

A Wireless, Wearable Carotid Doppler Ultrasound Aids Diagnosis and Monitoring of Pericardial Tamponade: A Case Report

BACKGROUND: Pericardial tamponade can often be diagnosed through clinical findings and echocardiography; however, the diagnosis can be aided by demonstrating the hemodynamic consequences of the effusion. We describe the use of a wearable carotid Doppler device to help diagnose and monitor pericardial tamponade.

CASE SUMMARY: A 54-year-old man developed hypotension after an endobronchial biopsy for a lung mass. Echocardiography showed a pericardial effusion with sonographic evidence of tamponade. A wearable carotid Doppler device demonstrated low corrected carotid flow time (CFT) (a surrogate for stroke volume) with significant respiratory variation, supporting the diagnosis of tamponade. The patient underwent pericardiocentesis which revealed purulent pericardial fluid from a mediastinal abscess. After drainage there was increased CFT and reduced respiratory variability in Doppler, surrogates of improved stroke volume.

CONCLUSION: A wearable carotid Doppler device is a noninvasive tool that can help determine the hemodynamic impact of a pericardial effusion, and potentially aid in the diagnosis of pericardial tamponade.

KEY WORDS: Doppler; echocardiography; point-of-care; tamponade

In many cases, the diagnosis of pericardial tamponade is made using a combination of clinical findings and 2D echocardiography (1). For complex patients, or patients with other potential etiologies of shock, additional hemodynamic data including tricuspid and mitral valve inflow velocity variation, or respiratory variation in stroke volume (SV), is useful to confirm the diagnosis (1). These additional hemodynamic data also help clinicians weigh the risks and benefits of intervention. Hemodynamic assessment using echocardiography is an advanced skill, which may limit generalizability to all acute care practitioners.

In this case study, we describe the use of a novel wearable, noninvasive, carotid Doppler monitor that provides corrected carotid flow time (CFT), a real-time surrogate for SV, including its respiratory variation, to aid in the diagnosis and management of pericardial tamponade.²

CASE PRESENTATION

A 54-year-old man was admitted to the hospital ward and underwent an endobronchial ultrasound-guided mediastinal node biopsy for a workup of a new lung mass. On postadmission day 8, he developed chest discomfort, dyspnea, and hypotension with systolic blood pressures in the 70s–80s. He was treated with 2 liters of intravenous fluids with transient improvement in blood pressure and the ICU was consulted. Bedside ultrasound performed by the intensivist

Ross Prager, MD¹

Michael Pratte, MD²

Jon-Emile Kenny, MD^{3,4}

Philippe Rola, MD⁵

Copyright © 2023 The Authors. Published by Wolters Kluwer Health, Inc. on behalf of the Society of Critical Care Medicine. This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

DOI: 10.1097/CCE.0000000000000911



KEY POINTS

Question: Can a noninvasive carotid Doppler device aid in the diagnosis and management of cardiac tamponade?

Findings: A wearable carotid Doppler device was able to successfully identify low stroke volume (SV), as well as respiratory variation in stroke volume due to cardiac tamponade, and also demonstrated improvement in these parameters after pericardiocentesis.

Meaning: A wearable carotid Doppler device may be a useful adjunct to quantify the hemodynamic impact of pericardial effusions and to monitor response to treatment.

revealed a moderate to large pericardial effusion, right atrial collapse, and marked mitral inflow variation (Fig. 1B). A carotid Doppler patch (Flopatch, FloSonics Medical, Sudbury, ON, Canada) that uses 4 MHz continuous wave Doppler was applied, which showed a low CFT, a surrogate of SV, of around 275–285 ms (normal $320 \text{ ms} \pm 20 \text{ ms}$), with significant respiratory variation in the CFT, velocity time integral (VTI), and

peak systolic velocity (PSV) (Fig. 2A). A diagnosis of pericardial tamponade was made.

A bedside percutaneous pericardial pigtail catheter was inserted under ultrasound guidance, draining cloudy, serous fluid (Fig. 3). Hemodynamics were recorded continuously during the decompression procedure via the Doppler patch. After drainage of 300 mL of fluid, there was a marked improvement in CFT from 277 ms to greater than 326 ms (+15% change) and a marked decrease in PSV (45–8%; –37% change), VTI (52–17%; –35% change), and CFT (37–20%; –17% change) respiratory variation (Fig. 2). This correlated with a significant clinical improvement, with resolution of dyspnea and an increase in mean arterial pressure of 20%.

