and pre-existing heart failure with reduced ejection fraction are potential confounders.[7] Patients included in this study may possess favorable clinical profiles which allowed for the use of NOAC, or did not have concomitant co-morbidities that require warfarin. Thus, the cases studied may only represent a select population and the results may not be generalizable to the entire population of LV thrombus patients. By extension, the authors could perhaps provide supplemental data on the reasons why these patients were started on NOAC rather than warfarin, which may be of use to clinicians in similar situations.

All in all, whereas limitations inherent to the nature of observational studies exist, this study by Fleddermann et al is a welcoming addition to the current literature and advances our understanding of NOAC use in LV thrombus treatment.

Disclosures

The authors have no conflicts of interest to disclose.

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1. Fleddermann AM, Hayes CH, Magalski A, Main ML. Efficacy of direct acting oral anticoagulants in treatment of left ventricular thrombus. *Am J Cardiol* 2019;124:367–372.
2. Ruff CT, Giugliano RP, Braunwald E, Hoffman EB, Deenadayalu N, Ezekowitz MD, Camm AJ, Weitz JI, Lewis BS, Parkhomenko A. Comparison of the efficacy and safety of new oral anticoagulants with warfarin in patients with atrial fibrillation: a meta-analysis of randomised trials. *Lancet* 2014;383:955–962.
3. Leow AS-T, Sia C-H, Tan BY-Q, Loh JP-Y. A meta-summary of case reports of non-vitamin K antagonist oral anticoagulant use in patients with left ventricular thrombus. *J Thromb Thrombolysis* 2018;46:68–73.
4. Kajy M, Shokr M, Ramappa P. Use of direct oral anticoagulants in the treatment of left ventricular thrombus: systematic review of current literature. *Am J Ther* 2019; https://www.ncbi.nlm.nih.gov/pubmed/30730331.
5. McCarthy C, Murphy S, Venkateswaran RV, Singh A, Chang LL, Joice MG, Rivero JM, Vaduganathan M, Januzzi JL, Bhatt DL. Left ventricular thrombus: contemporary etiologies, treatment strategies, and outcomes. *J Am Coll Cardiol* 2019;73:2007–2009.
6. Maniwa N, Fujino M, Nakai M, Nishimura K, Miyamoto Y, Kataoka Y, Asaumi Y, Tahara Y, Nakanishi M, Anzai T. Anticoagulation combined with antiplatelet therapy in patients with left ventricular thrombus after first acute myocardial infarction. *Eur Heart J* 2017;39:201–208.
7. Delewi R, Nijveldt R, Hirsch A, Marcu CB, Robbers L, Hassell ME, de Bruin RH, Vleugels J, van der Laan AM, Bouma BJ. Left ventricular thrombus formation after acute myocardial infarction as assessed by cardiovascular magnetic resonance imaging. *Eur J Radiol* 2012;81:3900–3904.

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On the QT

The significant study concerning the QT interval in atrial fibrillation as presented by Tooley et al[1] adds meaningful information to a complicated and still somewhat enigmatic subject. They conclude that the value of their study indicates that atrial fibrillation in itself is not causative of an abnormal QTc and that the differences between QTc in atrial fibrillation and sinus rhythm exist because of imperfect heart rate correction formulas. It is common knowledge that the use of Bazett's formula (QTc = QT/RR 0.5) has minor limitations, especially at the higher and lower heart rates of correction. It is still the most utilized "standard" by the cardiology community. It is accepted that in determining the QTc, the Bazett formula tends to overcorrect the interval at high heart rates and undercorrects it at low heart rates. The importance of their second opinion, that by carefully using the correction methods described by the authors, more definitive decisions can be made with respect to therapy for or against the use of specific antiarrhythmic drugs if contemplated. It is interesting that the authors found negligible difference between the manual and computer-derived versions of the QT interval. The errors of computer electrocardiography are well known and significant.[2] These errors have been noted primarily in the clinical interpretation − rhythm, depolarization and repolarization, wave forms, and other specific idiosyncrasies. The accuracy of conduction times (PR, QRS, and QT) has not been given the same attention. It is not unusual to find two different conclusions on the same patient, the same day and same electrocardiogram (ECG) recorder. Similarly, the same holds true from one ECG recorder to another and one program to another. Cardiologists are intimately aware of the difficulties in separating and diagnosing the genetically − induced forms of the long QT syndrome from those medically acquired. Even more difficulty can arise when both are simultaneously present. The QT interval can normally vary from lead to lead (50 to 65 ms) due to inherent QT dispersion just as it can during the same day, other days or from program and instrument differences. In a timely editorial, Conti stated "computers don't often measure the QT interval accurately, and for anyone concerned about QT prolongation, that it be measured by hand."[3] Another perplexing problem and question relates to the significance of depolarization conduction time and its influence on the prolonged QT interval. Is its presence of greater, lesser, or of irrelevant significance and consequence as an additional contribution? This would include the various time durations and forms of left- and right bundle branch block, and nonspecific intraventricular conduction delays. For example, a patient with a QTc of 544 ms and a QRS of 152 ms (left bundle branch block). Assuming a top normal ventricular conduction time as 100 ms, there is a 52 ms contribution of additional time to the QTc caused by depolarization which if subtracted from the total QTc results in a QTc of 492 ms. This obviously poses the question as to whether or not conduction delays in the QRS complex (depolarization) have the same or lesser relevance as those in the ST-T segment (repolarization) in terms of the abnormal QTc prolongation? This would of course, only apply to nonchannelopathy, medically acquired

QTc prolongation. Unfortunately the difference between life and death really can be measured in milliseconds!

