transfusion of red blood cells.

Clinical transfusiology is new medical discipline which accumulates fundamental knowledge in Hematology, Immunohematology, Pathophysiology, Biochemistry, Biophysics, Histology fields and other adjacent medical specialties. Unfortunately, today exist erroneous and potentially dangerous point of view regarding the importance of the discipline among some anesthesiologists and resuscitators. Inertia of the false traditional views on blood loss and an archaisms of fundamental knowledge in the field of Clinical Transfusiology are influences on quality writing of the clinical protocols transfusion of red blood cells. Availability of large clinical trials, where absent information of mechanisms resulting in physiological compensation for the anemia caused by blood loss forms among physicians a false concept about adequate assessment severity of the patients' clinical condition. Intensive care physicians and anesthesiologists should be aware of treatment priority actions which maintain the compensatory-adaptive reactions of an organism in case severe blood loss. The primarily, the therapy must be directed at the elimination of acute deficit circulating blood volume, secondly, correction of coagulation factors, and only in the last instance - to a

TABLE 1

<table>
<thead>
<tr>
<th>Key recommendations for transfusion of red blood cells</th>
</tr>
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<tbody>
<tr>
<td>Clinical recommendations</td>
</tr>
<tr>
<td>The threshold for transfusion of red blood cells should be a hemoglobin level of 7 g per dL (70 g per L) in adults and most children.</td>
</tr>
<tr>
<td>A restrictive transfusion strategy (hemoglobin level of 7 to 9 g per dL [70 to 90 g per L]) should not be used in preterm infants or children with cyanotic heart disease, severe hypoxemia, active blood loss, or hemodynamic instability.</td>
</tr>
</tbody>
</table>

Notes: RCT = Randomized Controlled Trial. A = Consistent, good-quality patient-oriented evidence; B = Inconsistent or limited-quality patient-oriented evidence. For information about the SORT evidence rating system, go to https://www.aafp.org/sort.xml.

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possible increase in the number of the oxygen-carrier (7). The severity of the patient’s condition in acute massive blood loss does not depend directly on the hemoglobin and hematocrit indicators.

At rest, there is a large reserve in oxygen delivery, since the rate of delivery normally exceeds consumption by a factor of four. Thus, if intravascular volume is maintained during bleeding and cardiovascular status is not impaired, oxygen delivery theoretically will be adequate until the hematocrit falls below 10 percent because greater cardiac output, rightward shift of the oxygen-hemoglobin dissociation curve, and increased oxygen extraction can compensate for the decrease in arterial oxygen content. These predictions were confirmed in a study in which healthy resting individuals underwent acute isovolemic reduction of their hemoglobin to 5 g/dL (equivalent to a hematocrit of approximately 15 percent) (8). Though some individuals did develop electrocardiogram (ECG) changes consistent with myocardial ischemia, there was little evidence of inadequate oxygen delivery, and the fall in hemoglobin was associated with progressive increases in stroke volume and heart rate (and therefore cardiac output), and a progressive reduction in the systemic vascular resistance. Heart rate was found to increase linearly in response to the acute isovolemic anemia (9). Of note, cognitive function measured by reaction time and immediate memory was impaired when the hemoglobin concentration was reduced to 5 to 6 g/dL (10).

The oxygen-carrying capacity of blood does not directly reflect the delivery of oxygen to tissues (11). The severity of the patient’s condition depends of individual ability of the organism to resist hypoxia, mechanisms resulting in physiological compensation for the anemia caused by blood loss (12). As rule blood transfusion is usually administered to patients who are ill with underlying comorbidities, and there is concern that compensatory mechanisms may be impaired in critically ill patients, particularly in patients with underlying cardiovascular disease. Therefore, continuous improvement has become an important strategy in improving personalized express-diagnostic systems of functional state of the organism, estimation algorithms of the patient’s severity in case of blood loss (13). Many hospitals have developed general guidelines for the appropriate use of blood transfusion, and an “implementation blueprint” for establishing a patient blood management program has been published by members of the High Value Practice Academic Alliance (14). A patient blood management program uses “an evidence-based multidisciplinary approach to optimizing the care of patients who might need transfusion.” Patient blood management programs “include interventions taken early in the preparation of medical and surgical patients for treatment, as well as techniques and strategies in the preoperative, operative, and postoperative periods or completion of treatment” (15). Three pillars of this type of program include optimizing hematopoesis, minimizing blood loss and bleeding, and harnessing and optimizing tolerance of anemia (16). I fully support this the authors’ point of view. I’m in favor of such programs, because they attempt to reduce unnecessary transfusion and may reduce costs.