Case Evolution: Given the cloudy, purulent, appearing pericardial fluid, a computed tomography scan was performed which revealed an air-containing collection in the mediastinum contiguous with the pericardium, measuring 2.5 cm in maximum diameter, potentially suggestive of abscess at the site of the previous endobronchial biopsy. This led to the final diagnosis of purulent pericarditis as the cause for tamponade, with the pericardial fluid eventually growing *Streptococcus anginosus*. Unfortunately, the patient had progression of the malignancy with rapid growth of cerebral metastases and died several

weeks after the pericardiocentesis. The patient's next of kin provided consent for this case study.

DISCUSSION AND CONCLUSION

There are many ways in which pericardial tamponade—once suspected—may be diagnosed, from the traditional triad of muffled heard sounds, pulsus paradoxus, and hypotension to the various bedside ultrasound findings (plethoric inferior vena cava, chamber collapse, and elevated flow variations). In this case, the diagnosis was made by bedside clinical history and

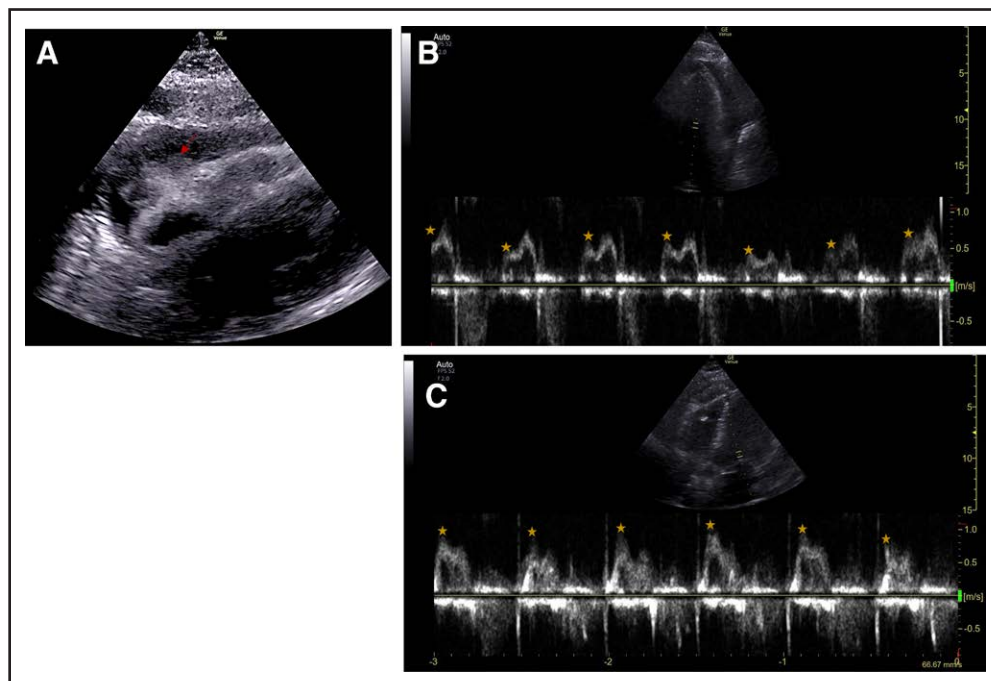


Figure 1. **A**, Subxiphoid view showing large pericardial effusion (red arrow). **B**, Pulse wave Doppler showing significant mitral valve inflow variation (> 25%). **C**, Postpericardiocentesis improvement in mitral valve inflow variation. Gold stars denote peak velocity of figures.

