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Letter to the Editor

Dear Editor,

We read with extreme interest the original article “The handheld blood lactate analyser versus the blood gas based analyser for measurement of serum lactate and its prognostic significance in severe sepsis.” by Singh et al. published in *Med J Armed Forces India* 2016;72:325–331.¹ The study compared the utility of handheld lactate analyser vs lactate measurement from a regular arterial blood gas (ABG) machine. The study is well crafted and highlights the importance of lactate value in prognostication and management of severe sepsis.

The incidence of sepsis requiring intensive care unit admission is increasing alongside the growing epidemic of antibiotic resistance. It is associated with increased morbidity and mortality and causes a significant burden on the healthcare resources. Sepsis remains to be a hot topic of research, and there is continued development and evaluation for new diagnostic and resuscitative interventions aimed to decrease mortality. Lactate is one such effort which has come in to recent focus.

Lactate measurements are recommended early in the diagnosis of sepsis according to the surviving sepsis guidelines.² Some authors have added clinical parameters to capitalize on the theoretical, predictive, and diagnostic value of this test. Serial lactate measurements are useful in guiding therapy and predicting survival.³ Authors have used the SAPS score in addition to lactate measurements and have shown it to be good predictor of mortality.

In the field setting where resources are limited and the decision to transfer a patient needs to be taken, a test which will help triage and guide decision making is a welcome change. ABG machine has lactate as one of the parameters and sometimes the machine might not be calibrated to show this value. It requires regular maintenance, expertise to operate and interpret the values. It might not be feasible to transport or make it available in all peripheral hospitals. Handheld lactate analyzers might be useful in these settings providing immediate results and being cost effective.

However a device just to measure lactate value might not be useful in a resource limited setting as an ABG machine will

give additional parameters like acid base status, hemoglobin, and electrolytes which might be preferred in a field setting. The authors have not mentioned the limitations of the study and the downside of using this device in place of an ABG machine.

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Reply

Dear Editor,

We thank the readers for showing interest and commenting on our article.

From the last paragraph of the letter it seems that the impression gained from the article is that we advocate the use of the hand held lactate meter as a replacement of the arterial blood gas (ABG) machine. This is incorrect as we have compared the results obtained from both the machines in an effort to validate the results obtained from the hand held machine.

Providing an ABG machine in all field settings may not be economically feasible, moreover, even provision of the basic support systems for the ABG machines (which do require extensive service and engineering back up) may not be possible everywhere. It is in these settings where the hand held lactate meter would provide invaluable inputs for resuscitation and follow up of patients suffering from the sepsis syndrome. We therefore in fact do not consider it as a

limitation of the study. Moreover, we would also not agree that 'field settings are resource limited'. Compared to any other health care organization in the country, AFMS field hospitals are very well equipped with pulse oximeters, transport ventilators, infusion pumps, etc. provided from various sources. Therefore, provisioning of handheld lactate meters at the periphery is very much possible and economical.

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Letter to the Editor

Metabolic syndrome and BPH: What do we know?

Dear Editor,

At the outset, I offer my compliments to the authors of the original article "Association between components of metabolic syndrome and prostatic enlargement: An Indian perspective" by Nandy et al. in *Med J Armed Forces India* 2016;72:350–355.¹ The authors in this present article have delved into an unexplored subject as far as the Indian subcontinent is considered and have thus added to the Indian database, which is lacking on the subject. They have brought to light the association of metabolic syndrome with that of the prostatic volume and Lower Urinary Tract Symptoms (LUTS), in the Indians, an ethnically different subpopulation. Although the subject of the article has been taken well, the presentation, however, lacks the lustre to attract the readers and has also failed to discuss the plausible mechanisms whereby the metabolic syndrome leads to the development of Benign Prostatic Hyperplasia (BPH). This not only leaves the readers wanting for more but also makes the article to look incomplete.

There have been a number of publications which have established the association of BPH with that of metabolic syndrome. But the exact mechanism which leads to the development of BPH in this subset of patients with metabolic syndrome is not clear. Various researchers have proposed different theories which include, but are not restricted to, changes in insulin resistance, increased autonomic activity, impaired nitrergic innervation, increased Rho kinase activity,

pro-inflammatory status and changes in sex hormones that occur in association with metabolic syndrome. However, the exact underlying mechanisms that regulate the potential relationship between MetS and BPH/LUTS still need to be clarified. Recently, Vignozzi et al. introduced an interesting three-hit hypothesis on the development of BPH in such subgroup of patients.² This hypothesis proposes that an overt or sub-clinical inflammation which is the first hit could be overlapped by the metabolic alternations in the body – the second hit and changes in sex-hormone levels and the third hit, leading to the over-expression of toll-like receptors, transformation of prostatic cells into antigen-presenting cells and up-regulation of growth factors (andromedins), resulting in prostate enlargement.

Hyperinsulinaemia, which is one of the most common derangements found in metabolic syndrome, leads to increased sympathetic activity, which in turn leads to smooth muscle contraction throughout the genitourinary tract contributing to the LUTS.³ Hyperinsulinaemia may also indirectly increase the risk of BPH by increasing the level of insulin-like growth hormone 1.³

The elevation of pro-inflammatory markers in metabolic syndrome tends to reduce the synthesis of nitric oxide (NO) in the endothelial cells. The reduced NO activity in the prostate in turn leads to smooth muscle proliferation, prostatic enlargement and thus contributes to LUTS.³

The association of altered lipid profile, especially high serum triglycerides and low serum High Density Lipoproteins