Turk J Anaesthesiol Reanim 2017; 45: 176-8 DOI: 10.5152/TJAR.2017.24041

Letter to the Editor

The FALLS-Protocol, Another Way to Assess Circulatory Status Using Lung Ultrasound

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Dear Editor,

By reading the titles of the issue of the Turkish Journal of Anesthesiology and Reanimation (vol 44, Issue 5, October 2016) (1-4), we felt that everything was not completely over regarding volemia assessment. Several tools still seem to compete, each school advocating its tool as the one which definitely answers this endless issue, so in the absence of recognized gold standard, may we propose one more?

The FALLS-protocol (Fluid Administration Limited by Lung Sonography) is a different pathophysiological concept that we want to share, with major humility, to the curiosity of the reader. The FALLS-protocol is based on eight sequential considerations.

1) A circulatory failure is a deadly condition, even if managed in the best institutions. The community should pay attention to any new concept aiming at decreasing this mortality rate.

2) Likely, there is a characterized absence of gold standard for a fast assessment.

3) The tools for measuring volemia are rather indirect. Even echocardiography, definitely an elegant method, assesses only one actor of the circulatory system. Can't we find more direct?

4) The FALLS-protocol considers the weakest pump as the left ventricle, at least on admission of the critically ill patient in acute circulatory failure. In this acceptation, the fragile organ will be the lung, and the very definition of hemodynamic pulmonary edema (HPE) will be revisited with a new light (cases where the weakest pump is the right ventricle are on focus too, but should be taught at a later step for didactic reasons).

5) Interstitial edema always preceedes alveolar edema when a HPE occurs (5, 6).

6) The FALLS-protocol considers an alveolar volume, substantial (several liters) and an interstitial volume (i.e., the necessary volume for saturating the interstitial tissue) as minute, say 20 or 30 mL maybe. The alveolar floading is deadly like having our head under the water; the interstitial edema is as innocuous as walking through the rain.

7) Lung ultrasound (in the critically ill – LUCI) appears as a providence, since it is able to detect interstitial syndromes, here the edema of the subpleural interlobular septa, through an hydro-aeric artifact, the B-line[1]. The B-line allows first an qualitative diagnosis of pulmonary edema (concentrated B-lines, i.e., lung rockets[1] present or not), as well as a quantitative approach, based on the distribution of the lung rockets, their pattern (septal versus ground-glass rockets[1]). The B-line is generated on an on-off basis: while the fluid therapy is done continuously, the B-line appears all of a sudden (after 28 years and years spent behind a screen, we are unable to describe any intermediate artifact between the A-line[1] and the B-line). On-off markers of volemia are not current in medicine, and this is the starting point of the FALLS-protocol.

8) We just now have to demonstrate a correlation between LUCI and the pulmonary artery occlusion pressure (PAOP). We have found a 18-mm-Hg cutoff when the A-lines are replaced by B-lines (7). Some can argue that the PAOP is an obsolete

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Received : 10.04.2017
Accepted : 22.04.2017

data, dangerous to obtain, but we all have a PAOP. We are sure that most doctors, today, would appreciate a noninvasive approach to this parameter.

The FALLS-protocol wants to be analyzed, commented, criticized. It does not pretend to have found the magic wand to hemodynamic assessment - such a complex issue. It is built with a maximal humility, but also a pathophysiological basis. It wants to help the first-line physician in the first hours of management of a shock, by providing a diagnosis. Once a diagnosis done, in the ICU e.g., other rules should be applied for assessing the daily needs in fluids. The FALLS-protocol tries to answer to the two main questions: 1) which patients need a fluid therapy in the extreme emergency? 2) When to discontinue it once initiated?

Let us now visit a patient with a circulatory failure without clinically obvious cause. We use a simple ultrasound machine (1982 technology, gray-scale without Doppler) and a universal, microconvex long range probe. We will follow the Weil's classification of shocks (8). Maybe obsolete for some, this is the one that allows the FALLS-protocol to be applied.

The pericardium is first scanned. No substantial pericardial effusion (it would be here, in a shocked patient, an equivalent of tamponade)? We then scan the right ventricle (RV) volume. No RV dilatation? The diagnosis of pulmonary embolism, or any disease generating such a disorder, pulmonary hypertension e.g., can be reasonably ruled out. Then the universal probe is shifted laterally for checking the absence of pneumothorax. No pericardial tamponade, pulmonary embolism, pneumothorax? An obstructive shock is, likely, not the cause of this circulatory failure. The FALLS-protocol goes on.