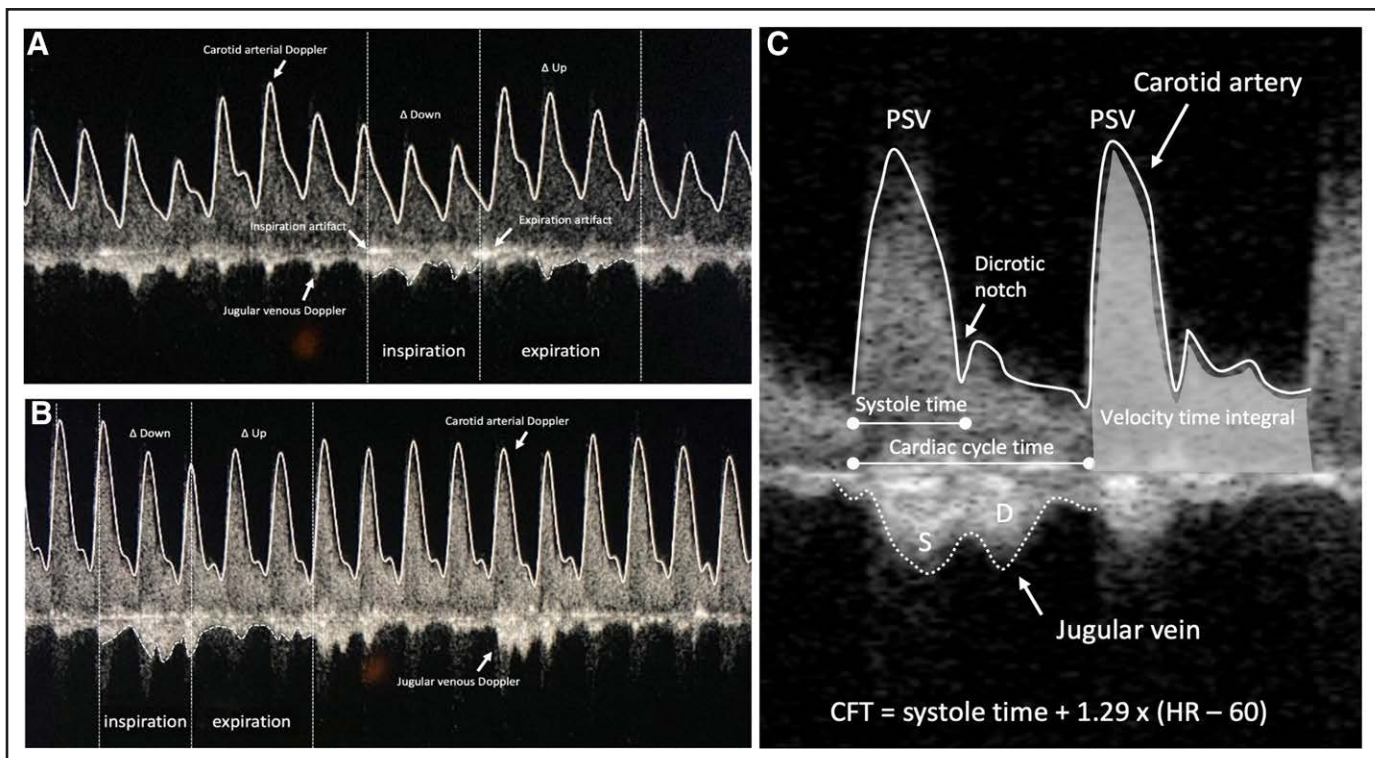


Figure 2. Carotid Doppler tracings before (A) and after (B) pericardiocentesis with associated changes in corrected carotid flow time (CFT) (277–326 ms; +15% change), peak systolic velocity (PSV) variation (45–8%; –37% change), velocity time integral variation (VTI) (52–17%; –35% change), and corrected carotid flow time variability (37–20%; –17% change).



Figure 3. Purulent pericardial fluid.

ultrasound, but also corroborated by the noninvasive Doppler signal. A wearable carotid Doppler device may be a useful adjunct to help diagnose tamponade or quantify the hemodynamic impact of a pericardial effusion, particularly in settings where advanced ultrasound expertise is not readily available. Low SV and significant respiratory variation in SV demonstrate a hemodynamic impact from the effusion and illustrate the pathophysiology of pulsus paradoxus.

The essentially instantaneous improvement of the CFT (a marker of SV) after pericardiocentesis, as well as the resolution of the excess respiratory flow velocity and CFT variation (a Doppler Kussmaul sign of sorts), supports the use of a Doppler patch as a monitoring tool for therapy in this population. In resting, supine, healthy volunteers, we observe an average CFT value of 320 ± 20 ms (2), thus the prepericardiocentesis value of 277ms was significantly reduced, indicative of diminished SV. Furthermore, we have shown that a 2–4% increase in CFT correlates with a 10% SV augmentation (3, 4). Accordingly, the +15% rise in CFT following pericardial drain could have represented a 30–40% rise in SV (4, 5) which is consistent with the notable blood pressure improvement after the effusion was drained. Changing CFT as a measure of changing SV has actually been reported since the mid-20th century (6–8). For example, respiratory variation in systolic time with constrictive pericarditis and pulsus paradoxus was reported in 1961 (6). Our findings are clinically relevant because real-time hemodynamic monitoring could be useful when a large pericardial effusion is discovered, and there is concern about rapid drainage of the effusion due to the potential for postpericardiocentesis

syndrome. In these cases, a wearable Doppler device could guide sufficient, but not excessive, pericardial drainage. Additionally, the Doppler patch could be used to assess the hemodynamic response of intravenous fluids (9–11) and vasopressors for patients before drainage or in situations where the risk and benefits of drainage are less clear.