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1. Tooley J, Ouyang D, Hadley D, Turakhia M, Wang P, Ashley E, Froelicher V, Perez M. Comparison of QT interval measurement methods and correction formulas in atrial fibrillation. *Am J Cardiol* 2019;123:1822–1827.
2. RuDusky B. Errors of computer electrocardiography. *Angiology* 1997;48:1045–1050.
3. Conti C. Inherited long QT syndrome: phenotype and therapy to use and avoid. *Clin Cardiol* 2006;29:429–430.

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Noninvasive Mechanical Ventilation in Combination With Propofol Deep Sedation in Left Atrial Ablation Procedures: Yes, But Should Be Cautious

We read with great interest the study by Vevecka et al[1] where the effectiveness of the combined use of noninvasive mechanical ventilation (NIV) and deep Propofol sedation during ablation procedures was determined. We congratulate the authors and agree that Propofol sedation may be the right choice. However, some aspects in this study are not adequately explained in context to both sedation and methodology, which needs serious consideration.

The sedation assessment in the methodology needs critical attention. Although authors have presented the data of total Propofol in different groups, their effect was not objectively assessed, and we believe the bias of varying sedation level and drug dose cannot be denied. Use of either processed electroencephalographic based or at least one sedation scale has given objectivity.[2] Moreover, drug dose per kilogram of body weight will be more informative.

Similarly, although the authors have used gasometry at 30 minutes apart, it is felt that the hypoventilation assessment was not adequate. End-tidal capnometry during NIV or nasal cannula oxygen supplement is not very reliable and needs alternative techniques.[3] Hypoventilation is also poorly associated with peripheral oxygen desaturation.[4] Therefore, the information on how the authors monitored hypoventilation continuously will be necessary for readers to assess and accept the results.

Furthermore, the basis for choosing the indication of NIV for respiratory depression with pH <7.25 and pCO_2 > 50 mm Hg needs more explanation and back-up by evidence. As the context condition resembles acute hypercapnic respiratory failure and respiratory acidosis, we believe that the use of the British Thoracic Society/Intensive Care Society guideline for ventilatory management for acute hypercapnic respiratory failure or so would have given more objectivity and acceptability.[5] This is very important, because, changing the indication for NIV has the potential to change the result. Moreover, a good number of patients had obstructive sleep apnea. It is unknown if any of these patients already used NIV or continuous positive airway pressure that could make the results more objective.

Upper airway permeability and NIV settings have a critical relation. The interaction of sedation on the patency of the respiratory tract should be considered, especially in patients with increased airways resistance and prone to tongue base collapse and obstruction as in obstructive sleep apnea.[6,7] Moreover, Propofol sedation even with subhypnotic doses impairs the pharyngeal muscle function,[8] and makes the patient vulnerable for bronchoaspiration; deep sedation increases the gravity of the situation. Therefore, the information whether authors have considered the settings for back rate inspiratory and expiratory ratios, preventive measures for aspiration, etc are crucial.

The authors have given a new dimension to the deep Propofol sedation by nonanesthesiologist; however, before we apply the results in clinical practice, further study with proper preanesthetic evaluation, monitoring, standard definition, and a multidisciplinary team involving anesthesiologists as well, will be required.

Thanking you and the authors

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1. Vevecka A, Schwab C, Forkmann M, Butz S, Issam A, Turschner O, Mahnkopf C, Brachmann J, Busch S. Predictive factors and safety of noninvasive mechanical ventilation in combination with propofol deep sedation in left atrial ablation procedures. Am J Cardiol. . pii: S0002-9149(19)30452-7. https://doi.org/10.1016/j.amjcard.2019.04.013. [Epub ahead of print].
2. Sheahan CG, Mathews DM. Monitoring and delivery of sedation. Br J Anaesth. 113(Suppl 2):ii37–ii47. https://doi.org/10.1093/bja/aeu378.
3. Gallagher JJ. Capnography monitoring during procedural sedation and analgesia. AACN Adv Crit Care. 29405–414. https://doi.org/10.4037/aacnacc2018684.
4. Sivilotti ML, Messenger DW, van Vlymen J, Dungey PE, Murray HE. A comparative evaluation of capnometry versus pulse oximetry during procedural sedation and analgesia on room air. CJEM. 12397–404.
5. Davidson AC, Banham S, Elliott M, Kennedy D, Gelder C, Glossop A, Church AC, Creagh-Brown B, Dodd JW, Felton T, Foëx B, Mansfield L, McDonnell L, Parker R, Patterson CM, Sovani M, Thomas L, BTS Standards of Care Committee Member, British Thoracic Society/Intensive Care Society Acute Hypercapnic Respiratory Failure Guideline Development Group, On behalf of the British Thoracic Society Standards of Care Committee. BTS/ICS guideline for the ventilatory management of acute hypercapnic respiratory failure in adults. Thorax. 71(Suppl 2): ii1–ii35. https://doi.org/10.1136/thoraxjnl-2015-208209.
6. Viana A, Zhao C, Rosa T, Couto A, Neves DD, Araújo-Melo MH, Capasso R. The effect of sedating agents on drug-induced sleep endoscopy findings. Laryngoscope. 129506–513. https://doi.org/10.1002/lary.27298.
7. Chang ET, Certal V, Song SA, Zaghi S, Carrasco-Llatas M, Torre C, Capasso R, Camacho M. Dexmedetomidine versus Propofol during drug-induced sleep endoscopy and sedation: a systematic review. Sleep Breath. 21727–735. https://doi.org/10.1007/s11325-017-1465-x.
8. Sundman E, Witt H, Sandin R, Kuylenstierna R, Bodén K, Ekberg O, Eriksson LI. Pharyngeal function and airway protection during subhypnotic concentrations of propofol, isoflurane, and sevoflurane: volunteers examined by pharyngeal videoradiography and simultaneous manometry. Anesthesiology. 951125–1132.

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