We scan again the lung, searching for lung rockets. If absent, we can rule out a hemodynamic pulmonary edema. Left cardiogenic shock is defined as low cardiac output associated with high LV filling pressure. Therefore, left cardiogenic shock can reasonably be ruled out. Right cardiogenic shock with low PAOP, typically from RV infarction, not a frequent cause, is dealt with apart in the FALLS-protocol (the usual practice of an ECG in a shock by the way simplifies the approach).

At this step, the A'-profile[1] of pneumothorax and the B-profile[1] of HPE have been excluded; the patient has usually the A-profile[1] or equivalents (A/B-profile[1], C-profile[1] on predominant A-lines). Now that obstructive and cardiogenic shock have been ruled out, we consider that both of the only remaining causes (hypovolemic and distributive) require fluids. At this step, the FALLS-protocol stipulates that a patient with an A-profile can, and mostly should, benefit from fluids. Such patients are called "FALLS-responders". The therapeutic part of the FALLS-protocol can begin.

The fluid therapy should improve the clinical signs of a hypovolemic shock, this is how this diagnosis is done using the FALLS-protocol.

If no clinical sign of shock improves, there is no clinical signal for discontinuing the fluid therapy. If fluids begin to saturate the lung interstitial compartment, lung rockets will appear. They will appear all of a sudden. This is the FALLS-endpoint, time to discontinue the fluid therapy. This therapy has generated a fluid overload at the interstitial step, which has the peculiarity to be clinically occult, biologically occult. Yet smart minds may consider that there is any way too much fluid. For withdrawing these few mL in excess (20, 30 mL in our hypothesis), various procedures can be applied (e.g., laying down previously raised legs, the FALLS-PLR protocol). The most practical is to order biological tests, including several blood cultures. Even if blood-lettings are today considered totally obsolete, they result anyway in decreasing the PAOP, i.e., a relief of HPE. The pathophysiology is independent from the trends.

If the patient "succeeded" to saturate the interstitial lung compartment without improving the circulatory status, we can reasonably consider that the diagnosis of hypovolemic shock can just be ruled out. The last remaining cause, distributive shock, should be, by default, considered. Spinal shock is rarely an issue, anaphyllactic shock also occurs in suggestive settings, usually. What remains then, but septic shock? Schematically, in the sequence of the FALLS-protocol, septic shock is defined by the transformation from A-lines to B-lines. At this step, other tools than simple fluid should be used for improving the circulation, mainly vasopressors.

As we guess that many practicionners will find this approach simplistic, we anticipate, in the alloted space, some questions.

The FALLS-protocol generates a pulmonary edema? Certainly. But the term "pulmonary edema" creates a major confusion: many doctors imagine alveolar edema, i.e, drowning of the patient. The "FALLS-edema" so to speak, is interstitial, not alveolar, i.e., clinically occult. This edema is relieved by the blood cultures, which will hopingly position the heart at the best place on the Frank-Starling function curve.

Which quantity of fluids in a septic shock? During years, doctors gave massive fluid therapy. Then recently, they were scared by the idea that water kills. And now, they try to come back to a more balanced behaviour. In septic shock, not one drop of fluid is given by the FALLS-protocol (fluid is even withdrawn). Fluid has been discontinued just once the diagnosis done.

Early fluid therapy? During years, doctors gave early fluid therapy in septic shock. The FALLS-protocol is much earlier, because it gives fluids far before knowing that this shock is septic. Hours are saved, hours of possibly deleterious hypovolemia. These hours may, maybe, make a difference in terms of survival.

The FALLS-protocol has limitations. First, a patient with diffuse lung rockets[1] on admission cannot benefit from any

FALLS-protocol (which detects the transformation from A-lines to B-lines). Other rules should apply for distinguishing a cardiogenic shock (the most frequent) from a lung sepsis with a B-profile. Second, these cases where the weakest cardic pump is suspected to be the right heart can make a huge debate, as some influent experts estimate that patients with pre-capillary obstacle are protected from pulmonary edema. For anticipating this kind of remark, we just, temporarily - before the opposite is proven - apply this rule: in a shocked patient, when a RV anomaly has been detected, and when no DVT has been found, the FALLS-protocol should... not be used (usual tools will be used).

For remaining in the alloted volume, many points of interest, not dealt with here, have a comprehensive answer in (9). How to be able to manage a circulatory failure without knowing the cardiac output e.g., how can difficult patients (bariatric, peri-operative, comorbid, specific settings and others) benefit from the FALLS-protocol, and many others.

We reiterate for concluding that the FALLS-protocol is open to any criticism... but just imagine it works (10).

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[1]For sake of brevity, we assume these terms as familiar in 2017. If not, read a recent review such as Chest 2008;134:117-125, Annals of Intensive Care 2014;4:1;1-12, Current Opinion in Critical Care 2014;20:315-322 or Chest 2015;147:1659-1670.