Also of note is the jugular venous Doppler trace captured by the wearable ultrasound system. Before pericardiocentesis, the jugular Doppler is very low velocity and high power (i.e., signal intensity) consistent with a dilated jugular vein (i.e., an elevated central venous pressure) (Fig. 2). This is particularly striking if the jugular Doppler power (i.e., signal intensity) is normalized to the carotid Doppler power. Before drainage, this ratio is observably high (i.e., the jugular Doppler signal intensity is greater than that of the carotid signal intensity), which we have previously shown to correlate with elevated central venous pressure (12). Following drainage, the jugular velocities increase and the jugular vein Doppler power falls, demonstrating a less distended jugular vein (12). These observations are noteworthy because high central venous pressure is an important aspect of pulsus paradoxus physiology in cardiac tamponade (13).

Although in textbooks the diagnosis of cardiac tamponade is often touted as a clinical diagnosis our complex, multimorbid, and potentially undifferentiated patients with shock and pericardial effusion often require additional hemodynamic data to confirm the diagnosis, and to help weigh the risks and benefits of pericardiocentesis. A wearable Doppler device is a noninvasive tool that can help clinicians quantify the hemodynamic impact of pericardial effusions with regard to both venous return and cardiac output.

1 Division of Critical Care, Western University, London, ON, Canada.

2 Department of Medicine, University of Ottawa. Ottawa, ON, Canada

3 Health Sciences North Research Institute, Sudbury, ON, Canada.

4 Flosonics Medical, Toronto, ON, Canada.

5 Internal Medicine and Intensive Care, Santa Cabrini Ospedale, Montreal, QC, Canada.

All authors contributed to all aspects of the analysis and article preparation.

Dr. Kenny is the Chief Medical Officer for Flosonics, the medical technology company that produces and sells the wearable

Doppler device described in this case report. The remaining authors have not disclosed any potential conflicts of interest.

For information regarding this article, E-mail: rprag011@uottawa.ca

Informed consent was obtained from the patient's next of kin to use their anonymized clinical information in this case report.

REFERENCES

1. Little WC, Freeman GL: Pericardial disease. *Circulation* 2006; 113:1622–1632
2. Kenny J-ES, Barjaktarevic I, Mackenzie DC, et al: Carotid doppler measurement variability in functional hemodynamic monitoring: an analysis of 17,822 cardiac cycles. *Crit care explor* 2021; 3:e0439
3. Kenny J-ES, Barjaktarevic I, Mackenzie DC, et al: Diagnostic characteristics of 11 formulae for calculating corrected flow time as measured by a wearable Doppler patch. *Intensive Care Med Exp* 2020; 8:1–11
4. Kenny J-ES, Barjaktarevic I, Mackenzie DC, et al: Carotid Doppler ultrasonography correlates with stroke volume in a human model of hypovolaemia and resuscitation: analysis of 48 570 cardiac cycles. *Br J Anaesth* 2021; 127:e60–ee3
5. Kenny J-ES, Barjaktarevic I, Mackenzie DC, et al: Carotid artery velocity time integral and corrected flow time measured by a wearable Doppler ultrasound detect stroke volume rise from simulated hemorrhage to transfusion. *BMC Res Notes* 2022; 15:7
6. Weissler AM, Peeler RG, RoehlWHJr: Relationships between left ventricular ejection time, stroke volume, and heart rate in normal individuals and patients with cardiovascular disease. *Am Heart J* 1961; 62:367–378
7. Weissler AM, Harris WS, Schoenfeld CD: Bedside technics for the evaluation of ventricular function in man. *Am J Cardiol* 1969; 23:577–583
8. Harley A, Starmer CF, Greenfield JC: Pressure-flow studies in man. An evaluation of the duration of the phases of systole. *J Clin Invest* 1969; 48:895–905
9. Kenny J-ES, Barjaktarevic I, Mackenzie DC, et al: Inferring the frank–starling curve from simultaneous venous and arterial doppler: measurements from a wireless, wearable ultrasound patch. *Front Med Technol* 2021; 3:676995
10. Kenny J-ES: Functional hemodynamic monitoring with a wireless ultrasound patch. *J Cardiothorac Vasc Anesth* 2021; 35:1509–1515
11. Kenny J-ES: Assessing fluid intolerance with Doppler ultrasonography: a physiological framework. *Med Sci (Basel)* 2022; 10:12
12. Kenny J-ES, Yang Z, Clarke G, Elfarnawany M, Munding CE, Eibl JK, et al. The internal jugular vein-to-common carotid artery Doppler power ratio as a central venous pressure surrogate during simulated hemorrhage and transfusion: Proof-of-principle measurements from a wireless, wearable ultrasound. 2022;abstract (MHSRS Research Symposium)(Sept. 12-15, 2022).
13. Fessler HE: Heart-lung interactions: applications in the critically ill. *Eur Respir J* 1997; 10:226–237