performed under moderate sedation or general anesthesia. However, the patient population studied in this previous report is different. These were patients with adenopathy not limited by size (mean size of lymph nodes sampled, 16 ± 3.6 mm; range, 8 to 32 mm), complete mediastinal and hilar lymph node screening was not performed, and fewer nodes were sampled (mean, 1.14 per patient). These three differences would lead to reduced time and increased simplicity of the procedure, making it more amenable to moderate sedation.

As more and more centers begin EBUS-TBNA programs, it is important that the procedure is performed in the correct environment and with realistic expectations of patients, operators, and equipment performance characteristics. If a consensus is reached that complete nodal staging is feasible and accurate with EBUS-TBNA, it may be more optimally performed under general anesthesia until the bronchoscopist feels comfortable in targeting these smaller lymph nodes in multiple stations in a conscious patient. We have recently described a safe option using propofol sedation with a laryngeal mask airway ventilation in the bronchoscopy suite that allows full access to the mediastinal lymph node stations. In contrast, EBUS-TBNA may be safely performed under conscious sedation in those patients with larger nodes without the requirement for complete lymph node staging.

Marcus P. Kennedy, MD
Yousef Shweihat, MD
University of Arkansas for Medical Sciences
Little Rock, AR
Mona Sarkiss, MD
Georgie A. Eapen, MD
University of Texas MD Anderson Cancer Center
Houston, TX

The authors have no conflicts of interest to disclose.

DOI: 10.1378/chest.08-1300

REFERENCES

Response

To the Editor:

We thank Dr. Kennedy and his colleagues for their comments regarding the level of sedation during endobronchial ultrasound (EBUS) transbronchial needle aspiration (TBNA). The use of propofol and a laryngeal mask airway is certainly an additional option available to the bronchoscopist. It is important to remember, though, that there is currently no proof that any level of anesthesia deeper than moderate sedation is required for performing the procedure. This applies for the goal of the procedure (full staging vs targeted biopsy) as much as for the level of experience. Even though we agree that general anesthesia may make it easier especially for the relatively inexperienced operator, some issues require consideration before asking the anesthesiologist to provide deep sedation or general anesthesia for a patient.

Part of the advantage of EBUS TBNA is the ease and minimal patient impact compared with surgical staging, as well as the potential economic advantage. Deeper levels of sedation may partially negate these advantages by adding additional personnel and requiring operating room-type facilities in some institutions. An additional drawback to adding more resources that really are probably not required is the recent severe reimbursement cut-back on the facility-based reimbursement (Hospital Outpatient Prospective Payment System) for EBUS TBNA procedures by the Centers for Medicare and Medicaid Services in January 2008. We need to choose the best approach for our patients but need to manage and minimize the resource use at the same time.

Armin Ernst, MD, FCCP
Boston, MA
Felix Herth, MD, FCCP
Ralf Eberhardt, MD
Heidelberg, Germany
Mark Krasnik, MD
Copenhagen, Denmark

The authors have no conflicts of interest to disclose.

DOI: 10.1378/chest.08-2022

REFERENCES
3 Centers for Medicare and Medicaid Services. Federal Register 2007, 72

Does Central Venous Pressure Predict Fluid Responsiveness?

To the Editor:

Marik and coauthors1 argue that the poor correlation between right atrial or central venous pressure (CVP) and indexes of fluid responsiveness after fluid administration limit the value of CVP as a predictor of fluid responsiveness. This appears to be at odds with the classic principles of circulatory physiology by Guyton and Hall, in which right atrial pressure is a key independent variable influencing both systemic venous return and right ventricular output. In these constructs, venous return may be independent of right atrial pressure at low pressures while right ventricular output may be independent of right atrial pressure at higher pressures, in agreement with the review, but not in the middle

www.chestjournal.org

DOI: 10.1378/chest.08-1300

Chest / 134 / 6 / December, 2008

The authors have no conflicts of interest to disclose.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestjournal.org/misc/reprints.shtml).

Correspondence to: Armin Ernst, MD, FCCP, Chief, Interventional Pulmonology, BIDMC, 330 Brookline Ave, Boston, MA 02215; e-mail: aerist@bidmc.harvard.edu

DOI: 10.1378/chest.08-2022

REFERENCES

Does Central Venous Pressure Predict Fluid Responsiveness?