However, this programs and broad guidelines should not supersede clinical judgment in decisions regarding transfusion, especially by clinicians who are familiar with the individual patient. As an example, if a patient is experiencing symptoms that are known to reflect cardiac ischemia in that individual, transfusion may be appropriate. Alternatively, if a patient is known to tolerate a lower hemoglobin than that specified in the guideline, then it may be possible for that patient to avoid transfusion.

Today one of the alternative variants of the objective clinical evaluation of patients can be PHUAS (Physiological Universal Analytic System) program (17,18).

The main task of therapy for acute massive blood loss is not urgent thoughtless transfusion of red blood cells for the fast recovery of the hemoglobin and hematocrit levels. The main tasks of therapy are timely maintaining appropriate and effective compensatory-adaptive reactions of an organism, providing of the sanogetic processes.

With regard to changes in hematocrit and hemoglobin, experienced practitioners know that decreases in hematocrit and hemoglobin levels do not always reflect the degree of blood loss. For example, hemo-dilution is causes a decrease in hematocrit and hemoglobin levels. On the contrary thickening of the blood is causes an increase in hemoglobin and hemoglobin levels. Examples of clinical of the variants of changes in hematocrit on the background of the reaction of the basic organism physiological parameters were presented in Table 2. This table is quickly and comfortable algorithm assessment changes in hematocrit which need used in practice.

| TABLE 2 |
| The variants of changes in hematocrit on the background of the basic organism physiological parameters |

<table>
<thead>
<tr>
<th>Variants</th>
<th>HCT</th>
<th>Pulse rate</th>
<th>BP</th>
<th>Urine output</th>
<th>Respiration rate</th>
<th>Shock index</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>↓</td>
<td>↑</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal/↑</td>
<td>↑</td>
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<tr>
<td>II</td>
<td>↓</td>
<td>Normal↓|</td>
<td>↑</td>
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<td>III</td>
<td>↓</td>
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<td>↓</td>
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<td>↓</td>
<td>↑ ↑</td>
</tr>
<tr>
<td>IV</td>
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<td>Normal↑|</td>
<td>Normal↑|</td>
</tr>
</tbody>
</table>

**Notes:** HCT – Hematocrit; BP - Blood pressure; Shock index = Pulse rate/ Systolic Blood pressure (normal = 0.54).

**DISCUSSION**

**Variant I**

Low hematocrit levels against the background of moderate tachycardia and tachypnea that increase after physical activity. Blood pressure and hourly urine output are normal. The shock index is moderately elevated. This variant reduction in hematocrit characterizes the hemic hypoxia. In this case, when absence of the dynamic reduction in hemoglobin and hematocrit levels transfusion of red blood cells is not advisable.

**Variant II**

Low hematocrit levels against the background of normal or slight decrease in heart rate. Blood pressure up. Urine output - decreased or normal. The shock index is always below normal (<0.54). This variant characterizes the hypovolemic state. At the same time transfusion of red blood cells in order to increase the level of hematocrit is not only unjustified but also danger to the patient’s life due to volume overload of the small circle of blood circulation.

**Variant III**

Low hematocrit levels against the background of expressed symptoms of tachycardia and tachypnea. Decreased blood pressure and hourly urine output to anuria. The shock index is elevated (>1.5). This variant characterizes the mixed form of hypoxia (circulatory + hemic hypoxia) that is caused by massive blood loss.

**Variant IV**

High hematocrit levels against the background of normal or high heart rate, increased blood pressure and urine output. At the same time dynamics of increased in hematocrit may be accompanied by decreased hourly urine output. The shock index is elevated. This variant characterizes the hypovolemic polythemia.

**CONCLUSION**

Thus, hematocrit and hemoglobin levels not only do not objectively reflect degree of blood loss and patient’s severity but also a priori are not targeted indicators for transfusion of red blood cells. Objective analysis hematocrit and hemoglobin levels should be carried out only in combination with data on blood pressure, pulse rate, respiratory rate, urine output and shock index. Recommendations for transfusion of red blood cells which are based only on hematocrit and hemoglobin data are not justified and unsafe for the patient.

**CONFLICT OF INTEREST**

None.

**REFERENCES**

Interpretation of changes in hematocrit