To the Editor:

Marik and coauthors argue that the poor correlation between right atrial or central venous pressure (CVP) and indexes of fluid responsiveness after fluid administration limit the value of CVP as a predictor of fluid responsiveness. This appears to be at odds with the classic principles of circulatory physiology by Guyton and Hall, in which right atrial pressure is a key independent variable influencing both systemic venous return and right ventricular output. In these constructs, venous return may be independent of right atrial pressure at low pressures while right ventricular output may be independent of right atrial pressure at higher pressures, in agreement with the review, but not in the middle
Controlling cardiac output. Physicians commonly mistake venous experiments demonstrated the role of peripheral vasculature in of Guyton and Starling are commonly misunderstood. The pump It is an interesting reminder of related misconceptions in classical a common myth that CVP is a good guide to fluid management. volume status and fluid responsiveness. They called attention to controlled studies, show that isolated central venous pressure of cardiac performance but by criteria such as pressor requirements or blood lactate concentrations. While the current recommendations guiding fluid replacement use CVP as only one of a number of clinical parameters to follow, and while dynamic parameters may have an increasing role in guiding fluid replacement, it may be premature to discard CVP measurements in ICU patients.

Randolph Cole, MD
Holy Name Hospital
Teaneck, NJ

The author has no conflict of interest to disclose.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestjournal.org/misc/reprints.shtml).

Correspondence to: Randolph Cole, MD, Holy Name Hospital, 718 Teaneck Rd, Teaneck, NJ 07666; e-mail: rc18@columbia.edu

DOi: 10.1378/chest.08-1846

REFERENCES
3 Berne RM, Levy MN. Cardiovascular physiology. 5th ed. St Louis, MO: CV Mosby, 1986

Central Venous Pressure as Popular Resuscitation Surrogate

Not Totally Unjustified

To the Editor:

Marik and colleagues, using statistical analysis and data from controlled studies, show that isolated central venous pressure (CVP) measurements are a poor guide to the assessment of volume status and fluid responsiveness. They called attention to a common myth that CVP is a good guide to fluid management. It is an interesting reminder of related misconceptions in classical works of cardiovascular physiology. For instance, the experiments of Guyton and Starling are commonly misunderstood. The pump is commonly seen as the main orchestrator of output. Guyton’s experiments demonstrated the role of peripheral vasculature in controlling cardiac output. Physicians commonly mistake venous inflow to the atria as the determinant of left ventricular stroke volume. Starling, working on denervated hearts, showed that atrial filling and thus “ventricular wall stretch” was the primary determinant of stroke volume. Perhaps these misconceptions are a result of confusion between the cause and the consequence. CVP is affected by a myriad of intrinsic and extrinsic factors including IV fluids, positioning, intrathoracic pressures, heart rate, contractility, and myocardial and venous compliance, among others. Further confusing the issue is discrepancy between normal physiology and disease state pathology. As an example, sepsis alters total effective vascular compliance. Thus while CVP reflects mean right atrial pressure, it is not expected to show an independent correlation with effective circulatory blood volume.

Apparently, it is the intuitive simplicity of CVP that attracts a new learner as compared to the lack of receptiveness encountered with terms such as upstream and downstream resistance, flow limitation, and capacitance. The CVP-centric practice may also be widespread because it comes with convenience and there are no controlled studies that prove definite disadvantage related to the method use for fluid management. The 2008 Surviving Sepsis Campaign guidelines say “… recommend fluid resuscitation initially target a CVP of at least 8 mm Hg.” Justification of this statement is mostly based on a single study. The attempt at getting at least one of the determinants (CVP in this case) of cardiac output right, ie, achieving physiologic limits, may be a reasonable approach toward attempting to optimize perfusion in critically ill patients.

To conclude, we feel that the findings reported by Marik and colleagues are not unexpected. CVP remains meaningful along with the other parameters (BP, heart rate, urine output, extra heart sounds, cool extremities, fractional excretion of sodium) that are used by experienced clinicians to assess volume status.

Nishith K. Singh, MD
Gaurav Sangwan, MD
Peter White, MD
Southern Illinois University School of Medicine
Springfield, IL

The authors have no conflicts of interest to disclose.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestjournal.org/misc/reprints.shtml).

Correspondence to: Nishith K. Singh, MD, Department of Internal Medicine, Southern Illinois University School of Medicine, 701 N First St, Springfield, IL 62794-9636

DOi: 10.1378/chest.08-2024

REFERENCES
2 Guyton AC. Determination of cardiac output by equating venous return curves with cardiac response curves. Physiol Rev 1955; 35:123–129

Response

To the Editor:

We thank Drs. Cole and Singh and colleagues for their thoughtful comments. We agree on most points. Decisions regarding fluid management are among the most difficult in clinical medicine in general and in the ICU in particular. We believe that this decision should be based on an in depth understanding of the disease process and treatment strategy, with a review of fluid balance, oxygenation index, urine output, chest radiograph, and renal function (and